This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
P23 .K25 1 STOR
Cyclopdia of the diseases of children :
24503356934

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885
LEVI COOPER LANE FUND

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
300 PASTEUR DRIVE
PALO ALTO, CALIF.

CYCLOPÆDIA

OF THE

DISEASES OF CHILDREN

MEDICAL AND SURGICAL.

THE ARTICLES WRITTEN ESPECIALLY FOR THE WORK BY
AMERICAN, BRITISH, AND CANADIAN AUTHORS.

EDITED BY
JOHN M. KEATING, M.D.

VOL. I.

ILLUSTRATED.

PHILADELPHIA:
J. B. LIPPINCOTT COMPANY.
1889.

LANE

Copyright, 1889, by J. B. Lippincott Company.

25
R35
1889
v.1

PREFACE.

ALTHOUGH it might almost seem presumptuous to have engaged in an undertaking of so vast a scope as that of the present work, the Editor feels that the excellence of the articles that have been contributed by the various collaborators justifies the course he has pursued, both in the selection of the writers and in the general plan.

His object has been to include not only the Medicine and Surgery of Pediatrics, but also all the specialties tributary to it, as well as all collateral subjects of interest and importance, many of which cannot be found treated in any other work of this character. He has also included articles upon certain prominent symptoms, such as cough, convulsions, etc., the proper significance of which could be estimated only after laborious research, unless the matter were considered in special chapters.

The work, in fact, consists of a collection of monographs—not mere dictionary articles—arranged in the form of a systematic treatise, and devoted to the consideration of the anatomy, physiology, medicine, surgery, and hygiene of infancy, childhood, puberty, and adolescence.

Each writer is alone responsible for his statements. Many of the collaborators have deemed it expedient to consider their subjects from the stand-point of the adult, viewing in detail the various deviations as affecting childhood. Some, on the other hand, taking for granted a perfect familiarity with adult diseases, have dwelt entirely upon the affections of childhood.

It is a matter of regret to us in this country that many of our practitioners enter upon their careers with but a meagre acquaintance with all that pertains to Pediatrics. Fortunately, however, nature has endowed the American mind with energy, enthusiasm, penetration, and natural aptitude, and as a consequence we are enabled to

14109

point to a brilliant array of honored names, of those who have fought the battle single-handed in the conflict and struggle of an active and extended practice. In this country the study of the diseases of children was fostered altogether at the bedside of private practice by the unremitting conscientious labor of a few,—unambitious as to the laurels they might win, ever bent upon arming others with their experience and inspiring them with their enthusiasm.

This Cyclopædia is the outgrowth of the work of these men, and is a tribute to the untiring efforts of such teachers and writers as Dewees, Eberle, Condie, Charles D. Meigs, W. V. Keating, John Forsyth Meigs and William Pepper, J. Lewis Smith, and A. Jacobi, —names which rank to-day on an equal footing with those of the ablest European teachers of Pediatrics.

As each contributor has been selected with special reference to his familiarity with the subject, it must follow that the articles will not only be of immediate practical utility, but will also serve as standards for future reference. The introduction of the surgical essays and certain special articles has necessitated a more minute subdivision of the subject than is usual in works of this kind, which naturally causes a certain amount of overlapping, and possibly an occasional conflict of opinion, but we believe this is rather an advantage than otherwise.

A few words should be said in regard to the illustrations. We have endeavored to reproduce as accurately as possible and by the best processes all the illustrations which the authors have deemed necessary for the elucidation of their subjects. A large number of plates reproduced directly from the photographic negatives form a special feature. In the article on Anatomy, the author has preferred the introduction of photographs of a few special dissections and preparations, instead of depending entirely upon diagrams, thus sacrificing clearness in detail to accuracy. The great difficulty of arranging the camera which has to be suspended, and the imperfect lighting of the subject, will be readily understood by all who have attempted the photographic reproduction of anatomical specimens.

Although the work is intended chiefly for the American physician, the Editor has sought the co-operation of a few of the most distinguished teachers of Great Britain, the outcome of whose ripe experience and profound research cannot but prove a valuable addition to our literature.

The aim of the Editor has been, as far as possible, to impress upon each writer the importance of giving his article an individuality of its own, avoiding too liberal quotation, which tends to fatigue and confuse rather than to entertain and instruct the reader. He believes that he has succeeded in uniting in a single work a collection of monographs expressive of the views of most of the distinguished teachers of this country and Great Britain, and that these volumes, owing to their scientific excellence and practical value, will be of equal importance to the busy practitioner and to the student and teacher.

The Editor feels himself under a special obligation to the writers who have so heartily joined with him in this work, often at great personal inconvenience to themselves, many of them indeed laboring under the serious disadvantage of extreme professional pressure. He wishes to take this opportunity of expressing his thanks to Drs. William Osler, Thomas Barlow, T. M. Rotch, T. Lauder Brunton, J. Mitchell Bruce, B. Sachs, and W. A. Edwards, through whose able co-operation the plan of this work has been elaborated and its accomplishment realized. He also desires to acknowledge his indebtedness to Mr. Joseph McCreery, who has so carefully and critically examined the proofs.

Philadelphia, May 1, 1889.

CONTENTS OF VOLUME I.

PAGE

INTRODUCTORY. By A. Jacobi, M.D., New York, Clinical Professor of Diseases of Children in the College of Physicians and Surgeons, New York, late President of the New York Academy of Medicine 1

PART I.

GENERAL SUBJECTS.

ON THE ANATOMY OF CHILDREN. By George McClellan, M.D., Philadelphia, Pa., Lecturer on Anatomy, Pennsylvania School of Anatomy; Surgeon to and Lecturer on Anatomy, Philadelphia Hospital, etc. 11

THE PHYSIOLOGY OF INFANCY. By Angel Money, M.D., M.R.C.P., London, England, Assistant Physician to the Hospital for Children, Great Ormond Street, and to the University College Hospital 51

DIAGNOSIS. By James Finlayson, M.D., Glasgow, Scotland, Physician to Glasgow Hospital for Sick Children; Physician and Lecturer on Clinical Medicine, Glasgow Western Infirmary; formerly President of the Glasgow Pathological and Clinical Society; Honorary Librarian to the Faculty of Physicians and Surgeons, Glasgow 73

THE INFLUENCE OF RACE AND NATIONALITY UPON DISEASE. By J. Wellington Byers, M.D., Charlotte, North Carolina 132

OUTLINES OF PRACTICAL BACTERIOLOGY. By E. O. Shakespeare, M.D., Ph.D., etc., Philadelphia, Pa., Pathologist and Ophthalmologist to the Philadelphia Hospital; United States Commissioner to India for the Investigation of Cholera; Ex-President of the Philadelphia Pathological Society 147

MATERNAL IMPRESSIONS. By William C. Dabney, M.D., University of Virginia, Professor of Obstetrics and Practice of Medicine in the University of Virginia 191

DISEASES OF THE FŒTUS. By Barton Cooke Hirst, M.D., Philadelphia, Pa., Associate Professor of Obstetrics in the University of Pennsylvania; Obstetrician to the Philadelphia and Maternity Hospitals 217

THE CARE OF THE CHILD AT AND IMMEDIATELY AFTER BIRTH, IN HEALTH AND DISEASE. By R. A. F. Penrose, M.D., LL.D., Philadelphia, Pa., Emeritus Professor of Obstetrics and Diseases of Women and Children in the University of Pennsylvania, etc. 236

PAGE

THE CLOSURE OF THE DUCTUS ARTERIOSUS AND OF THE UMBILICAL AND HYPOGASTRIC ARTERIES. By J. COLLINS WARREN, M.D., Boston, Mass., Associate Professor of Surgery, Harvard University; Surgeon to the Massachusetts General Hospital 252

INJURIES OF THE NEW-BORN. By THEOPHILUS PARVIN, M.D., LL.D., Philadelphia, Pa., Professor of Obstetrics and Diseases of Women and Children in the Jefferson Medical College; Visiting Obstetrician to the Philadelphia Hospital 258

INFANT-FEEDING—WEANING. By T. M. ROTCH, M.D., Boston, Mass., Assistant Professor of Diseases of Children in Harvard University; Physician to Boston City Hospital, Children's Hospital, Infants' Hospital, and West End Nursery 270

WET-NURSES. By WM. H. PARISH, M.D., Philadelphia, Pa., Obstetrician to the Philadelphia Hospital; Professor of Obstetrics, Dartmouth Medical College; Professor of Anatomy, Woman's Medical College of Pennsylvania; Member of the American Gynæcological Society 330

DIET AFTER WEANING. By SAMUEL S. ADAMS, A.M., M.D., Washington, D.C., Professor of the Theory and Practice of Medicine, Medical Department of National University; Attending Physician to the Children's Hospital, etc.. . 337

NURSING OF SICK CHILDREN. By MISS CATHERINE WOOD, London, England, Great Ormond Street Hospital for Children 347

NURSERY HYGIENE. By L. M. YALE, M.D., New York, Lecturer Adjunct on Diseases of Children at Bellevue Hospital Medical College 360

DENTITION. By JOHN DORNING, M.D., New York, Instructor in Diseases of Children, New York Polyclinic; Attending Physician to St. Joseph's Hospital . 373

PUBERTY: ITS PATHOLOGY AND HYGIENE. By THOS. MORE MADDEN, M.D., F.R.C.S., Dublin, Ireland, President of the Obstetric Section of the British Medical Association; Physician to Hospital for Sick Children (St. Joseph's), Dublin; Obstetric Physician to Mater Misericordiæ Hospital; Ex-President of the Obstetric Section of the Academy of Medicine in Ireland; formerly Examiner in Obstetrics and Diseases of Women and Children, Queen's University 389

PART II.

FEVERS AND MIASMATIC DISEASES.

FEVER—GENERAL CONSIDERATIONS AND TREATMENT.—SIMPLE CONTINUED FEVER.—THERMIC FEVER. By WILLIAM PASTEUR, M.D., F.R.C.P., London, England, Physician to North-Eastern Hospital for Children; Medical Registrar to Middlesex Hospital 417

ENTERIC OR TYPHOID FEVER. By JAMES C. WILSON, M.D., Philadelphia, Pa., Physician to the Philadelphia Hospital, the Jefferson College Hospital, and St. Agnes's Hospital 441

TYPHUS FEVER. By ALEXANDER COLLIE, M.D., M.R.C.P., London, England, Medical Superintendent of the Eastern (Fever) Hospitals, etc. 497

RELAPSING FEVER. By ROLAND G. CURTIN, M.D., Philadelphia, Pa., Lecturer on Physical Diagnosis in the University of Pennsylvania; Visiting Physician to the Philadelphia and Presbyterian Hospitals, etc. 504

PAGE

CEREBRO-SPINAL FEVER. By J. Lewis Smith, M.D., New York, Clinical Professor of Diseases of Children, Bellevue Hospital Medical College; Physician to the New York Foundling Asylum; Consulting Physician to the New York Infants' Asylum . . . 514

SCARLET FEVER. By Samuel C. Busey, M.D., LL.D., Washington, D.C., Fellow of the American Gynæcological Society, etc. . . . 555

DIPHTHERIA. By J. Lewis Smith, M.D., New York, Clinical Professor of Diseases of Children in Bellevue Hospital Medical College; Physician to the New York Foundling Asylum; Consulting Physician to New York Infants' Asylum, etc. . . . 580

MEASLES. By F. E. Waxham, M.D., Chicago, Ill., late Professor of Diseases of Children, College of Physicians and Surgeons of Chicago; Professor of Otology, Rhinology, and Laryngology, College of Physicians and Surgeons of Chicago, etc. . . . 675

RUBELLA (Rötheln). By Wm. A. Edwards, M.D., San Diego, California, formerly Instructor in Clinical Medicine, and Physician to Medical Dispensary, in the University of Pennsylvania . . . 684

PERTUSSIS. By T. M. Dolan, M.D., F.R.C.P., Halifax, England, Fellow of the Medical Society and of the Obstetrical and Gynæcological Societies of London . 703

VARIOLA. By A. D. Blackader, M.D., Montreal, Canada, Instructor in Diseases of Children, McGill University; Physician to the Out-Door Department for Diseases of Children, Montreal General Hospital; Physician to St. Margaret's Nursery and Protestant Infants' Home . . . 722

VACCINATION. By W. T. Plant, M.D., Syracuse, N.Y., formerly Passed Assistant Surgeon, United States Navy; Professor of Pediatrics in the Medical Department of Syracuse University . . . 743

VARICELLA. By Charles G. Jennings, M.D., Detroit, Mich., Professor of Diseases of Children and Chemistry, Detroit College of Medicine; and Physician for Diseases of Children, Out-Patient Department, St. Mary's Hospital . . . 754

PAROTITIS. By O. P. Rex, M.D., Philadelphia, Pa., Physician to Presbyterian and Jefferson College Hospitals; Clinical Lecturer on Diseases of Children, Jefferson Medical College . . . 767

ERYSIPELAS. By Jos. O. Hirschfelder, M.D., San Francisco, Cal., Professor of Clinical Medicine in Cooper Medical College; Visiting Physician to the City and County Hospital . . . 771

RHEUMATISM. By W. B. Cheadle, M.D., F.R.C.P., London, England, Physician to St. Mary's Hospital and Lecturer on Medicine at St. Mary's Medical School; Senior Physician to the Hospital for Children, Great Ormond Street, London, etc. . . . 785

MALARIA. By F. Forchheimer, M.D., Cincinnati, Ohio, Professor of Physiology and Clinical Diseases of Children, Medical College of Ohio; Physician to Home for Sick Children; Physician to Children's Ward, Good Samaritan Hospital; Physician to Cincinnati Hospital; Member of Association of American Physicians, etc. . . . 825

YELLOW FEVER. By John Guitéras, M.D., Charleston, S.C., Passed Assistant Surgeon, United States Marine Hospital Service; Professor of Pathology in the University of Pennsylvania; formerly Professor of the Theory and Practice of Medicine, Medical College of South Carolina, etc. . . . 853

DENGUE. By Rudolph Matas, M.D., New Orleans, La. . . . 878

CHOLERA INFECTIOSA. By E. O. SHAKESPEARE, M.D., Philadelphia, Pa., Pathologist and Ophthalmologist to the Philadelphia Hospital; United States Commissioner to India for the Investigation of Cholera, etc. . . . .

JOINED TWINS. By WILLIAM WRIGHT JAGGARD, M.D., Chicago, Ill., Professor of Obstetrics, Chicago Medical College, Medical Department of Northwestern University; Obstetrician to Mercy Hospital, Bellevue . . . .

EMBRYOLOGY. By HORACE JAYNE, M.D., Philadelphia, Pa., Professor of Vertebrate Morphology in the University of Pennsylvania . . . .

THE GENERAL THERAPEUTICS OF CHILDREN'S DISEASES. By ROBERTS BARTHOLOW, M.D., LL.D., Philadelphia, Pa., Professor of Materia Medica, General Therapeutics, and Hygiene in the Jefferson Medical College, Philadelphia, etc. . . . .

LIST OF ILLUSTRATIONS TO VOLUME I.

PLATES.

PAGE
Photograph of Skull of Infant Seven Months Old, front view . . . . facing 12
The same, side view . . . . " 12
Photograph from Dissection of Brain of Infant, showing Convolutions and Fissures . . . . " 12
Vertical Section (frozen) from a Child Six Years Old, by Symington . . . . " 14
Another Section of the same . . . . " 14
Vertical Section (frozen) from Child's Head, Six Years Old . . . . " 16
Horizontal Section (frozen) from Child's Head, Six Years Old, by Symington " 16
Skull of Child Seven Years Old, showing Permanent and Temporary Sets of Teeth . . . . " 18
Jaws of Child Seven Years Old, showing Permanent and Temporary Sets of Teeth . . . . " 18
Photograph from Dissection showing Relations of Bladder and Uterus in Infant . . . . " 18
Longitudinal Section (frozen) from Child Six Years of Age, by Symington . " 20
Photograph from Preparation showing Lymphatic Vessels of Head, Face, and Neck, from Mütter Museum . . . . " 22
Photograph from Dissection of Child Ten Months Old . . . . " 24
Photograph of Child Seven Months Old, showing Topographical Relations of Organs and Landmarks in front . . . . " 26
Photograph of Child Four Years of Age, showing Topographical Position of Organs and Landmarks in front . . . . " 28
Photograph of Child Four Years of Age, showing Topographical Position of Organs and Landmarks behind . . . . " 28
Photograph of Boy Ten Years of Age, showing Topographical Position of Organs and Landmarks in front . . . . " 30
Photograph of Recent Dissection of New-Born Child . . . . between 32 and 33
Diagram of the same . . . . " 32 and 33
Photograph of Recent Dissection of New-Born Infant, showing Position and Shape of Stomach . . . . facing 34
Photograph from Recent Dissection of New-Born Infant, showing Usual Size of Liver . . . . between 34 and 35
Diagram of the same . . . . " 34 and 35
Photograph of Child Seven Months Old, showing Topographical Position of Organs and Landmarks behind . . . . facing 36
Photograph of Skeleton of Child Five Months Old . . . . " 38
Photograph from Preparation in Mütter Museum, showing Lymphatic Vessels of Skull, Head, Face, and Neck of Child Six Years Old . . . . " 40
Photograph of Micro- and Streptococci . . . . " 182
Tubercle-Bacilli in Sputum . . . . " 182
Typhoid Bacilli . . . . " 182
Bacteria of Intestinal Contents, with Two Comma Bacilli . . . . " 182

P

Photograph of Colony of Cholera Nostras. Finkler . . . . facing
Photograph of Colony of an Air Micrococcus . . . . "
Photograph of Colony of Comma Bacillus of Koch . . . . "
Photograph of Colonies of Curved Bacillus resembling that of Koch . . . . "
Photograph of Culture in Gelatin-Tube of Comma Bacillus of Koch . . . . "
Comparison of Tube-Cultures. Comma Bacillus of Koch and Curved Bacillus of Finkler . . . . "
Photograph of Pure Culture of Deneke . . . . "
Photograph of Pure Culture of Finkler . . . . "
Photograph of Pure Culture of Comma Bacillus of Koch . . . . "
Charts. Enteric Fever . . . . "
Chart. Enteric Fever with Relapse . . . . "
Chart. Enteric Fever. Remittent Type . . . . "
Chart. Enteric Fever complicated by Measles . . . . "
Chart of Cases of Typhus Fever . . . . "
Rheumatic Nodules, and Microscopic Section of the same . . . . "
Charts of Cases and Death-Rate of Yellow Fever . . . . "
Charts of Cases and Death-Rate of Yellow Fever . . . . "
Charts of Temperature of Yellow Fever . . . . "
Photo-micrograph of McLaughlin's Dengue Micrococcus Culture . . . . . "
Map of Geographical Distribution of Dengue . . . . "
Iniodymus (Depaul), Opodymus (Soemmering), Dicephalus Dibrachius . . . "
Rita-Christina (Serres), Dicephalus Tetrabrachius (Serres), Rose-Marie Drouin, "the Saint Benoit Twins" . . . . "
Ischiopagus (Prochaska). Pelvis of an Ischiopagus (after Du Verney), Helen and Judith. The Two-Headed Nightingale, Dipygus Tetrapus . . . . . "
Dipygus Dibrachius Tetrapus, Dipygus Parasiticus, Janiceps (Bordenave), Prosopo-Thoracopagus . . . . "
Janiceps Asymmetros (Sterley) . . . . "
Craniopagus Parietalis (De Baer), The Siamese Twins, Sternopagus (Maternité) "
Sternopagus (Herrgott), Sternopagus Parasiticus . . . . "

FIGURES.

Hydrocephalic Skull of Infant . . . .
Diagram of Fissures . . . .
Section of Temporal Bone . . . .
Transverse Section of Neck of Child Six Years of Age . . . .
Anterior Region of Neck of Child Nine Years of Age . . . .
The Fœtal Circulation . . . .
Human Embryo of the Ninth Week . . . .
Diagram of Nerve-Circuit . . . .
Diagrams of Cocci, Bacilli, Bacteria, and Spirilli, after Hüppe 150, 151, 153, 154, 155,
Instruments for Bacteriological Investigation 171,
Dry-Heat Steriliser . . . .
Steam Steriliser . . . .
[illegible] . . . .
Porcelain Filter of Chamberland . . . .
Femur showing Syphilitic Osteochondritis, Wegner . . . .
Microscopic Appearance of Syphilitic Osteochondritis, Wegner . . . .
Section of Femur showing Syphilitic Osteochondritis . . . .
[illegible], from Depaul . . . .

PAGE
Caput Succedaneum of Face and Vertex, from Depaul 260
Funnel-Shaped Depression of Left Parietal Bone, from Winckel 264
Fissure of Right Parietal Bone, from Winckel 264
Position after Pelvic Presentation 269
Chart showing Gain in Weight of Healthy Child for First Twenty-nine Weeks . . 272
Diagram of Actual Size of Stomach of Infant Five Days Old 294
Diagram showing Actual Size of Tube for each Feeding of Child during First Week 295
Diagram showing Actual Size of Stomach of Female Infant Twelve Months Old . 269
Diagram showing Actual Size of Stomach of Rachitic Infant Seven Months Old . . 297
Diagram of Tubes used in Infant Feeding 298
Sterilizer for Infant Feeding 299
Four-Ounce Erlmeyer Flask 328
Nursery Window-Sash 368
Diagram of Bacilli of Enteric Fever, Flügge 446
Chart of Relapsing Fever, from Murchison 508
Clonic Convulsion in Cerebro-Spinal Fever 530
Pigmented Body in Red Blood-Corpuscle 829
Small Vein of the Pia Mater, with Pigmented Hyaline Bodies, Councilman 829
Section of Kidney, showing the same 829
Chart of Thermic Fever 871
Micrococci in Blood of Dengue Fever 885
Section of Ovary 939
Section of Ovary 940
Ovum of the Cat 940
Formation of Polar Cells in a Star-Fish 941
Fertilization of Ovum of a Star-Fish 944
First Stages of Segmentation of a Rabbit's Ovum 945
Optical Sections of a Rabbit's Ovum 945
Rabbit's Ovum after Impregnation 946
Blastodermic Vesicle of a Rabbit 946
Embryonic Area of a Seven Days' Embryo Rabbit 947
Sections through the Blastoderm of a Mole 947
Embryo Rabbit of Eight Days and Nine Hours 948
Embryo Rabbit of Eight Days and Nine Hours with Five Protovertebræ 949
Transverse Section of an Embryo Fowl of Three Days' Incubation 949
Diagrammatic Longitudinal Section through the Axis of an Embryo 950
Five Diagrammatic Figures illustrating the Formation of the Fœtal Membranes of a Mammal 951
Diagram of the Fœtal Membranes of a Mammal 952
Diagram of the Circulation of the Yolk-Sac of a Chick at the End of the Third Day of Incubation 953
Structure of the Human Placenta 955

CYCLOPÆDIA

OF THE

DISEASES OF CHILDREN.

INTRODUCTORY.

By A. JACOBI, M.D.

Upon me has been conferred the honor of introducing to the medical public the essays of all the distinguished men contributing to this great work. Though with some hesitation, it is with still more satisfaction that I comply with this demand. For the very enterprise marks an immense progress in the history of both general medical and pediatric literature. Indeed, when I began my professional life, such a collection of monographs as will here be offered could not have been written. Now, that during a single generation there should have been such a thorough change in the methods of both medical thought and work, is a source of the most intense gratification as well to me as to every other man who has absolute faith in the persistent evolution of science and the improvement of the race.

That there should be any doubt as to the propriety of a large special work on the diseases of children, I can hardly believe in the present stage of development of American medical literature. As far as I am concerned, I never objected to being found among the adversaries of the wildfire of specialism which has been spreading among the groups of medical men. On the contrary, I am still of the opinion I expressed eight years ago when I opened the first session of the Section on Diseases of Children, of the American Medical Association, in its meeting at New York.

With more pertinacity than logic, pediatrics (comprehending the anatomy, physiology, pathology, and therapeutics of infancy and childhood) has also been claimed as a specialty. This is a mistake, however, which has been made more frequently on the continent of Europe than with us. It is there that practitioners and authors advertise themselves, for reasons of their own which would not be approved of here, as "children's physicians" and "specialists." Pediatrics, however, is no specialty in the common acceptation of the term. It does not deal with an organ, but with the entire organism at the very period which presents the most interesting features to the student of biology and medicine. Infancy and childhood are the links between conception and death, between the fœtus and the adult. The latter has attained a certain degree of invariability. His physiological labor is reproduction, that of the young is both reproduction and growth. As the history of a people is not complete with the narration of its condition when established on a solid constitutional and material basis, so is that of man, either healthy or diseased, not limited to one period. Indeed, the most interesting time and that most difficult to understand is that in which a persistent development, increase, and improvement are taking place.

This appears to have been felt, instinctively, from the very beginning. The history of pediatrics, therefore, is as old as that of medicine. Their literatures have developed uniformly, from superstitious beliefs to empirical statements and the methodical researches of the present time. The last centuries, particularly the last decades, are replete with text-books on the diseases of children, monographs on their pathology, physiology, and hygiene, and journals, quite a number of which are now published in the four principal languages of the civilized world.

These monographs and journals have contributed a great deal to the amount of medical knowledge. Special researches of the normal condition of embryonic, fœtal, and infant growth, the study of the functions of the organs in their constant development and changes, and anatomical and clinical investigations, have contributed to prove that pediatrics does not deal with miniature men and women, with reduced doses and the same class of diseases in smaller bodies, but that it has its own independent range and horizon, and gives as much to general medicine as it has received from it.

There is scarcely a tissue, or an organ, which behaves exactly alike in the different periods of life. The bones contain less phosphates in the young and exhibit other chemical differences, their anatomical structure is different, their increase less periosteal, than in advanced years. The cartilaginous condition of the epiphyses gives rise to a number of disorders; the cartilages between the epiphyses and diaphyses are subject to all forms of disease, from a simple irritation resulting in abnormal growth (for instance, after eruptive fevers) to a separation, by suppuration, of the epiphyses. There is hardly a chapter more interesting than that of the relation of the bones of the cranium to its contents. A solid skull serves as a support to the brain and its blood-vessels, or it may prove an obstacle to their development; an insufficient degree of ossification, and an undue amount of sutural substance, will enhance the possibility of enlargement of the blood-vessels and the liability to effusion. Premature ossification, however, either partial or general, is a cause of asymmetry, epilepsy, or idiotism, and influences the course of intercurrent diseases. The large size of the head, which is equalled by that of the thorax about the middle or the end of the third year only, is in close relation to the physiological growth of the brain and its pathological changes.

The vertebral column is quite flexible, but straight, and mainly so in its upper portion. Its very flexibility is a ready cause of the frequent occurrence of scoliosis. Its distance from the manubrium sterni is so small that occasionally a thymus, and frequently enlarged lymph-bodies, are a cause of irritation or compression. The base of the thorax is, however, relatively wide, while its height is less. This becomes particularly striking by the almost rectangular insertion of the ribs at the transverse processes of the vertebræ and the sternum, and by their almost horizontal and circular position, by which the respiration becomes less costal, and the viscera of the abdominal cavity, mainly the liver, appear more prominent. Changes of a pathological character are quite frequent about this time, and a frequent cause of disease in later life. Hueter's researches on the congenital contraction of the chest, and Freund's investigations on the premature ossification of the costo-cartilaginous junctures, are exceedingly important, inasmuch as they explain many of the isolated cases of thoracic insufficiency, phthisical habitus, and pulmonary incompetency.

The nervous system of the young is but in a preparatory condition. The brain is large, but contains a large percentage of water, is soft, and its gray and white substances differ but little in color and composition. The spinal cord has not yet the consistency of a later period ; the anterior horns are predominant, and therefore more frequently the seat of pathological changes. The peripheral nerves are relatively large, but little excitable, in the first days. Their excitability grows very fast, however, towards the end of the first year, and quite out of proportion with the slow development of the inhibitory centres. Thus it is that about that time convulsive symptoms are so very frequent. For a short time after birth the conducting fibres between the undeveloped brain (it takes the psycho-motor centres of Ferrier and Hitzig a month to exhibit the first signs of existence) and the pyramidal fibres of the cord perform no functions : thus the first movements of the newly-born are not controlled by will-power at all, but subject to reflex exclusively. After that time the brain develops very fast indeed, but far from uniformly in all its parts. It is a most interesting study thus to follow the evolution of the cerebral functions in their dependency upon the anatomical development.

The digestive organs of the infant exhibit a great many peculiarities in their anatomy, physiology, and pathology. The epithelial "pearls" along the median line of the palate, and the thinness of the mucous membranes over the roof of the oral cavity and along the gums, give rise to early trouble, the small size and vertical position of the stomach to a number of abnormal symptoms, the congenital malformations of the intestine to serious dangers, the abnormal length of the lower part of the colon to an unusual form of protracted constipation, the prevalence of polypi in the rectum to hemorrhages of a kind seldom found in advanced age. The glands required for the digestive processes are but gradually prepared for their functions. The salivary glands are but partially active at birth, the pancreas requires time for its full development, the secretion of lactic predominates over that of muriatic acid in the stomach, the intestinal lymph-bodies are in part, particularly the patches of Peyer, so behind their future size and formation as to change their functions considerably. The time of dentition adds to the interest of the period, more, it is true, from a physiological and anatomical stand-point than on account of pathological reasons; for its alleged causal connection with the large number of diseases attributed to its mere occurrence has been greatly exaggerated.

In connection with these brief remarks on some of the peculiarities of the alimentary tract of infancy, I may be permitted to merely allude to the question of nutrition and feeding. Several meetings of the Children's Section of the German Association of Physicians and Naturalists, the last one of that in the American Medical Association, and the deliberations of every medical society in every land, prove its importance. These questions belong, as special studies, eminently to pediatrics; physiology and chemistry can teach the general principles only, and to clinical observation is left the final settlement of the hygiene of infancy. The relation of nurse's to mother's milk, the utilization of cow's milk in all its different forms as one of the constituents of artificial foods, the value of farinaceous admixtures, the addition of animal foods, the proportions of salts and water, the quantity to be administered, the length of intervals between meals, the alterations required in sickness, are just so many questions which demand persistent study and special industry.

The blood and the organs of circulation exhibit the most interesting differences in the young as compared with the adult.

The young infant (and child) has less blood in proportion to its entire weight; this blood has less fibrin, fewer salts, less hæmoglobulin (except in the newly-born), less soluble albumin, less specific gravity, and more white blood-corpuscles than the blood of advanced age.

There are some other differences, depending on age, in the composition of the blood, more or less essential. The fœtal blood and that of the newly-born contain but little fibrin, but vigorous respiration works great changes in that respect. Nasse found the blood of young animals to coagulate but slowly. In accordance with that observation, it strikes us, in regard to cerebral apoplexy of the newly-born, that the time for coagulation of the blood must be longer than in the adult; for hemorrhages are apt to be most extensive in the infant. In the sanguineous tumor (kephalhæmatoma) of the newly-born, the blood remains liquid in the sac for many days. In apoplexy it is apt to spread all over the hemispheres, and has plenty of time to perforate and penetrate the pia in all directions, destroy much of the cerebral tissue, and flow down the spinal cavity. These occurrences are so frequent in the infant, and so rare in the apoplectic adult, that they can hardly be explained except through the insufficient coagulability of fœtal and infant blood.

The size and vigor of the newly-born heart offer a ready explanation of the rapid growth of the infant body, and mainly those organs which are in the most direct communication with the heart by straight and fairly large blood-vessels. In this condition are the head and brain. Thus the latter has an opportunity to grow from 400 grammes to 800 in one year; after that period its growth becomes less marked. At seven, boys have brains of 1100 grammes; girls, of 1000. In more advanced life its weight is relatively less,—1424 in the male and 1272 in the female. At the same early period the whole body grows in both length and weight. The original length of 50 centimetres of the newly-born increases to 110 with the seventh year; the greatest increase after that time amounts to 60 (in the female 50) centimetres only. In the same time the weight increases from 3.2 kilogrammes to 20.16 in the boy, from 2.9 to 18.45 in the girl. This gives a proportion of 1 to 6 or 7, while after that time the increase is but three- or fourfold.

The normal relation of the weight of the heart to that of the lungs, between the second and twentieth year, is 1 : 5–7; in scrofula it is 1 : 8–10. That means, the heart is smaller than normal, in the latter condition. Other parts of the system of circulation exhibit traits of their own. It is particularly in the "torpid" form of scrofula that, by virtue of insufficient circulation, the lymphatic system participates pre-eminently. This fact is the more important, as the size, patency, and number of lymphatics are quite unusual in infancy. Sappey found that they could be more easily injected in the child than in the adult, and the intercommunication between them and the general system is more marked at that than at any other period of life. These facts have been confirmed by S. L. Schenk, who, moreover, found the net-work of the lymphatics even in the skin of the newly-born endowed with open stomata, through which the lymph-ducts can communicate with the neighboring tissue and cells.

In rhachitis, the heart is of average size, but the arteries are abnormally large. Great width of the arteries lowers blood-pressure. This allows of the best explanation of the murmur first discovered by Fisher, of Boston, over the open fontanelles of rhachitical babies, a very much better one than that proposed by Jurasz, who looks for their cause in irregularities of the canalis caroticus. Still, it is a mistake to believe that these murmurs, audible over the brain, belong to rhachitis only. They are found in every condition in which the blood-pressure in the large arteries of the cranial cavity is lessened.

E. Hoffmann discovered the peculiar fact that the arterial pressure is very small in the newly-born animal. Even as large arteries as the carotid, when cut, do not spurt as in the adult. This low arterial pressure is one of the reasons why cords not ligated will often not bleed, with the exception of those cases in which the arterial pressure is increased by a moderate degree of asphyxia, or when the lungs are not inflated in consequence of incomplete development of the muscular strength in the prematurely-born fœtus.

According to a number of actual observations made by R. Thoma, the post-fœtal growth is relatively smallest in the common carotid, and largest in the renal and femoral arteries. Between these two extremes there are found the subclavian, aorta, and pulmonary arteries. These are differences which correspond with the differences in the growth of the several parts of the body supplied by those blood-vessels. In regard to the renal artery and the kidney, it has been found that the size of the former increases more rapidly than the volume and weight of the latter. Thus it ought to be expected that the frequency of congestive and inflammatory processes in the renal tissue will be almost predestined by this disproportion between the size of the artery and the condition of the tissue. Moreover, the resistance of the arterial current offered by the kidney-substance depends also upon the readiness with which the current is permitted to pass the capillaries. Now, it has been found experimentally that their permeability is greater, and that within a given time more water proportionately can be squeezed through them, in the adult, than in the child. This anatomical difference may therefore be the reason why renal diseases are so much more frequent in infancy and childhood from all causes, with the exception of that one which is reserved for the last decades of natural life, viz., atheromatous degeneration.

In the arteries of medium and small calibre the elastic membrane is a thin and simple membrane; it is only in larger arteries that elastic fibres will also extend into, and mix with, the adjoining layers. The elastic membrane is particularly thin, may even be entirely absent, where the branches are given off from the arteries. It is here that spontaneous hemorrhages are most apt to take place. It is here also that, in later life, aneurisms are met with, such as find no ready explanation by an injury.

The anatomical structure of the three umbilical vessels differs from that of all the rest of either arteries or veins in many points, principally in this, that there is no elastic membrane and no intima in the arteries. Some elastic tissue is found near the umbilicus, and it gradually increases in the abdominal cavity; but the intima is not developed in the arteries until they are in close proximity to the iliac. Thus by the massive and powerful development of the muscular layer it is explained why there are so few hemorrhages though no ligature have been applied to the cord.

The umbilical vein differs from the arteries very much less than is usual with veins and arteries in any other parts of the body. The muscular layer is very large and strong in the vein. There is no intima. None of the three vessels emits branches; there are no vasa vasorum and no nerves in their walls.

Altogether, the growth of the internal organs and the whole body does not proceed uniformly. In this respect the blood-vessels do not stand alone. What Beneke called the morbid disposition of the several ages, is best explained by these variations in growth and power. That author spent much time and labor on the measuring of blood-vessels in particular. It was he that found the arteries proportionately wide until the period of puberty. From that time the heart increases rapidly, and the arteries less. In infancy the relation of the volume of the heart to the width of the ascending aorta is 25 : 20, before puberty 140 : 56, and after puberty 290 : 61. Thus it is that the general arterial blood-pressure of infants is less and the heart-beats are more frequent.

After birth the pulmonary artery is much larger than the aorta; after the first year the width of the former compared with that of the latter is 46 : 40, in the adult 35.9 : 36.2, in advanced age 38.2 : 40.4. It is easily understood to what extent both the normal development and the diseases of the lungs may be influenced by these relative sizes of the vessels. That the size and strength of the right heart should have a favorable influence on the course of a pneumonia is an inference deserving of credit.

The reverse of the normal oversize of blood-vessels in the infant and child is found in abnormal smallness, particularly of the arteries. The worst, and mostly incurable, forms of chlorosis are the results of this anomaly. They have been studied by Trousseau, Virchow, Sée, and others, in connection with a small, or normal, or fatty heart, and in their complications with occasional hemorrhagic diathesis. All forms of persistent anæmia may depend on this insufficient development of the arteries: the specimens taken from a woman of thirty-two years, who died with all the symptoms of "essential" anæmia, are in my possession.

To the consideration of the organs of circulation I have given so much prominence because of their pre-eminent influence in etiology. The changes of periods of life and advancing age are mainly occasioned by the alterations in the structure of the walls of the blood-vessels. Their original thinness and fragility occasion hemorrhages in the newly-born, as does their anomalous condition in senility. Nor is there any organ which is not constantly under the control of the blood-current. This chapter would, however, grow to undue length, and encroach too much upon the legitimate province of the special essays devoted to the consideration of the subjects to which I could only allude, were I to continue to enlarge upon them. A few more remarks, therefore, may suffice.

There are anomalies and diseases which are met with in the infant and child only. Among this class we meet congenital diseases and malformations, the affections of the umbilical cord, of the ductus arteriosus, and of the tunica vaginalis of the spermatic cord, atelectasis and cyanosis, the diseases of the thymus, the anomalies of the intestinal tract, congenital constipation, as I have called it, resulting from the exaggeration of the normal length of the long sigmoid flexure, and, finally, rhachitis.

Other diseases are mostly found in children, or with a characteristic symptomatology and course. Both acute and chronic hydrocephalus, acute eruptive diseases, whooping-cough, and diphtheria are mostly found at an early age. Diphtheria is very liable to assume different characters in different ages; even the simple inflammations of the tonsils vary in severity and nature according to the amount of tissue destroyed or new hyperplastic connective tissue formed in the course of repeated attacks. Almost all the diseases of the intestinal tract in children have their peculiarities, and require the special study of foods and hygiene. The majority of cases of intussusception take place in infants, in localities and with symptoms of their own.

There are diseases which affect both the young and the old: in them the size or nature of the organ, or the difference in the degree of irritability, affect the symptomatology of the case considerably. In the narrow larynx of the child, diphtheria gives rise to the complex symptoms of pseudo-membranous croup. Tracheotomy and intubation are subjects eminently belonging to pediatrics. In the vulnerable infant only, intestinal worms will give rise to convulsions; and the large majority of cases of poliomyelitis and polioencephalitis also are reserved for infancy; indeed, so great is the difference between the ages, that the infant is the proprietor of the medio-canellata, while the adult glories in the tænia solium as a tenant. Let me add that there are differences of many degrees in many other diseases, accordingly as they occur in the young or in the old. The pneumonia, tuberculosis, typhoid fever, rheumatism, epilepsy, and diabetes of the young differ considerably from the same affections of the adult, in their clinical and, sometimes, anatomical aspects.

Therapeutics of infancy and childhood are by no means so similar to those of the adult that the rules of the latter can simply be adapted to the former by reducing doses. The differences are many. Among the antifebriles cold is tolerated less, quinia more, in proportion, than in the adult. So are antipyrin and antifebrin, also phenacetin. Heart-stimulants are also borne in relatively large doses: thus, digitalis, strophanthus, and sparteine. Caffeine is less advisable except where there is positively no cerebral complication of a congestive or inflammatory nature. Of the narcotics, opium must be watched; its doses must be relatively small. Belladonna is borne in rather large doses, and hyoscyamus can be given in much larger doses proportionately in spasmodic conditions of the bladder than in advanced age. Some of the powerful medicines are required in smaller, some in larger doses. Chlorate of potassium demands great care; carbolic acid becomes poisonous in small doses given to the very young, even externally; preparations of arsenic are borne in rather larger doses for many weeks and months; corrosive sublimate—mercurials generally—in rather large doses, because of the extraordinary immunity in regard to stomatitis and to the gastric and intestinal irritation so often observed in the adult.

Now, what has been done to facilitate the acquisition of knowledge on all these points by the student and practitioner of medicine? Very little indeed. There never was any systematic instruction in the diseases of children, by a teacher appointed for that branch of medicine exclusively, until (in 1860) I established a weekly children's clinic in the New York Medical College, at that time in East Thirteenth Street. That was the first of its kind in the United States. When the college ceased to exist (in 1865) I established a children's clinic in the University Medical College, and in 1870 in the College of Physicians and Surgeons. In both these institutions, as also in the Bellevue Hospital Medical College, such clinics have existed since, and a number of the medical schools of the country have imitated the example.

In them, a single hour weekly, during the regular courses of the winter, is given to the student of medicine for the special study of the diseases of children, who will, in his future practice, form the majority of his patients. In the course of two so-called years, which the legislatures of our States pronounce sufficient for the attainment of all medical knowledge required for the welfare of the country, the student is pressed very hard for time. There are a number of branches which he is taught to deem worth his while and attention, by being told that he will be examined in them before obtaining his diploma; but the diseases of children are not among these. To my knowledge, there is no school in the country which lays the least stress on that branch of instruction; for I hope there is nobody nowadays, even among the teachers of medicine, who believes that a few didactic lectures of the professor of "theory and practice" are a sufficient preparation for the preservation of the children of the people. No examination being required by those to whom the student looks for direction and enlightenment, he neglects the study, to find out too late the mistake he has made in so doing.

It is no consolation that in Great Britain the same complaints are made. But a few months ago the chairman of the Section of Diseases of Children, Dr. Cheadle, spoke in feeling terms of the neglect in the schools and clinical institutions of Great Britain of this most important part of practical medicine, before the British Medical Association. The continent of Europe has made more rapid progress. Most of both the large and the small universities have their chair of the Diseases of Children, not a "clinical" one, which means the authority given an enthusiastic worker to teach as much or as little as he can in an hour weekly, without recognition, thanks, or reward, of a doctrine not officially recognized; they have hospitals in which to teach practically every day what has been taught in didactic lectures and learned from books, and their students know beforehand that they will have to prove, before being permitted to practise, their acquaintance with what they are *compelled* to learn of the diseases of children. Thus it is in France and Italy, in Germany, Austria, and Sweden; thus it is now in Russia, but not so in England and in our country.

What can be done to improve this state of things?

Every future improvement in general medical education will favor study of pediatrics. There will be a time in the near future when student in medicine will be aware that he will have to pass an examinati in the subjects connected with the physiology and pathology of the you There will be another time when the medical courses will be both long a numerous enough to permit of clinical instruction in the diseases of child being given three or six times a week, and another in which there will bedside teaching. For that purpose it is that either special hospitals large wards in general hospitals are an absolute necessity. It is in th only that the student, and the professional man also, may learn under sup vision, and without the danger of each having to fill with victims a buryi ground of his own, both how to diagnosticate a disease in a child and h to nurse and treat a sick one. In hospitals alone can good observations made in reference to the course of diseases, and the effects of remedies a methods of treatment.

Moreover, special societies must be founded for the purpose of studyi questions connected with pediatrics, or special sections formed in larger a established associations. The new Children's Section in the New Yo Academy of Medicine, that of the American Medical Association, and t successful organization of the American Pediatric Society prove the inte sity of the interest the American profession has commenced to take in t subjects legitimately belonging to that part of medical science and practic

Finally, all of the latter, as well those to which I could but inco pletely allude, as all others suggesting themselves to the careful observ and thorough student, must be the themes of persistent individual stud Besides, as there must be time to learn other men's observations, so ti must be found to contribute what is new and valuable in every profession man's life. The basis on which to proceed is to be furnished by this Cycl pædia, the introductory remarks to which I am kindly permitted to off This book bids fair to contain all that is known at present on the anatom physiology, pathology, and therapeutics of infancy and childhood. May t American profession see to it that this same book, while being a digest bo of the labors of the past and the attainments of the present, shall beco the solid foundation of successful scientific work in the near and dista future.

PART I.

GENERAL SUBJECTS.

ON THE ANATOMY OF CHILDREN.

By GEORGE McCLELLAN, M.D.

Of the differences between the anatomy of children and that of adults it may be said, in a general way, that the bones are more elastic and less firm; the muscles softer, and less capable of great effort; the arteries, veins, lymphatic vessels, and nerves larger in proportion to the parts which they furnish, drain, or supply than they afterwards appear, and the thoracic and abdominal organs developed to a greater degree of perfection in comparison to the brain and the organs of generation.

In approaching this subject, however, it should be borne in mind that the modifications which occur before and after birth, at the separate periods of growth towards puberty, are so subtle that it is difficult to assert more than a limited number of general truths, and impossible to draw any reliable deduction, except by the special study of each period.

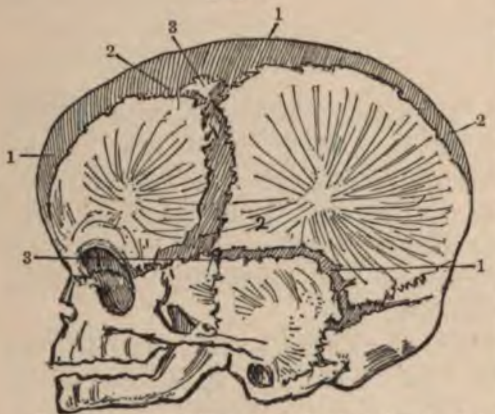
In addition, therefore, to describing the important anatomical variations which may from time to time be noted in each part of the body separately considered, in the long gamut of changes which slowly progress through infancy to manhood, the subject will be considered *regionally*, as it is believed that the comparative study of the relations which these parts bear to one another at different ages will give the most practical and useful knowledge to the reader.

THE HEAD.

The skin of the scalp in children is thicker than it is in any other part of the body; it is closely adherent to the aponeurosis of the subjacent muscle (the occipito-frontalis), and whenever that muscle moves, it moves with it. This mobility of the scalp is even more noticeable in infancy.

The pericranium is very slightly attached to the skull-bones, but at the sutures it is intimately blended with the membrane between the soft and growing bones of the young child's head; it is somewhat lax, and admits of limited extravasations of blood beneath it, which are usually congenital, and are due to pressure on the head at birth, when the bones are very vascular as well as soft. In the temporal region the pericranium is more adherent to the bone than anywhere else. Embryology shows that the vault of the skull is formed in membrane, and the base in cartilage; and pathology often makes the distinction more manifest. The bones of the vertex appear before those of the base, but at birth ossification is more advanced in the base. The occipital and sphenoid bones are united at their basilar portions about the twentieth year. The sutures usually become obliterated some time after the complete formation of the skull, but the time of their disappearance is extremely variable. The fontanelles, or the intermembranous spaces at the angles of the parietal bones, normally disappear before the age of four years. The anterior fontanelle, at the junction of the coronal and sagittal sutures, is the largest, and is usually closed at the end of the second year. Sometimes it persists throughout life; but these cases are generally due to hydrocephalus (see Fig. 1). The yielding nature of the young child's skull is such that it may be indented by a blow, or compressed by a bandage, or its shape may even be altered by the weight of the contained brain if the infant is habitually allowed to lie on one side. Supernumerary bones (ossa Wormiana), varying in size and number, are frequently found in the course of the sutures. Congenital fissures occasionally occur from the arrest of the ossifying process, and are liable to be mistaken for fractures. The frontal suture between the two fœtal portions of the frontal bone usually becomes obliterated shortly after birth, but may remain (see Fig. 2). All the hollows or sinuses are rudimentary at birth, and remain of small size up to about the ninth year, after which they gradually increase until puberty, when they undergo great enlargement,—notably the frontal, which is indicated in the adult skull by the prominence of the superciliary ridges. The thickest parts of the skull-cap are in those portions which originally were developed in cartilage.

FIG. 1.

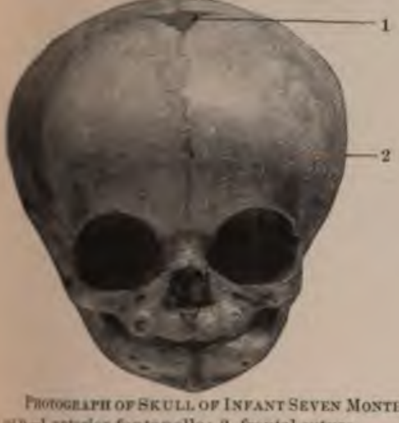


HYDROCEPHALIC SKULL OF AN INFANT, showing the sutures, 1, and fontanelles in exaggeration, 2; Wormian bones, 3.

THE BRAIN AND ITS MEMBRANES.

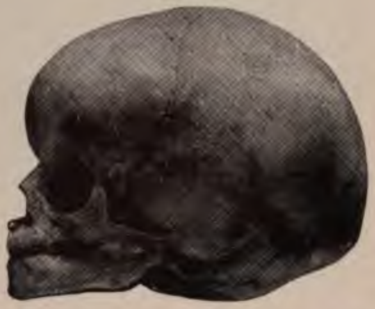
The dura mater in the young is adherent, and does not allow extravasations to collect between it and the bone. There is a greater amount of fluid in the subdural space in childhood than is usually found later in life,

Fig. 2.



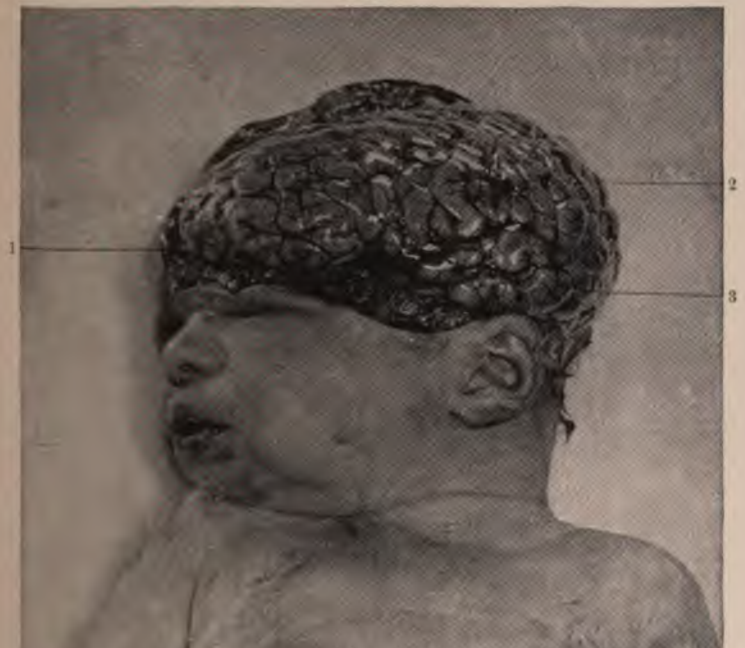
Photograph of Skull of Infant Seven Months old.—1, anterior fontanelle; 2, frontal suture.

Fig. 10.



Photograph of Skull of Infant Seven Months old, showing Proportionate Size of the Face to the Head.

Fig. 3.



Photograph from a Dissection, showing the Convolutions and Fissures of the Brain of the Infant.—1, fissure of Sylvius; 2, fissure of Rolando; 3, occipito-parietal fissure.

and the subarachnoidean space is always well filled. The foramen of Magendie, which is an opening in the pia mater at the inferior boundary of the fourth ventricle, may become impervious and give rise to hydrocephalus by an excessive accumulation of fluid within the ventricles (Hilton).

During intra-uterine life the development of the brain is very active, and at birth the organ is relatively of large size, presenting an approximation in form and relations to the adult brain (see Fig. 3). Up to the seventh year the brain grows rapidly, and increases slowly in weight after that period throughout life. The primary fissures of the cerebral hemispheres—viz., the Sylvian, hippocampal, parieto-occipital, and calcarine—appear during the third month of fœtal life. The secondary fissures, the most important being the fissure of Rolando, appear between the fifth and sixth months. The further development of the fissures, and consequently the convolutions, occupy the last two months of fœtal life, and the first five or six weeks after birth, at which time the cerebral surface can be clearly mapped out. Much attention has been given to the relation which the cerebral fissures and convolutions in the adult bear to the skull and scalp, on account of its practical importance in surgery. These investigations have recently been attended with remarkably accurate results; but very few have studied the relations between the fissures and the skull-sutures in children. The main peculiarities found in the latter are doubtless due to the contemporaneous development and growth of the surrounding parts. Symington, whose recent work in this department of topographical anatomy is most thorough and reliable, shows that the most important difference is the higher position in children of the fissure of Sylvius, in its relation to the spheno-parietal and squamous sutures (see Fig. 4). He found it, in numerous frozen sections of children of different ages under seven, always above the squamous suture and covered by the parietal bone. The author has verified this by dissections of children under three years of age, as in Fig. 3. Also see Fig. 5, a diagram from

FIG. 5.

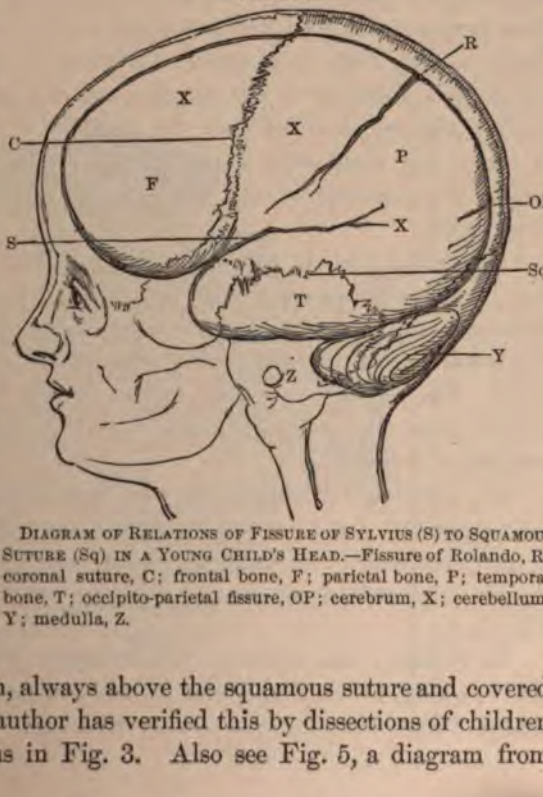


DIAGRAM OF RELATIONS OF FISSURE OF SYLVIUS (S) TO SQUAMOUS SUTURE (Sq) IN A YOUNG CHILD'S HEAD.—Fissure of Rolando, R; coronal suture, C; frontal bone, F; parietal bone, P; temporal bone, T; occipito-parietal fissure, OP; cerebrum, X; cerebellum, Y; medulla, Z.

dissections, as suggested by Symington. From careful study of his observations it is probable that the adult relations, where the squamous suture is said to correspond with the horizontal branch of the Sylvian fissure, are attained by the changes which occur in the growth of the skull, which tend to raise the squamous suture, and as the base of the skull increases in its breadth it is more likely to modify the form of the brain resting upon it, occasioning a descent of the Sylvian fissure.

The position of the fissure of Rolando in an infant six months old has been found to correspond with that of the adult (as in Fig. 3). Huschke has shown that the cerebellum is much smaller at birth as compared with the cerebrum, and that the latter overlaps the former as it does in the adult.

The convolutions of the brain in the child have a less complex arrangement than in the adult, and the sulci between them are less deep. The number and extent of the convolutions appear to bear close relation to the intellectual power of the individual at all ages.

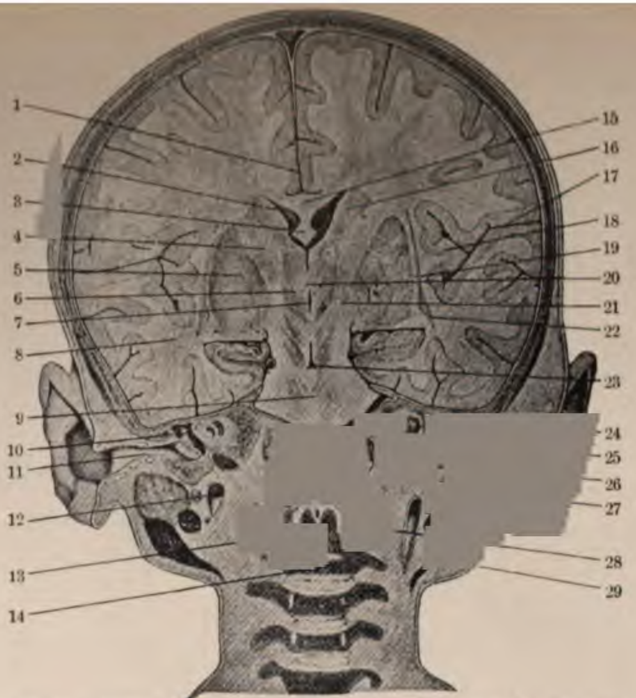
THE EYE AND ORBIT.

The first stage of the development of the eyes in the fœtus begins at a very early period, about the first week, and proceeds steadily towards perfection at the time of birth. There is a vascular tunic surrounding the lens, the front of which closes in the pupil, and is called the pupillary membrane. This becomes absorbed and atrophied in the human subject before birth. There is very little comparative variation in the structure of the child's eye and its relations to the muscles and nerves in the orbit (see Fig. 6). In fact, the eye, considered as the organ of vision, is probably as perfect in the young child as it ever is, only the power of perceiving requires further development of the brain-centre which presides over intelligent observation.

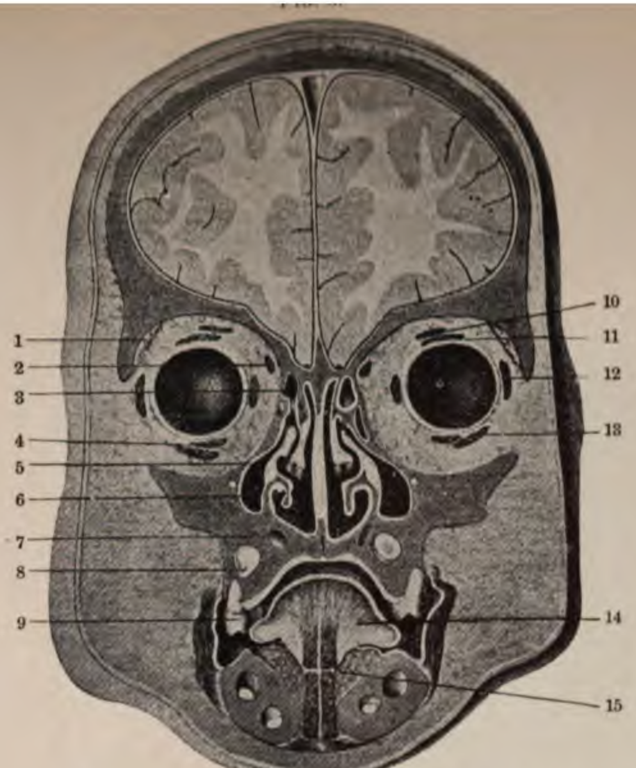
The orbital plate of the frontal bone is often incomplete at puberty, and may be deficient in bony matter throughout life. Fatal injuries have been frequently reported in consequence of children falling upon sharp objects which have penetrated the orbital plate at this weak point and entered the brain. The capsule of Tenon in the young is markedly attached to the recti muscles, and connects them with the globe of the eyeball and the margins of the orbit.

In operating for strabismus the capsule requires to be divided after the conjunctiva has been cut through, else the section of the muscle cannot be properly made. A quantity of fat occupies the orbit behind Tenon's capsule, and will accommodate foreign bodies of considerable size (see Fig. 7).

In children the sclerotic coat is somewhat transparent, and appears bluish, owing to the deeper hue of the subjacent choroid coat. The convexity of the cornea appears to vary with age.



VERTICAL SECTION (FROZEN) FROM A CHILD SIX YEARS OLD, BY SYMINGTON.—1, longitudinal fissure; 2, lateral ventricle; 3, fornix; 4, internal capsule; 5, nucleus lenticularis; 6, optic thalamus; 7, third ventricle; 8, temporo-sphenoidal lobe; 9, pons; 10, tympanic cavity; 11, external auditory meatus; 12, internal jugular vein; 13, odontoid process; 14, body of axis vertebra; 15, corpus callosum; 16, nucleus caudatus; 17, horizontal limb of fissure of Sylvius; 18, island of Reil; 19, external capsule; 20, middle commissure; 21, internal capsule; 22, claustrum; 23, posterior perforated space; 24, cochlea; 25, basilar artery; 26, internal carotid; 27, parotid gland; 28, lateral mass of atlas vertebra; 29, sternomastoid muscle.



VERTICAL SECTION (FROZEN) FROM A CHILD'S HEAD, SIX YEARS OLD, BY SYMINGTON.—1, lachrymal gland; 2, superior oblique muscle; 3, ethmoid sinus; 4, inferior rectus muscle; 5, infundibulum; 6, antrum of Highmore; 7, socket for permanent canine tooth; 8, second bicuspid; 9, second temporary molar; 10, levator palpebræ muscle; 11, superior rectus muscle; 12, external rectus; 13, inferior oblique; 14, tongue; 15, sublingual gland.

THE EAR.

The development of the ear in its several parts is very unequal, the structure of the internal ear and the tympanic cavity and auditory ossicles being fully formed at birth, while the external auditory meatus, Eustachian tube, and mastoid portion of the temporal bone undergo many modifications before their completion at puberty. The meatus in the new-born child consists of an external part, which is cartilaginous, and an internal part, which is osseo-fibrous, instead of completely osseous as in the adult. In the child, the osseous portion is relatively shorter than it subsequently becomes. The entire meatus is as long proportionately in the child as in the adult (see Fig. 4). At birth the meatus passes inward and inclines downward. The upper walls of the meatus are comparatively thin, and are apt to give way in abscess or bone-disease and occasion meningitis. The pain felt by children in eating when suffering with inflammation of the meatus is owing to the distribution of the auriculo-temporal nerve.

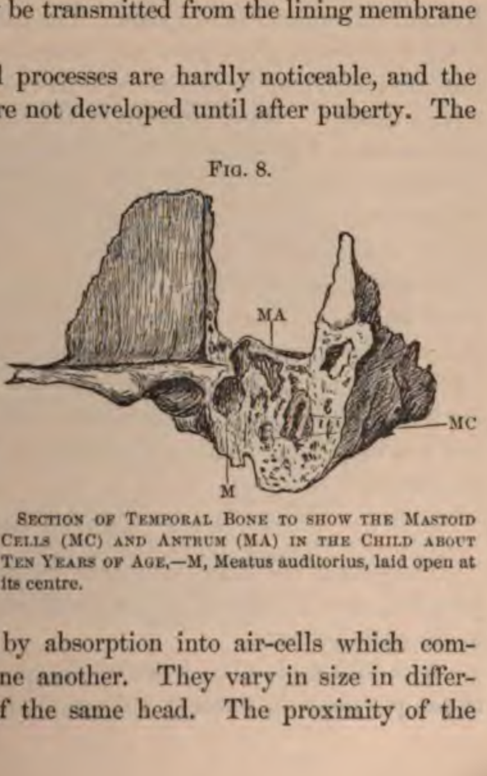
At birth the membrana tympani is nearly horizontal, but becomes more oblique as age increases.

The notch of Rivinus is the deficiency in the upper part of the bony ring in which the membrane is attached, and rupture may occur at this point from concussion. Suppurative disease of the middle ear may damage the chorda tympani nerve.

There is a petro-squamous suture in the roof of the tympanum in infancy by which inflammatory action may be transmitted from the lining membrane to the dura mater (Symington).

During childhood the mastoid processes are hardly noticeable, and the mastoid cells, although existing, are not developed until after puberty. The mastoid antrum is a large cavity in the mastoid part of the petro-mastoid bone, having, like the tympanic cavity, a thin roof separating it from the cranial cavity. From infancy to puberty there is a continuous formation of new bone from the periosteum on the surface, rendering the outer walls of the antrum thicker with age. It consists of cancellous tissue, and can be readily penetrated by a knife in mastoid disease (see Fig. 8). At puberty this cancellous tissue becomes hollowed by absorption into air-cells which communicate with the antrum and one another. They vary in size in different bodies and on the two sides of the same head. The proximity of the

FIG. 8.



SECTION OF TEMPORAL BONE TO SHOW THE MASTOID CELLS (MC) AND ANTRUM (MA) IN THE CHILD ABOUT TEN YEARS OF AGE.—M, Meatus auditorius, laid open at its centre.

lateral sinus renders it liable to become involved by extension of inflammation in suppurative disease of the mastoid cells, and especially so in the adult, owing to the thin bony septa which separate the cells from the sinus.

The Eustachian tube is at first horizontal, but becomes gradually directed downward. The tympanic orifice to the tube is as large in the child as in later life, but the opening from it into the pharynx is smaller. There is scarcely any osseous tissue in the composition of the Eustachian tube at birth, and even in old age it is still principally cartilaginous (see Fig. 9).

Arnold's nerve supplies the back of the concha near the mastoid process, and its connection with the pneumogastric has been shown by the production of coughing and vomiting in consequence of irritation by foreign bodies having been introduced by children into the ears in play.

THE NOSE.

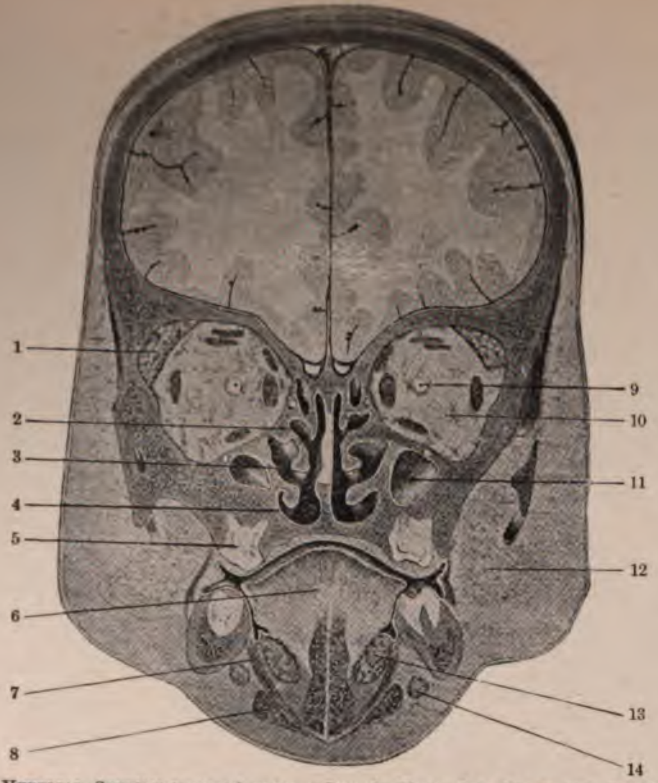
The first depression which indicates the nose in the early embryo occurs about the fourth week. Its formation continues with the growth of the face, and it is of relatively small size in the young child. The centre of the nasal fossæ is the widest part, and foreign bodies are often retained there for long periods of time. At all ages the fossæ are very narrow above the lower border of the middle turbinated bones. The septum of the nose is usually straight up to the seventh year; after that it very commonly inclines to one side. The infundibulum connects the antrum of Highmore with the frontal sinus (see Fig. 6). When there is a direct passage into the middle meatus it is not so common as supposed. The suture between the nasal and frontal bones sometimes gives place to a protrusion,—*i.e.*, meningocele or encephalocele,—which may be mistaken for nævoid growths such as frequently occur at this locality. Severe catarrh of the nasal mucous membrane in infancy may occasion depression of the bridge of the nose. A coryza may be extended by continuity of structure to the Eustachian tube, lachrymal sac, and conjunctiva, and even the frontal sinus and antrum. In children there is almost always a communication between the nasal veins and the superior longitudinal sinus through the foramen cæcum. This is usually closed about puberty. There is a venous plexus about the inferior turbinated bone, and the paroxysms of whooping-cough often bring on epistaxis through interference with the venous circulation of the nose. The anterior part of the nose is supplied by the nasal nerve, which is a branch of the ophthalmic, and hence the lachrymation which follows the introduction of irritants into the nostrils.

THE FACE.

The skin of the face is everywhere very thin, having loose cellular tissue excepting over the alæ of the nose and the chin, where it is closely adherent to the parts beneath.

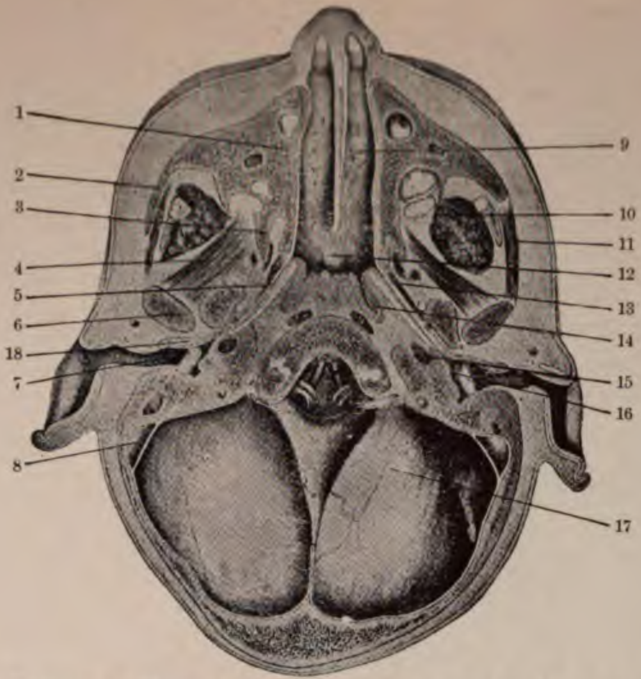
A careful study of the face in young children will prove of great value to the physician in forming a diagnosis in many diseases peculiar to them.

FIG. 7.



VERTICAL SECTION (FROZEN) FROM A CHILD'S HEAD, SIX YEARS OLD, BEHIND FIG. 4, BY SYMINGTON.—1, lachrymal gland; 2, superior meatus; 3, middle meatus; 4, inferior meatus; 5, first permanent molar; 6, tongue; 7, mylo-hyoid muscle; 8, anterior belly of digastric; 9, optic nerve; 10, capsule of Tenon; 11, antrum of Highmore; 12, sucking cushion; 13, sublingual gland; 14, submaxillary gland.

FIG. 9.



HORIZONTAL SECTION (FROZEN) FROM A CHILD'S HEAD, SIX YEARS OLD, BY SYMINGTON.—1, upper jaw; 2, zygomatic arch; 3, external pterygoid plate; 4, external pterygoid muscle; 5, Eustachian tube; 6, condyle of lower jaw; 7, external auditory meatus; 8, lateral sinus; 9, floor of nose; 10, temporal muscle; 11, masseter muscle; 12, pharynx; 13, tensor palati muscle; 14, fossa of Rosenmüller; 15, internal carotid artery; 16, tympanic cavity; 17, posterior fossa for the cerebellum; 18, tympanic bone.

An infant is unable to communicate ideas by speech, and the only way of obtaining information is by carefully noticing the expression and gestures. The particular locality of pain is often ascertained by this means. Contraction of the brows indicates pain in the head; sharpening of the nostrils, pain in the chest; and drawing of the upper lip, pain in the abdominal region.

M. Jadelot long ago pointed out certain furrows or lines which become marked in the face of a child suffering from serious diseases. According to his view, the oculo-zygomatic line, which begins at the inner corner of the eye and passes outward towards the cheek-bone beneath the lower lid, shows disorder of the cerebro-nervous system. The nasal line, passing from the ala of the nose round the corner of the mouth, points to disorder of the digestive tract, and especially if associated with marked dimpling of the cheek. The labial line, extending from the angle of the mouth to the lower part of the face, is a sign of diseases of the respiratory organs.

There is much fat in the subcutaneous tissue, especially in the cheeks, in children. Over the buccinator muscles, in addition to the ordinary subcutaneous layer of fat, there is an arrangement of fatty lobules surrounded by a capsule on either side. These have been called "sucking cushions," because they are thought to be instrumental in distributing the atmospheric pressure, and prevent the buccinator muscles being pressed inward between the alveolar arches when a vacuum is created in the mouth. These sucking pads are best developed in infants, but may be found at all periods of life (see Fig. 7). They are even present when the other fat in this region is absorbed during the wasting diseases of childhood. The bony walls of the cheeks in the young are thicker than in the adult, because the maxillary sinuses, or antra of Highmore, are hollowed out, chiefly by absorption.

The face is frequently the seat of nævi, owing to the great vascularity of its tissues. Injections of nævi in infancy may occasion thrombosis, which has in some instances proved fatal, owing to the direct communication between the facial vein and the internal jugular. Mumps may give rise to cerebral hyperæmia through pressure upon the internal jugular vein.

The lips are very vascular, and are often the seat of vascular tumors. The branches of the coronary arteries run close to the mucous lining of the mouth beneath the outer-lying muscles, and can be readily felt pulsating by pressing the lip from within. There are many submucous glands about the lips, which are supposed to cause enlarged lip by becoming hypertrophied.

THE JAWS AND TEETH.

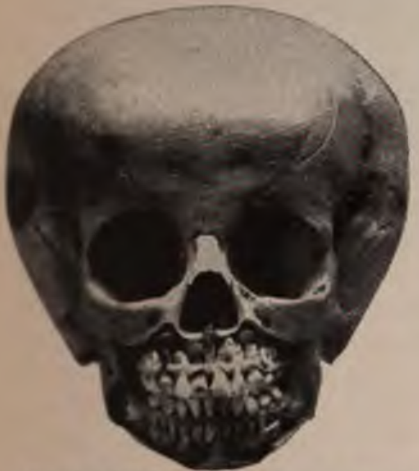
The small size of the facial portion of the skull at birth and during early childhood is due to the rudimentary condition of the jaws and teeth (see Fig. 10). The upper and lower maxillary bones commence to ossify at a very early period, the lower one first. They are developed very slowly, and undergo various modifications, depending mainly upon the eruption of the teeth, until their complete form is attained at puberty. The gums are composed of a dense fibrous tissue, covered by a vascular mucous membrane of very slight sensibility. They form a tough protecting covering to the developing teeth until the eruption of the latter. They are closely connected to the periosteum of the alveolar processes and surround the necks of the teeth. The gums in new-born children do not meet, and until the further development of the alveolar arches and teeth there is always a separation between them. There are two sets of teeth, both of which appear at different periods during childhood,—the first, called the temporary, giving way to the second, called the permanent. The development of the temporary teeth in the fœtus begins with the first formation of the jaws, about the seventh week. The teeth are ultimately simply calcified mucous membrane. The stages of their development have been more carefully studied than perhaps any other portion of the body in this important period of life. Briefly stated, the process may be summed up as follows:

The primitive dental groove is caused by a turning inward or depression of the oral epithelium, forming a furrow in the edges of the jaws, from the bottom of which a vascular ridge of papillæ springs up contemporaneously. Each of these papillæ gradually assumes the shape of a future tooth and is covered with a cap of epithelial cells, which undergo a differentiation so as to form the dentine, the enamel, and the cement. The changes which take place in the bones of the jaws relate only to the formation of the sockets of the teeth. At first there is no appearance of alveoli, but as the changes occur in the mucous membrane by which the teeth are developed, there is also a groove formed in the jaw itself, which by degrees becomes wider and is divided across by thin bony partitions. The edges of the alveoli are turned towards one another shortly after birth, so as to protect the developing temporary teeth from injury.

The germs of the temporary teeth make their appearance from the seventh to the twelfth week of embryonic life. They are not set vertically opposite one another in the two dental arches, the upper jaw teeth being in front of the lower. They are very imperfectly developed at birth, and are only fully formed about the age of four and a half years. They number twenty,—four incisors, two canine, and four molars in each jaw. Their periods of eruption after birth are, approximatively, the central incisors about the seventh month, the lateral incisors from the eighth to the tenth month, the anterior molars from the twelfth to the eighteenth month, the canine from the fourteenth to the twentieth month, the posterior molars from the eighteenth to the thirty-sixth month. The lower teeth generally precede the upper ones.

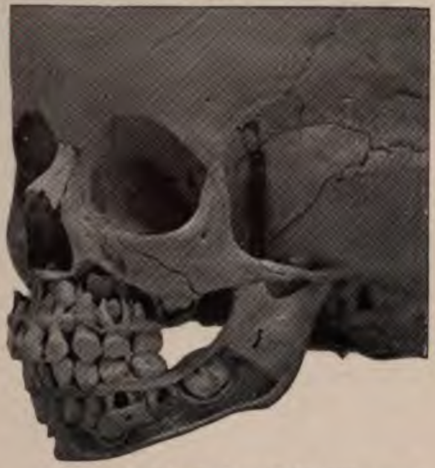
The permanent teeth consist of two groups,—those which have, and those which have not, predecessors. To the former group belong the incisors, canines, and bicuspids; to the latter, the molars. The incisors, canines, and bicuspids directly succeed to the positions occupied by the temporary teeth, and correspond in number to them. The molars, three in number, on either side of either jaw, are the additional permanent teeth. The development of the first group of the permanent teeth is effected in a manner

Fig. 11.



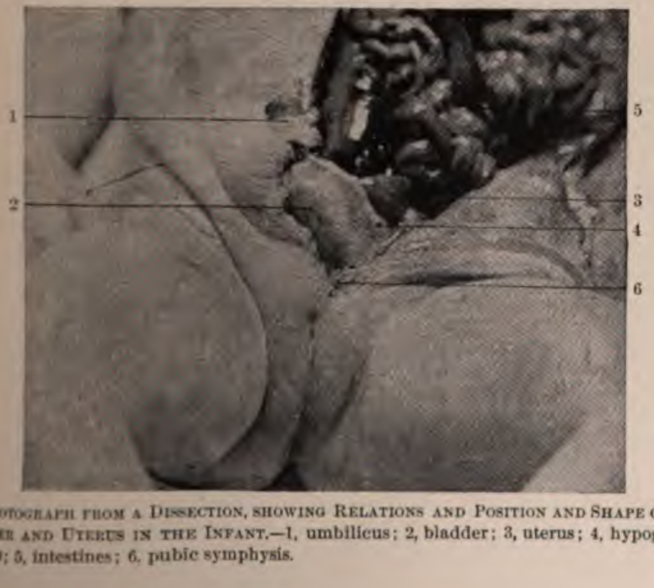
Skull of Child Seven Years old, showing Permanent and Temporary Sets of Teeth.

Fig. 12.



Jaws of Child about Seven Years old, showing Temporary and Permanent Sets of Teeth, except the Wisdom.

Fig. 29.



Photograph from a Dissection, showing Relations and Position and Shape of the Bladder and Uterus in the Infant.—1, umbilicus; 2, bladder; 3, uterus; 4, hypogastric arteries; 5, intestines; 6, pubic symphysis.

analogous to that of the temporary teeth, having a second dental furrow formed out of the epithelial lining of the gums and vascular papillæ. In the process, the sac which encloses each tooth-germ becomes attached to the back of the sac of a temporary tooth. The three additional permanent teeth, the molars, are developed by successive prolongations of the epithelial tissue towards the angles of the jaws. The calcification of the permanent teeth extends from before birth to about the twelfth year of life. The fangs, or roots, of the temporary teeth disappear by absorption as the permanent teeth become developed, and the loose crowns gradually become detached, giving place to the new-comers. The eruption of the permanent teeth takes place usually as follows: the first molars at six and a half years, the middle incisors in the seventh year, the lateral incisors in the eighth year, the first bicuspid in the ninth year, the second bicuspid in the tenth year, the canine about the twelfth year, the second molars from the twelfth to the thirteenth year, the third molars (wisdom-teeth) from the eighteenth to the twenty-first year, or later. The lower jaw teeth precede the upper jaw teeth, as in the temporary set.

About the sixth year of age, before the temporary incisors are shed, the jaws contain all the temporary and permanent teeth, except the wisdom-teeth (see Figs. 11 and 12).

During the growth of the teeth, the lower jaw increases in depth and length, and changes its form. At birth this bone consists of two lateral halves united by fibro-cartilage. The body is a mere shell of bone, and the angle of the jaw is obtuse. About the first year the two halves become jointed at the symphysis. The jaw becomes gradually elongated behind the mental foramen, so as to accommodate the three extra permanent molars. The angle also steadily becomes less obtuse until adult age is reached, when it is nearly a right angle. In old age it becomes again obtuse. The difference in width between the incisors of the temporary and permanent sets is compensated for by the smallness of the bicuspids in comparison with the temporary molars to which they succeed.

THE TONGUE.

The tongue is rarely the seat of congenital defects, and "tied tongue," or contraction of the frænum linguæ, is not so often met with among children as commonly supposed. The ranine vessels occupy the position of the elevated folds of mucous membrane on the under surface of the tongue converging at its tip. The artery is more deep than the vein on either side (see Figs. 6 and 7). If the frænum and subjacent muscular fibres be too freely divided in operating for tongue-tie, there is danger of the child, in its efforts at sucking, tearing these lax fibres farther open, so that the tongue may be forced down upon the epiglottis by the muscles of deglutition and occasion suffocation.

THE PALATE.

The arch of the hard palate varies in height and shape in diffe individuals, and, according to Treves, it is particularly narrow and high congenital idiots. Cleft palate is a congenital defect in the middle line, may involve the soft palate or uvula, extending sometimes forward to alveolus. In front of this the cleft occurs at the suture between the up maxillary and intermaxillary bones.

Hare-lip is a fissure in the upper lip opposite this suture. It may double, occurring on both sides of the os incisivum,—this bone appear to be attached to the septum of the nose. The suture referred to is o noticeable in very early life, and the so-called inter- or pre-maxillary b does not exist in the human subject, except upon the occurrence of congeni defects at these sutures. Most commonly hare-lip is uncomplicated w cleft palate. Hare-lip and cleft palate are due to the imperfect closure the fœtal gaps in these situations. The mucous membrane covering t hard palate cannot be separated from the periosteum; it is thickened at t alveoli. The descending palatine branch of the internal maxillary arte supplies the tissues of the hard palate. It comes through the posteri palatine canal at the side of the last molar tooth and runs forward to t anterior palatine canal lying close to the bone. A cleft in the soft palate widened by the action of the levator and tensor palati muscles.

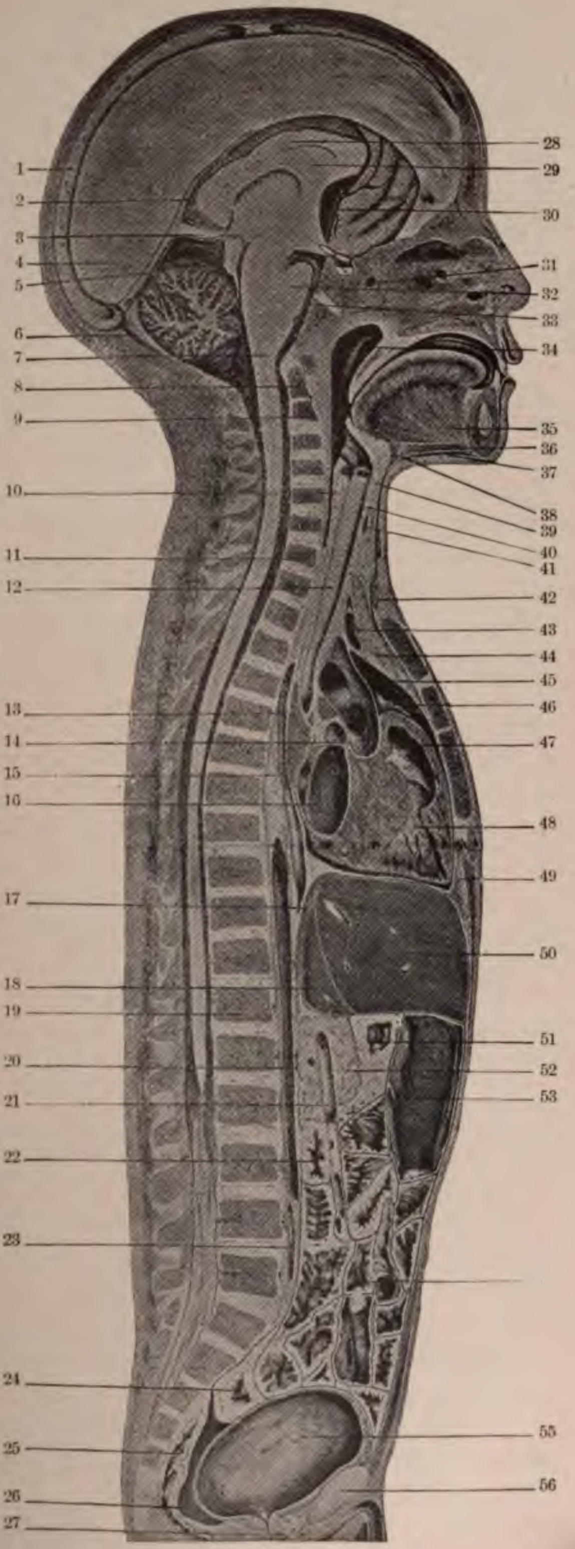
THE PHARYNX.

The pharynx is always widest near the hyoid bone and narrowest opp site the cricoid cartilage. A finger can easily reach the latter part in t child, where foreign bodies are apt to lodge. The connective tissue betwe the pharynx and the spine is very lax. The internal carotid artery and t pneumogastric, glosso-pharyngeal, and hypoglossal nerves are in relation t the walls of the pharynx on either side (see Fig. 9). The artery can b felt by the finger introduced through the mouth.

THE TONSILS.

The tonsils are situated between the palatine arches opposite the angle of the lower jaw, and when enlarged project into the cavity of the pharynx and cannot so readily be felt externally as commonly supposed. The tonsils are moved inward by the superior constrictor and outward by the stylo-pharyngei muscles. A child with prominent anterior arches of the palat and vigorous muscles of deglutition offers great difficulty to the operator i removal of the tonsils. The orifice of the Eustachian tube may be blocke by interference with the function of the tensor palati muscles, but not b pressure of an hypertrophied tonsil. The tonsils derive their blood fro the branches from the facial artery, mainly. The internal carotid is no in immediate relation with this gland, as usually described. In the chil it is comparatively out of the way, and is always behind it. The ascendin

Fig. 13.



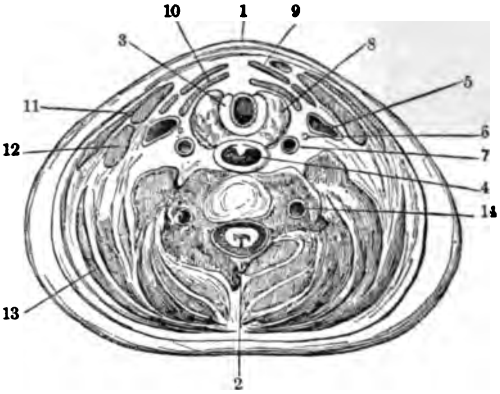
Longitudinal Section (frozen) from a Child about Six Years of Age, by Symington.—1, superior longitudinal sinus; 2, inferior longitudinal sinus; 3, veins of Galen; 4, falx major; 5, straight sinus; 6, falx minor; 7, medulla; 8, odontoid process; 9, body of atlas vertebra; 10, cricoid cartilage; 11, seventh cervical vertebra; 12, trachea; 13, lymphatic glands; 14, right pulmonary artery; 15, œsophagus; 16, left auricle; 17, diaphragm; 18, lobulus Spigelii; 19, twelfth dorsal vertebra; 20, aorta; 21, superior mesenteric vein; 22, third part of duodenum; 23, left iliac vein; 24, sigmoid flexure; 25, rectum; 26, recto-vesical pouch of peritoneum; 27, perineum; 28, corpus callosum; 29, septum lucidum; 30, anterior cerebral artery; 31, sphenoidal sinus; 32, pons Varolii; 33, basilar artery; 34, Eustachian tube; 35, genio-hyoglossus; 36, genio-hyoid; 37, mylohyoid; 38, hyoid bone; 39, thyroid cartilage; 40, cricoid cartilage; 41, isthmus of thyroid gland; 42, anterior jugular vein; 43, left innominate vein; 44, thymus gland; 45, pericardial cavity; 46, aorta; 47, conus arteriosus; 48, right ventricle; 49, ensiform cartilage; 50, left lobe of liver; 51, pylorus; 52, pancreas; 53, transverse colon; 54, small intestine; 55, bladder; 56, symphysis pubis.

pharyngeal artery is in close relation with the tonsil, and severe hemorrhage caused by wounds of this vessel has often been attributed to injury of the internal carotid artery.

THE NECK.

The anatomy of this region in childhood varies somewhat from that of the adult, and there is generally more subcutaneous fat and greater laxity of connective tissue. The assertion that the neck is very short in new-born children is probably due to the slight development of the face. The size of the various parts is commensurate with the stature of the individual, and in considering this region it should be remembered that the larynx is connected with the tongue and hyoid bone, and that their position is associated with that of the jaws. The larynx increases in size and grows downward, as does the face, from birth to puberty. Careful observations, made by Symington and others, show that there is very little difference in size in the two sexes during childhood. As boys approach puberty, it is well known that there is a very marked increase in the size of the larynx. The neck is limited in front by the symphysis of the lower jaw and the top of the manubrium sterni; and it is important to note that the latter is higher in relation to the vertebral column in the child than in the adult. In surveying the relations and taking measurements for surgical purposes by the finger-breadth, it is well to employ the finger of the patient in question rather than that of the observer.

FIG. 14.



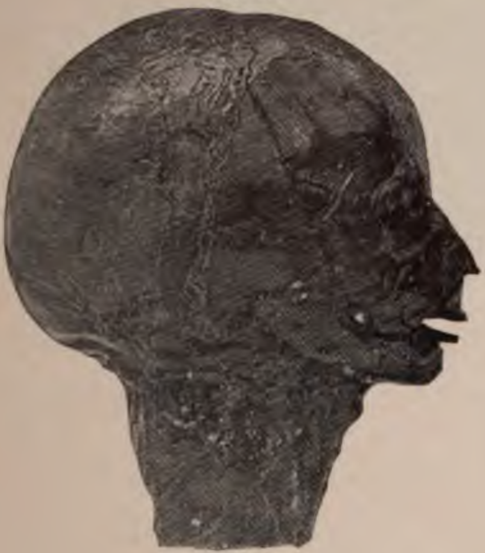
TRANSVERSE SECTION OF THE NECK OF A CHILD ABOUT SIX YEARS OF AGE, SHOWING RELATIONS OF THE PARTS TO THE CRICOID CARTILAGE IN FRONT AND TO THE SIXTH CERVICAL VERTEBRA BEHIND.—1, middle line; 2, sixth cervical vertebra; 3, cricoid cartilage; 4, pharynx; 5, internal jugular vein; 6, pneumogastric nerve; 7, carotid artery; 8, thyroid body; 9, sterno-hyoid muscle; 10, sterno-thyroid muscle; 11, platysma; 12, sterno-mastoid; 13, trapezius; 14, vertebral artery. (By the author.)

The cricoid cartilage is always prominent in infancy and old age, in fat as well as lean subjects, and is, when the head is upright, about opposite the fifth cervical vertebra (see Figs. 13 and 14). When the head is stretched so as fully to extend the neck, it is raised opposite the fourth vertebra; when the neck is flexed acutely, it corresponds to the sixth vertebra, being depressed. The cricoid cartilage is the most reliable landmark in the neck. A line drawn across from the cricoid cartilage to the fifth cervical vertebra will indicate the position of the top of the gullet, and also the point where the common carotid artery is crossed by the omo-hyoid muscle. The cartilages of the larynx are small and insignificant in the child and easily compressible, but towards puberty they are well developed, and notably so in the male, notch in the top of the thyroid cartilage becoming prominent (the pom Adami). The thyro-hyoid and crico-thyroid membranes are respectiv about as wide as the breadth of the child's finger. The upper rings of trachea can be detected only in very thin subjects, and then by stretchi the head backward over a pillow or block so as to distend and force upwa the neck. By the latter procedure, however, the innominate artery a veins are pulled upward into the supra-sternal notch. The middle li here, as elsewhere in the body, is regarded surgically as the line of safe owing to the feeble arterial anastomosis from side to side. About t breadth of a finger from the cricoid cartilage is the isthmus of the thyro body, and below it are the inferior thyroid veins. Occasionally short tran verse connecting links between the two anterior jugular veins exist also the middle line. The only artery is the crico-thyroid, which passes alo the lower border of the thyroid cartilage over the crico-thyroid membran There is little danger from interfering with either the latter or the isthm in tracheotomy, but much trouble may arise from the venous links or i ferior thyroid veins above mentioned (see Fig. 15). It is difficult to mak an injection travel across the isthmus of the thyroid gland. In little chi dren the neck is usually very fat, and the great difficulty in tracheotom is in fixing the trachea in the middle line, owing to its depth and mobility The calibre of the trachea may be said, in a general way, to correspond t the size of the patient's forefinger. The trachea naturally descends with th downward growth of the larynx, and we find in the new-born child that it bifurcation is about opposite the third dorsal vertebra, while in the adult i is opposite the fourth. The isthmus of the thyroid gland is usually very small in children, and it is connected by strong fibrous tissue to the sub jacent cartilage.

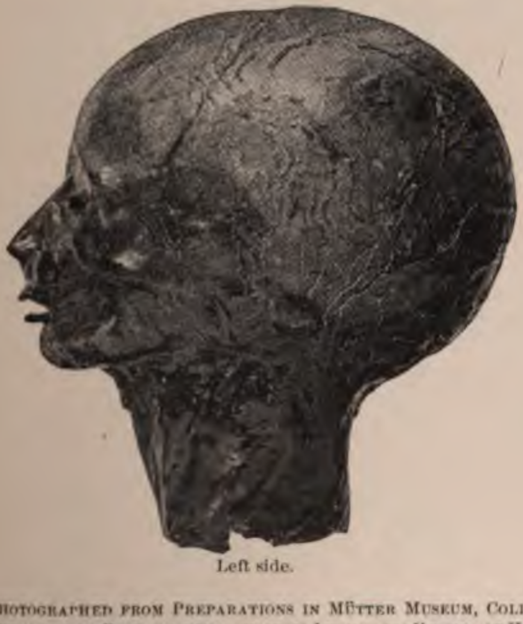
The condition known as wry-neck, common in childhood, is produced by some congenital defect or spasmodic contraction of the sterno-mastoid muscle on one side or paralysis of the corresponding muscle on the opposite side. Holden has pointed out that the affection may also be due to the permanent contraction of the splenius on one side. The scaleni and trapezius muscles may be involved as well. The contraction is due to reflex irritation through the cervical plexus and its connection with the spinal accessory nerve. In tenotomy upon the sterno-mastoid muscle just above the clavicle it should be remembered that the anterior jugular and external jugular veins are respectively situated at the anterior and posterior borders of the muscle. Sometimes at birth a tumor is noticed in the sterno-mastoid muscle which has been attributed to tearing of its fibres in delivery. The deep layers of fascia in this region are more lax in infancy, and, although they form distinct sheaths for the various parts, they do not offer the same resistance to growths and abscesses as in later life, and not always then as definitely as has been supposed.

The lymphatic glands in the neck are very often enlarged and inflamed

FIG. 17.



Right side.

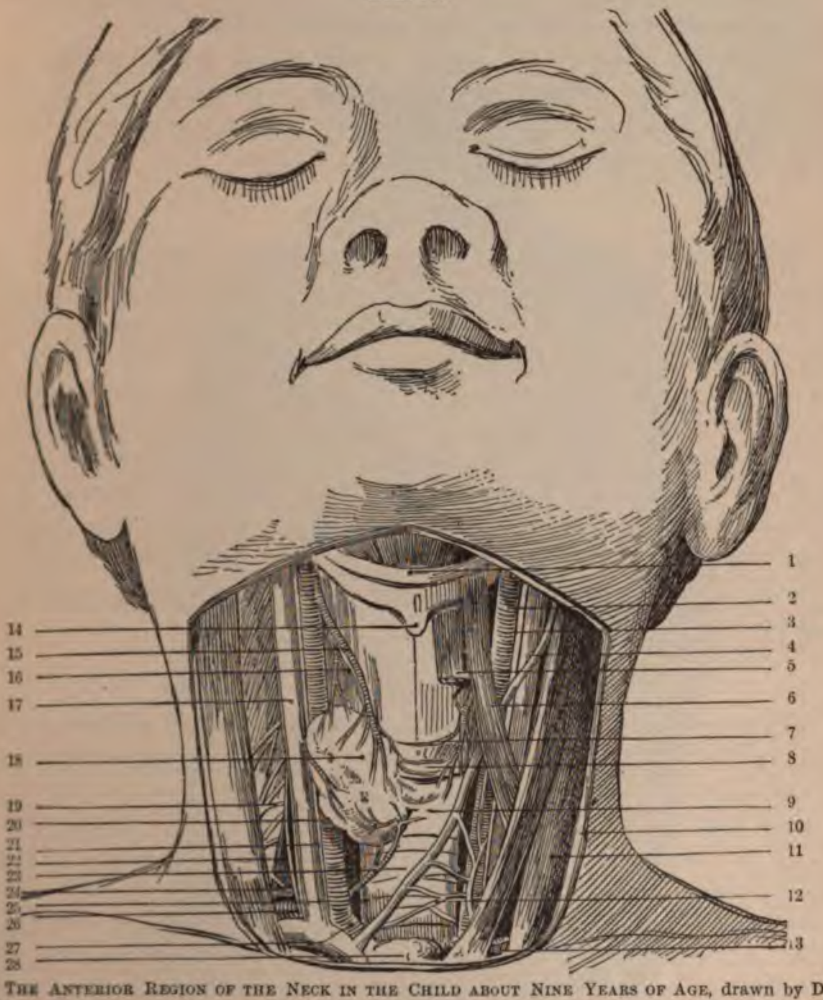


Left side.

PHOTOGRAPHED FROM PREPARATIONS IN MÜTTER MUSEUM, COLLEGE OF PHYSICIANS OF PHILADELPHIA, SHOWING LYMPHATIC VESSELS OF HEAD, FACE, AND UPPER PART OF THE NECK IN THE INFANT.

in scrofulous affections of children (see Figs. 16 and 17). They are numerous, and have been tabulated as follows:

FIG. 15.



THE ANTERIOR REGION OF THE NECK IN THE CHILD ABOUT NINE YEARS OF AGE, drawn by Dr. McClellan. The sterno-thyroid and hyoid muscles are removed, to show the deeper relations of the vessels and thyroid body.—1, hyoid bone; 2, left carotid artery, near its bifurcation; 3, left pneumogastric nerve; 4, left internal jugular vein; 5, upper portion of sterno-thyroid muscle; 6, omo-hyoid muscle; 7, crico-thyroid artery; 8, cricoid cartilage; 9, isthmus of thyroid body; 10, left external jugular vein; 11, sterno-mastoid muscle; 12, inferior thyroid veins; 13, lower end of sterno-thyroid muscle; 14, top of thyroid cartilage; 15, right superior thyroid artery; 16, superior laryngeal nerve; 17, right internal jugular vein; 18, right lobe of thyroid body; 19, cervical plexus of nerves; 20, right common carotid artery; 21, right pneumogastric nerve; 22, scalenus anticus muscle; 23, right inferior thyroid artery; 24, suprascapular vessels; 25, right recurrent laryngeal nerve; 26, right phrenic nerve; 27, innominate vessels; 28, remains of thymus gland in the supra-sternal notch.

"Submaxillary ten to fifteen, situated along the base of the jaw; suprahyoid one or two, between the chin and hyoid bone; superficial cervical four to six, along the external jugular beneath the platysma muscle; deep cervical ten to twenty, about the bifurcation of the carotid artery and upper part of the internal jugular; lower deep cervical ten to sixteen, about the lower part of the internal jugular, extending to the supraclavicular fossa and continuous with the axillary and mediastinal glands."

There are congenital fistulæ occurring occasionally which are due to persistence of the so-called branchial clefts of fœtal life.

There is a tendency for foreign bodies which descend through the trachea to pass into the right bronchus, owing to the bronchus of that side being larger than that of the left, and to there being a septum at the bottom of the trachea occupying the left of the median line.

THE ŒSOPHAGUS.

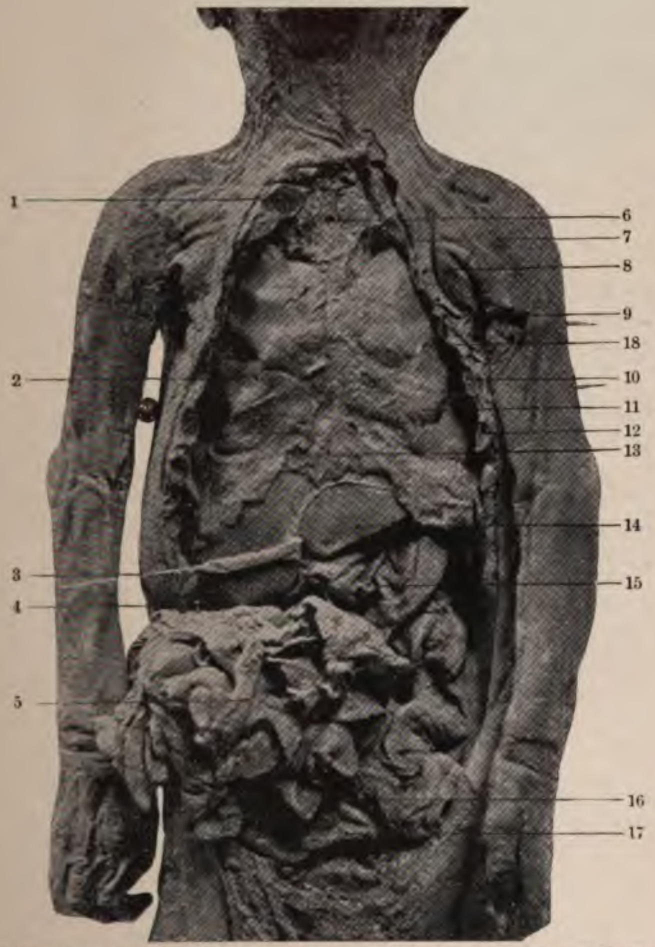
The œsophagus presents an antero-posterior curve corresponding to the spinal column, and deviates at its commencement to the left side, returning at the root of the neck to the middle line behind the trachea, and finally just before it pierces the diaphragm it turns again to the left. There are three slight constrictions in the tube, corresponding to the above points of deviation, but the lowest one at the cardiac opening in the stomach is most contracted and offers the greatest obstacle to the further passage of a foreign body (see Fig. 13). Its diameter in the adult is about three-quarters of an inch, and diminishes proportionately with the youth of the patient. In many cases within the writer's observation the diameter of the tube has not exceeded half an inch in children about ten years of age, although capable of distention to nearly twice that extent.

THE THORAX.

There is much variability in the form of the thorax in children as well as in adults, even when in health; and very commonly a want of symmetry is noticed in the two sides of the chest, the circumference of the right being greater than that of the left. In early infancy the top of the sternum is about on a level with the first dorsal vertebra, while in the adult it is opposite the second. This change is due to the causes which produce the normal curvatures of the spinal column. The ribs are very yielding throughout childhood, and any undue continued pressure or direct violence may cause lateral flattening or depression. In rickets, changes occur leading to the formation of bony beads at the juncture of the ribs and their cartilages. Pigeon-breast is a peculiar protuberance of the sternum caused, it is thought, by interference with inspiration. In strumous children, affected with chronically enlarged tonsils, there is sometimes such an impediment to the entrance of air in inspiration that the thoracic walls yield to the unbalanced atmospheric pressure brought to bear upon them externally (Shaw).

The most elastic part of the thorax is where the ribs join their cartilages. In early childhood the ribs are flatter and less hooped, and up to the end of the third year breathing is more abdominal than thoracic, while after that age in boys, and in men too, it is effected by the action of the muscles attached to the lower seven ribs as well as the diaphragm. In adult

Fig. 18.



Photograph from a Dissection in which the Viscera were held in Position by Transfixion with Pins: from a child about ten months old.—1, clavicle; 2, right lung, covered with pleura; 3, round ligament of liver; 4, right lobe of liver; 5, small intestine pulled aside to show arrangement of sigmoid flexure; 6, thymus gland; 7, first rib; 8, second rib; 9, third rib; 10, fourth rib; 11, apex of heart, covered with pericardium; 12, fifth rib; 13, diaphragm; 14, left lobe of liver; 15, stomach; 16, sigmoid flexure; 17, bladder; 18, left lung, covered with pleura, approaching the right in the middle line.

females the upper ribs are brought more into play, which is a natural adaptation of the chest to the condition of the abdomen during pregnancy.

The intercostal muscles have very little power over the ribs until towards puberty, and chiefly aid the diaphragm in the motions of tranquil breathing; but in forced inspiration the shoulders are steadied by the great staying muscles attached to them and to the collar-bones and upper part of the chest, so as to allow greater expansion of the thoracic cavity. Expiration is performed mainly by the simple elastic properties of the chest-walls relaxing after the effort of inspiration. Respiration is in a measure under the control of the will, but the phenomena attendant upon it are mainly due to the influence of the pneumogastric and phrenic nerves. Drs. Carpenter and Marshall Hall have shown that there are other sources of respiratory excitation. The fact that a new-born infant first begins to draw breath vigorously when the air comes in contact with its face leads to the inference that the surface-nerves play an important part in inciting the first inspiratory effort and no doubt assist the effort of respiration at all times.

THE THYMUS GLAND.

This glandular body is situated just behind the top of the sternum, extending when fully developed at the end of the second year into the root of the neck over the trachea, and separated from the great vessels by the thoracic fascia. It rests below upon the pericardium just above the point where the pleuræ approach each other (see Fig. 18). After the second year it diminishes until it entirely disappears or is substituted by a mass of fat. It is of a pinkish-gray color and lobulated, and at birth weighs half an ounce. It is very vascular, and is now supposed to be concerned in the production of the colored blood-corpuscles.

THE PLEURÆ.

The pleuræ in a young child are quite thick in their costal relations. The pleuræ generally reach as low as the articulation of the twelfth ribs with their vertebræ, and sometimes to the transverse process of the first lumbar vertebra. This should be remembered in operating upon the kidney.

THE LUNGS.

The lungs are of a pinkish-gray color at birth, and become more gray, and finally mottled gray and black, owing to the chemical changes in their tissue derived from the air and blood in the processes of respiration. The vesicles increase in size from birth to old age. "In the fœtus at the full period or a still-born child, the lungs, comparatively small, lie packed at the back of the thorax, and do not entirely cover the side of the pericardium; after respiration has been established they expand and completely cover the pleural portions of the sac, and are also in contact with almost the whole extent of the thoracic wall, where it is covered with the pleural membrane. At the same time their previously thin sharp margins become more obtuse, and their whole form is less compressed." (Quain.)

In children the respiratory efforts are very rapid, being forty-four in a minute at birth, twenty-six in a minute at five years of age, twenty in a minute from fifteen to twenty years, and after that age about sixteen in a minute.

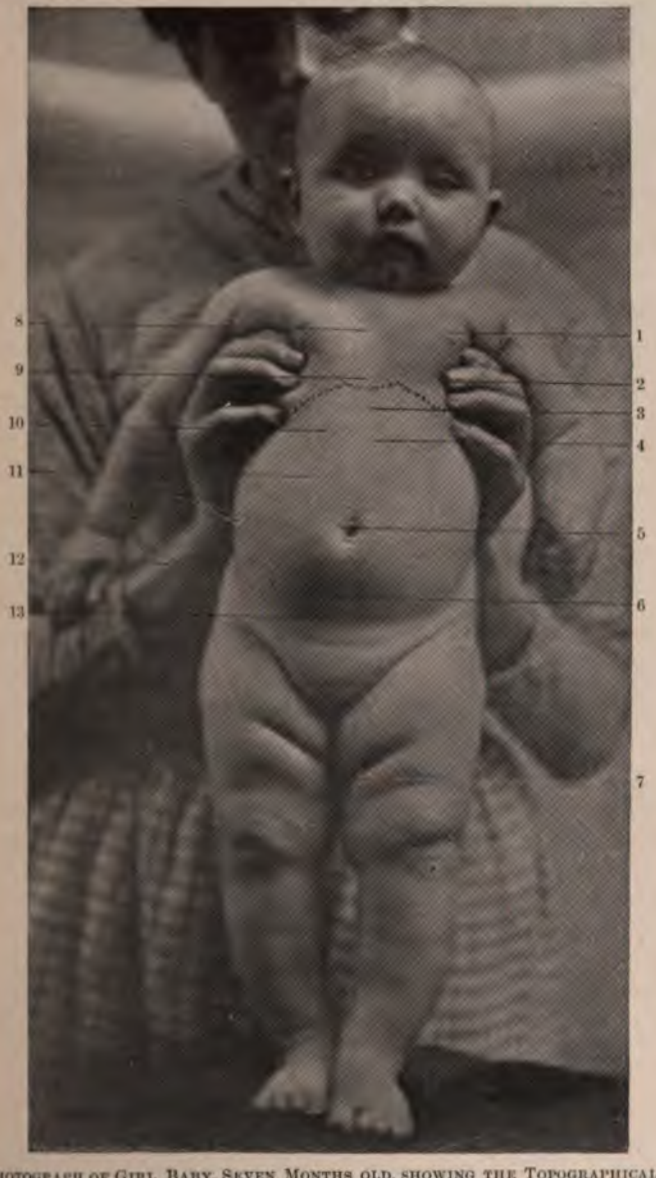
The apices of the lungs in children very closely correspond in their relations to those of the adult, mounting as high in the neck above the clavicle as two finger-breadths behind the subclavian arteries. The vesicular sounds can best be heard below the clavicle, and the bronchial sounds at the upper part of the sternum (see Figs. 19, 20, and 21). In the latter situation there is no lung-tissue overlying the bronchial tube, the lungs converging from the sternal ends of the clavicles towards the middle line, where their borders nearly meet opposite the junction of the second ribs and the sternum. Below this latter point to the level of the fourth costal cartilage the inner margins of each lung run parallel, and, although not so close in the child as in the adult, overlap the great vessels at the root of the heart (see Fig. 18).

THE HEART.

In the early stages of fœtal formation the heart occupies nearly the whole of the thoracic cavity, and, comparatively speaking, is much larger than at later periods or subsequent to birth. The auricular portion exceeds the ventricular, and the right auricle is more capacious than the left, the right ventricle being smaller than its fellow. The organ is also placed vertically within the thorax in its early stages. Just before birth, however, these peculiarities disappear, and the ventricular portion becomes the larger part, the left having the thickest walls, and the whole organ rapidly approaches its normal condition for life. The internal structure of the fœtal heart is chiefly different from that of the adult in having an oval opening (foramen ovale) between the two auricles, which allows a communication from side to side, and in the large size of the Eustachian valve which directs the blood from the inferior vena cava through the foramen ovale. The latter generally becomes closed within the first week or ten days after birth, but may remain open longer, and in some instances has been found to be slightly pervious at a great age. The Eustachian valve speedily dwindles after the establishment of the functions of the lungs and the proper circulation of the blood.

Contemporary with these structural alterations are changes in the great vessels upon which the independent circulation of the blood also depends. The pulmonary artery of the fœtus, after leaving the right ventricle, gives off the right pulmonary branch, and then divides into two other branches, the first of which is quite as large as the pulmonary artery itself, about half an inch long, and directly joins the aorta at the termination of its arch, while the other goes to the left lung. The connecting branch between the pulmonary artery and the aorta is named the *ductus arteriosus* (see Fig. 22).

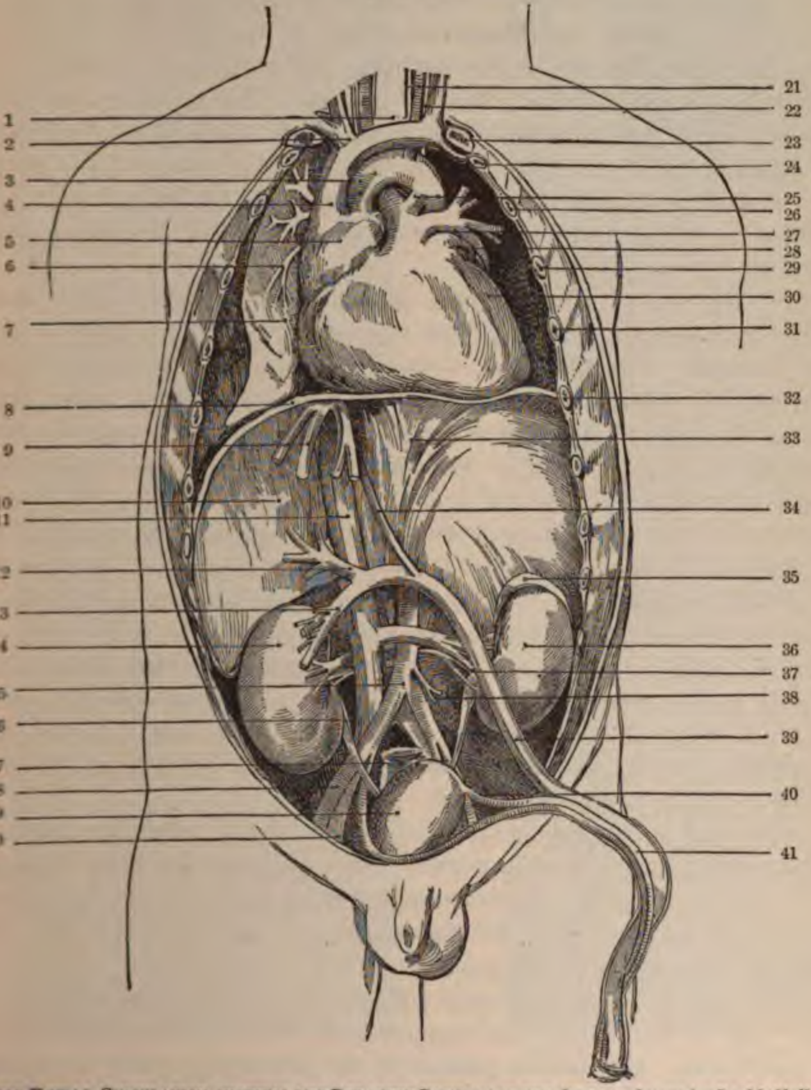
FIG. 19.



PHOTOGRAPH OF GIRL BABY, SEVEN MONTHS OLD, SHOWING THE TOPOGRAPHICAL RELATIONS OF SOME OF THE ORGANS AND LANDMARKS IN FRONT.—1, left nipple pushed upwards by the mother's hand; 2, position of apex-beat of the heart; 3, ensiform cartilage; 4, position of stomach in contact with abdominal wall; 5, umbilicus; 6, position of the bladder; 7, fatty fold noticeable before child can walk; 8, position of the base of the heart; 9, diaphragm; 10, lower margin of costal cartilages; 11, lower border of the liver; 12, position of the vermiform appendix; 13, position of the sigmoid flexure of the colon in the infant.

It is really the continuation of the pulmonary artery, and is in such close relation with the left branch of that vessel that in later life the fibrous

FIG. 22.



THE FŒTAL CIRCULATION, SHOWING THE RELATIVE POSITION OF THE ORGANS, drawn by Dr. McClellan from dissections and preparations in his cabinet.—1, the trachea, with the carotid arteries on either side; 2, the right innominate vein, overlying the innominate artery; 3, the arch of the aorta; 4, the superior vena cava; 5, the right auricle; 6, the right pulmonary vessels passing into the unexpanded lung; 7, the right lung packed in the back of the thorax; 8, the diaphragm; 9, hepatic veins; 10, the right lobe of the liver, dissected to show branches of portal and hepatic veins; 11, inferior vena cava; 12, right branch of portal vein; 13, portal vein coming from the intestines, which are removed; 14, right kidney; 15, bifurcation of aorta into the two common iliac arteries; 16, right ureter; 17, rectum tied; 18, right external iliac artery and vein; 19, bladder; 20, right hypogastric artery; 21, left recurrent laryngeal nerve; 22, left pneumogastric nerve; 23, left clavicle; 24, first rib; 25, ductus arteriosus; 26, second rib; 27, left pulmonary vessels; 28, left auricle; 29, third rib; 30, left ventricle; 31, fourth rib; 32, fifth rib; 33, crura of diaphragm; 34, ductus venosus (the left lobe of the liver, stomach, pancreas, and spleen are removed); 35, left supra-renal capsule; 36, left kidney; 37, left renal vessels; 38, inferior mesenteric artery; 39, umbilical vein; 40, left hypogastric artery; 41, umbilical cord.

cord which indicates its remains is attached to the root at the arch of the aorta.

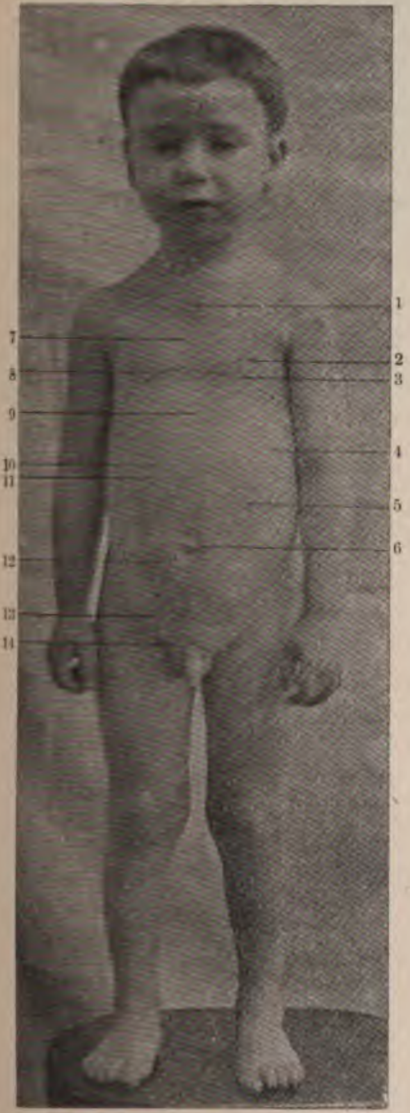
The blood which has circulated in the fœtal system is curiously returned to the placenta by means of the hypogastric arteries, which are the continuation of the superior vesical branches of the internal iliac arteries. They pass out of the abdomen at the umbilicus and coil round the vein which brings the blood to the fœtus from the placenta. The umbilical vein up to the moment of birth distributes the blood chiefly to the liver of the fœtus by branches to the portal vein and to the lobes of the organ, but a portion is conveyed by a small communicating branch to the inferior vena cava without passing at all through the substance of the liver, and this is called the *ductus venosus* (see Fig. 22).

At birth the only blood which goes to the liver is by means of the portal vein, and after being purified it is conveyed by the hepatic veins to the vena cava. The umbilical vein and the ductus venosus become empty and contract, and are ultimately converted into the fibrous cords which occupy the fissure of the ductus venosus of the liver and become its round ligament. They are usually obliterated about the fifth day after birth. Holden wisely says, "It is well to bear in mind that these important vascular changes do not take place suddenly at birth, but that they are the result of gradual development which is completed at or soon after birth, mainly by the act of inspiration, whereby the blood passes through the lungs, the placental circulation at the same time being interrupted." A just reflection upon this subject should help to interpret the anomalies which are met with in these parts and the possible results following interference with the natural order of things, and will show the importance of active and varied exercise in children to assist the healthful development of the thoracic organs, upon which so much in after-life depends.

The heart of the child beats more rapidly than in adult age, and is accelerated by general muscular activity. The pulse-rate has been estimated to be 130 to 140 in the new-born infant, 100 to 115 in the second year, 80 to 90 from the seventh to the fourteenth year, and 75 to 80 after that, although there are many exceptions.

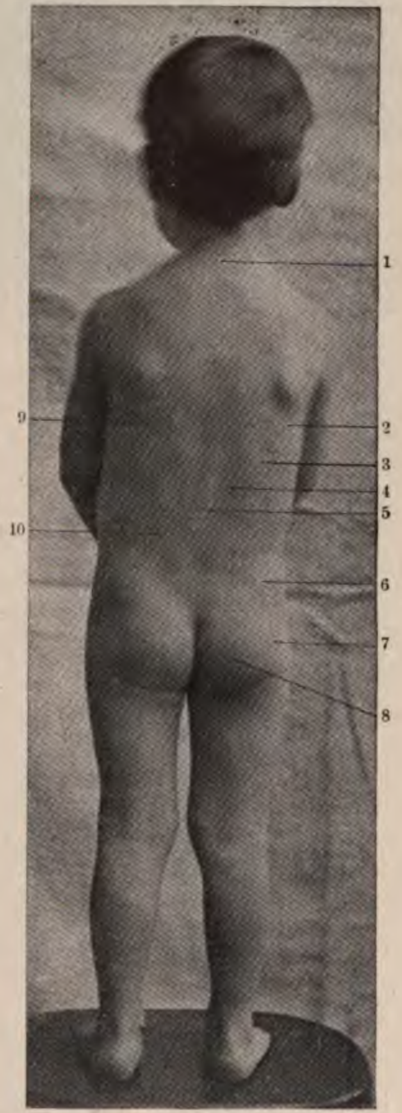
Normally, the heart is situated obliquely between the lungs behind the lower half of the sternum, and occupies more of the left than the right side of the thorax. It is held in position by the pericardium, which is attached to the central tendon of the diaphragm, and by the thoracic fascia, which is continuous with the deep cervical fascia, and embraces the great vessels at its base. In the adult the apex of the heart may be felt beating at each contraction between the cartilages of the fifth and sixth ribs to the left of the sternum, and its base corresponds to the junction of the third costal cartilage on the right side with the sternum. In early infancy the heart is of greater breadth in comparison with the chest, and therefore the apex-beat in relation to the nipple is changed. The normal apex-beat in the adult is about an inch internal to the mammillary line, while in children it

FIG. 20.



PHOTOGRAPH OF BOY, AGED FOUR YEARS, SHOWING THE TOPOGRAPHICAL POSITION OF SOME OF THE ORGANS AND LANDMARKS IN FRONT.—1, supra-sternal notch; 2, left nipple; 3, position of apex-beat of heart; 4, spleen; 5, left kidney; 6, umbilicus; 7, base of heart; 8, diaphragm; 9, ensiform cartilage; 10, margin of ninth rib; 11, lower border of liver; 12, position of vermiform appendix; 13, internal abdominal opening; 14, external abdominal opening.

FIG. 28.



PHOTOGRAPH OF BOY, AGED FOUR YEARS, SHOWING THE TOPOGRAPHICAL RELATIONS OF SOME OF THE ORGANS AND LANDMARKS BEHIND.—1, spine of the seventh cervical vertebra; 2, inferior angle of the scapula; 3, posterior point of liver dulness; 4, right kidney; 5, twelfth spine of the dorsal vertebra; 6, crest of ilium; 7, position of great trochanter of the femur; 8, tuberosity of the ischium; 9, inferior angle of the left scapula; 10, position of left kidney.

is often directly at this line, or even external to it. Careful observation upon young children has detected the apex-beat at the fourth interspace; and this is probably due to the mounting up of the diaphragm as well as to the more oblique shape and position of the ribs (see Figs. 19 and 20). Dissections made upon fresh bodies of infants, with the thoracic viscera held in place by being transfixed with long pins, have shown the author that the position of the heart is higher than in later life, so that the apex would probably be felt above the fifth rib (see Fig. 23).

It is difficult at any age to fix definitely the points where the sounds of the cardiac valves can be detected by the ear applied to the chest, owing to the interposition of lung-tissue between the chest-walls and the heart. This difficulty is greatly increased in examining young children, in whom the higher position of the heart still further confuses the sounds of its valves with those of the respiratory organs. The position of the heart always varies with the position of the body, and in children there is perhaps a greater laxity of the supporting membranous attachments of the heart, which permits still further latitude.

THE DIAPHRAGM.

The diaphragm occupies comparatively a higher position in children than in adults, and is naturally well developed, because it plays such an important *rôle* in their respiration, as has already been pointed out. The lungs in their pleuræ rest upon the muscular portions of the diaphragm upon each side in childhood as in later years, while the heart in the pericardium lies above the central tendon (see Figs. 22 and 23). After death the diaphragm mounts higher within the thorax, owing to the collapsed state of the lungs from expiration. During life, when the diaphragm contracts there is a general descent of the muscular partition, more especially at the sides. The ever-changing position of the diaphragm in life renders the study of its relations to the adjacent viscera of the thoracic and abdominal cavities of peculiar interest.

THE ABDOMEN.

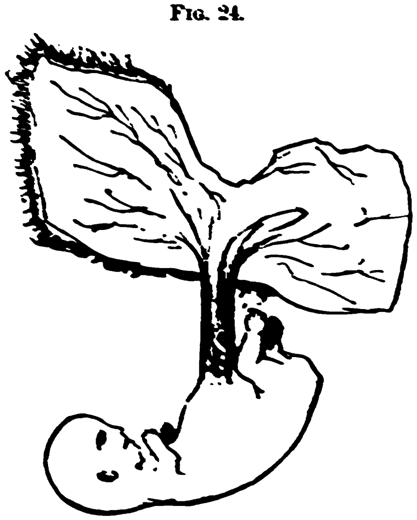
The protuberance of the abdomen which is so marked in young children is mainly due to the relatively large size of the liver and the small size of the pelvis. In infancy the bladder and upper portion of the rectum are in the abdominal region.

The skin over the front of the abdomen is elastic and loosely attached in the groins, but partially adherent to the subjacent fasciæ in the middle line. The superficial fascia generally contains much fat, and between its two layers over the lower part of the abdomen are the superficial vessels and nerves. The thickness of the abdominal wall depends upon the amount of subcutaneous fat, and not on the muscles, the three layers of which are quite thin at all times.

The linea alba is the fusion of the aponeuroses of the abdominal muscles, and is marked by a furrow above the umbilicus, where the recti muscles slightly diverge. It is the safe line in this region, owing to its freedom from blood-vessels.

The umbilicus is the cicatrix in the centre of the linea alba, resulting from the obliteration of the umbilical vessels and cord at birth. It consists of a dense fibrous ring resulting from adhesion of all the adjacent structures, skin, fascia, and peritoneum. The umbilicus is an important landmark, and is deeper and wider in the female than in the male; it is nearer to the pubes than to the ensiform cartilage. From birth until the end of the second year it occupies the central point of the body, but as the legs grow longer the latter point is at or about the pubes. It is sometimes the seat of congenital hernia, which works its way through the structures of the cord, separating the umbilical vein and arteries, and forming a cavity in the gelatinous and cellular tissue. Cases have been reported where the bowel was involved in the ligature of the cord at birth. The infantile form of hernia in this region is usually caused by straining, and occurs at the umbilicus a few months after birth, before its constituents have become firmly cicatrized. These herniæ may be referred to the persistence of one or other of the fœtal conditions, for about the eighth or ninth week of embryonic life there is found a coil of intestine within the umbilical cord (see Fig. 24). Owing to the cords which represent the obliterated hypogastric arteries and urachus being attached to the lower part of the umbilicus, it is thicker than the upper part, and on this account umbilical hernia in the adult occurs at the upper part of the umbilicus. The part of intestine involved in this variety of hernia is usually from the jejunum, and the hernia is very generally fatal, since a lesion of the bowel is more serious the nearer it approaches the stomach.

FIG. 24.

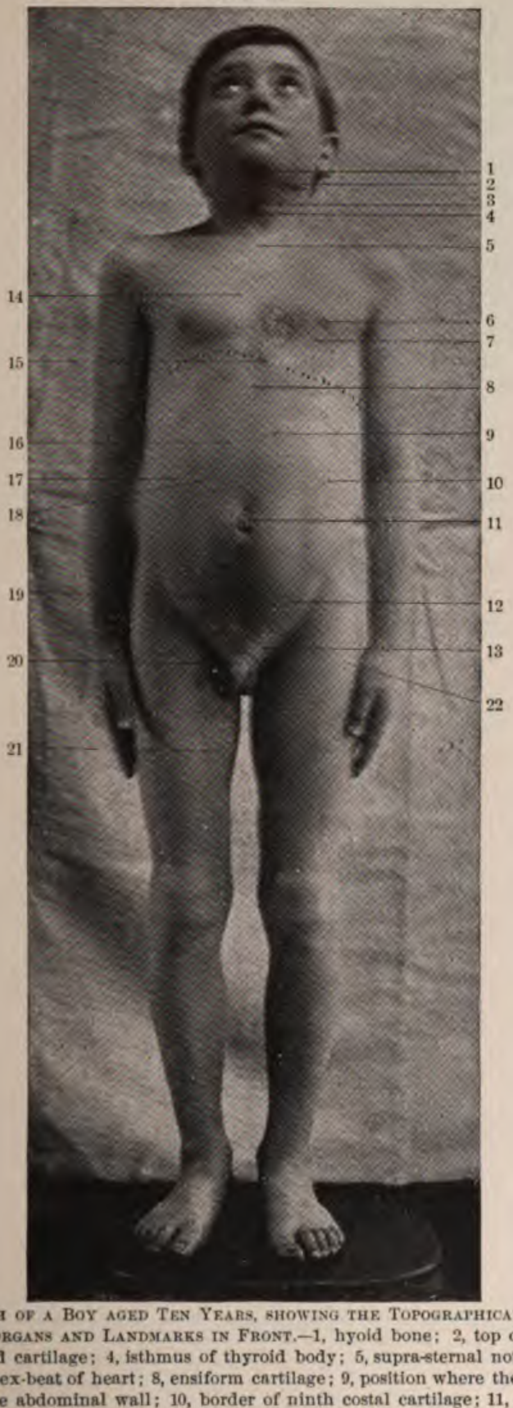


HUMAN EMBRYO OF THE NINTH WEEK, SHOWING A COIL OF INTESTINE IN THE UMBILICAL CORD.

There are rare cases met with of urinary fistulæ existing at the navel, which are due to the urachus not being entirely closed at birth, and allowing the urine to dribble out from the bladder; and fecal fistulæ have also been known, which are caused by persistence of Meckel's diverticulum, which "springs from the ileum from one to three feet above the ileo-cæcal valve" (Treves).

There is a close similarity between the vessels, arteries, and veins of the abdominal walls in the child and adult, and the same may be said of the nerves, but, as so many symptoms relating to the diseases of childhood can

FIG. 21.



PHOTOGRAPH OF A BOY AGED TEN YEARS, SHOWING THE TOPOGRAPHICAL POSITION OF SOME OF THE ORGANS AND LANDMARKS IN FRONT.—1, hyoid bone; 2, top of thyroid cartilage; 3, cricoid cartilage; 4, isthmus of thyroid body; 5, supra-sternal notch; 6, nipple; 7, position of apex-beat of heart; 8, ensiform cartilage; 9, position where the stomach is in contact with the abdominal wall; 10, border of ninth costal cartilage; 11, umbilicus; 12, internal inguinal opening; 13, external inguinal opening; 14, position of base of the heart; 15, diaphragm; 16, duodenum; 17, lower border of the liver; 18, gall-bladder; 19, position of vermiform appendix; 20, femoral ring; 21, apex of Scarpa's triangle; 22, position of great trochanter.

be interpreted only through a knowledge of the position of the nerves, they are here more particularly described. They are derived from the lowest seven intercostal and upper two lumbar nerves, and are placed parallel to each other, running obliquely downward and inward to the middle line, supplying not only the skin, but also the overlying muscles. This intimate association serves to protect the viscera from many injuries, such as are due to contusions and burns. Owing to their origin, the intercostal muscles are associated in the movements of respiration with the muscles of the abdomen. In Pott's disease of the spine, the nerves may be pressed upon at the vertebral foramina, giving rise to a sense of constriction about the abdomen, and children suffering with this affection often complain of pain in the region of the navel. The position of the caries may be determined by a careful study of the symptoms, as the cutaneous pain indicates what particular nerve or nerves are involved. The sixth and seventh intercostal nerves supply the skin over the epigastrium, the tenth nerve that about the umbilicus, and the upper lumbar nerves are distributed along Poupart's ligament. Locomotor ataxia and spinal sclerosis are often attended by the sense of constriction due to some nerve-disturbances in the same way. Moreover, the connection between the sympathetic system and the spinal nerves, in the dorsal region especially, brings into relation the parietes of the abdomen and the viscera which they overlie. This is seen in peritonitis, where the skin over the abdomen is extremely sensitive and the respirations are entirely thoracic.

THE GROIN.

The anatomy of the groin in the child is in many respects similar to that of the same region in the adult, modified only by growth. Poupart's ligament is the reflection of that part of the sheath of the external abdominal muscle which is inserted into the spine of the ilium, and, curving downward, with the concavity towards the abdomen, separates into two portions, the lower one going to the spine of the pubes and the upper one to the pubic symphysis and interlacing with the fibres of its fellow-muscle from the opposite side. The space between these portions is called the outer abdominal opening (or ring). It can be recognized by feeling for the insertion of the tendon of the adductor longus muscle while the thigh is abducted; and in the male the cord, or in the female the round ligament, will be detected issuing from the external opening. These latter structures occupy the so-called inguinal canal, which is rather a tract of tissue congenitally arranged for their passage, and capable of being distended or ruptured in various forms of hernia. It extends obliquely upward about the patient's finger's-breadth above, and to a point corresponding to the middle of, Poupart's ligament. This is over the opening in the extra-peritoneal fascia known as the internal abdominal opening (or ring) (see Figs. 20 and 21).

In the fœtus the testicle is formed below the kidneys in the lumbar region, and about the eighth month presents at the internal opening, gradually finding its way into the scrotum. The descent of the testicle is naturally accompanied with the formation of the cord by the aggregation of its developing constituents,—*i.e.*, vas deferens, veins, arteries, lymphatics, nerves, and gelatinous tissue,—and the progress of the testicle through the tract devised for it is liable to be attended with some congenital defect, which sooner or later may allow a portion of intestine to escape from the abdomen.

While it may be asserted that every hernia is due to some congenital or abnormal condition of the parts concerned, and that the effort which occasions the affection would not so operate except for the existence of some such condition, the ordinary forms of hernia which are described as congenital are assigned to defects in the vaginal process of the peritoneum. The inguinal canal is relatively shorter and less oblique in the fœtus and young child than it is in the adult, and in the female it is always smaller and narrower. In fact, about the time the testicle reaches the groin the internal abdominal opening is just behind the external opening, and its course from where it was originally formed below the kidney to its final normal position in the scrotum is more direct than indirect. The process of the peritoneum which passes through the inguinal tract, known as the vaginal process in the male, and in the female as the canal of Nuck, always precedes the descent of the testicle, and would seem to lead the way for it, although it is not pushed before it, as usually described, for in certain well-recognized cases, where the testicle was retained within the abdomen and subsequently descended into the scrotum, years after birth, the vaginal process had already occupied its normal position in the scrotum.

After the passage of the testicle along the inguinal tract from the internal to the external opening, and thence into the scrotum, it drops upon the vaginal process of the peritoneum. Ordinarily this process, after the descent of the testicle, becomes adherent to the adjacent structures at the internal opening in the extra-peritoneal fascia, and is separated distinctly from the rest of the peritoneum, becoming gradually blended with the cord above the testicle. When the vaginal process has not become separated from the general peritoneum, and there is no adhesion between it and the structures of the inguinal tract, there is naturally a direct passage-way between the abdomen and the scrotum, and if, at any age, a loop of intestine descends by it, the condition is called a congenital hernia. In this form of hernia the testicle is enveloped by the intestine, and there is no other sac than the vaginal process.

When the original vaginal process is only occluded at the internal opening and there is a continuation of its pouch above the testicle along the cord, there is apparently only a thin septum between its cavity and that of the peritoneum. A portion of intestine will sometimes push down this septum (encysted hernia), or it may be forced down behind the adhesion of the vaginal process at the internal opening, forming infantile hernia. In the latter it will be readily seen that there will be three layers of peritoneum covering the intestine,—viz., the two layers of the vaginal process, and the proper

DIAGRAM OF FIG. 23.

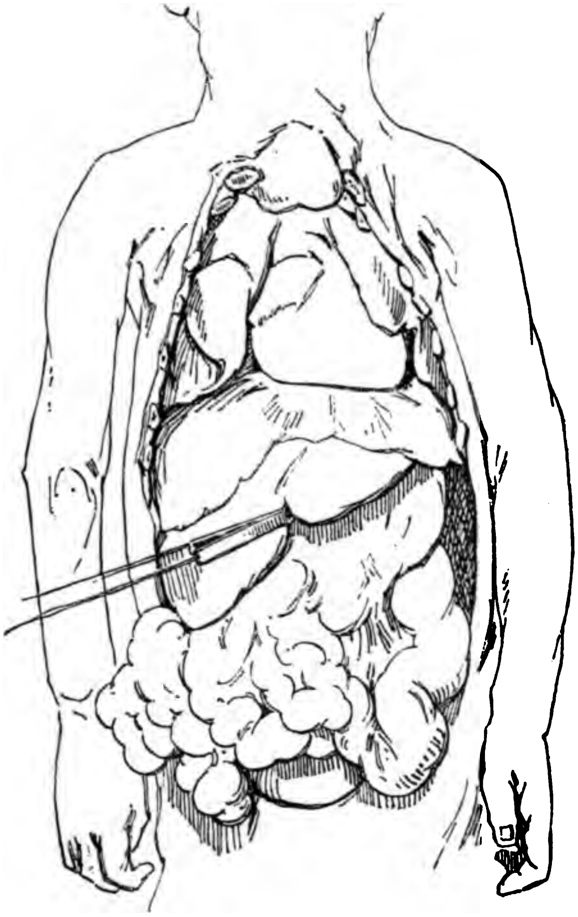
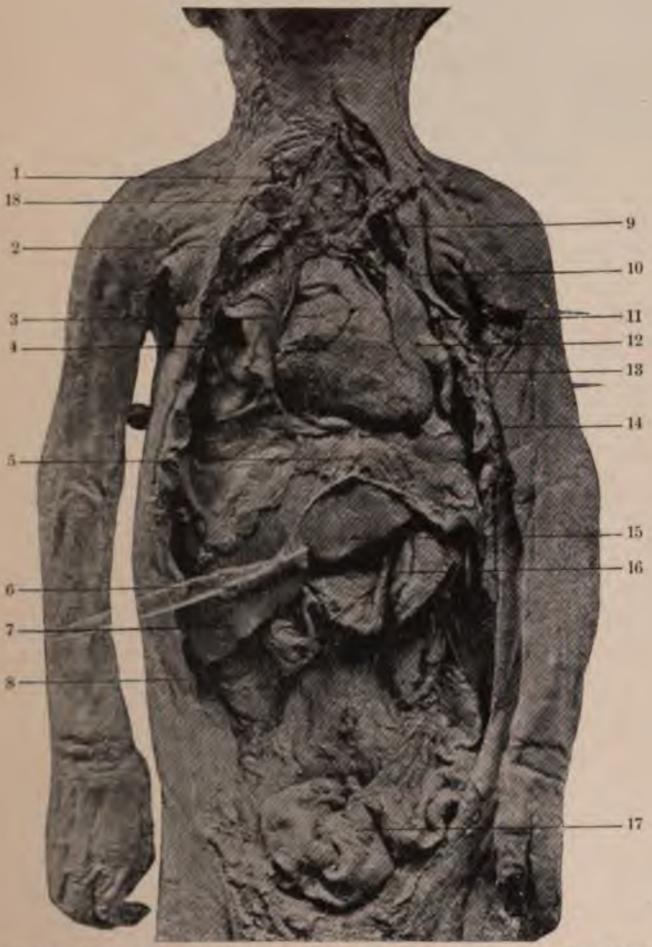


FIG. 23.



PHOTOGRAPH FROM A RECENT DISSECTION, IN WHICH THE VISCERA WERE HELD IN POSITION BY TRANSFIXION WITH PINS: from a new-born child.—1, trachea; 2, innominate vessels; 3, right auricle; 4, right lung (turned back to show heart), the pericardium is removed; 5, central tendon of diaphragm; 6, round ligament of liver; 7, right lobe of liver; 8, right kidney; 9, first rib; 10, second rib; 11, third rib; 12, left lung (turned back); 13, fourth rib; 14, fifth rib; 15, left lobe of liver; 16, stomach; 17, sigmoid flexure of colon; 18, clavicle. The small intestines are removed.

sac of the hernia itself. It has been suggested that the anatomical arrangement of the parts in the encysted variety favors the rupture of the septum above mentioned, and may explain the occurrence of a congenital hernia in adult life. The process of the peritoneum which accompanies the cord is sometimes not completely obliterated, and only closed at the top of the testicle. It is known as the funicular process, and gives its name to that form of rupture which it accommodates. The complete closure of the tunica vaginalis is peculiar to man, and has been considered as connected with his adaptation to the erect posture. The most common form of hernia in female children, occurring in the inguinal region, is occasioned by the patent condition of the canal of Nuck, which allows the intestine to escape along the course of the round ligament.

All truly congenital herniæ in the groin are indirect. There is apparently less resistance in the abdominal wall opposite Hesselbach's triangle, especially at the outer margin of the conjoined tendon of the internal oblique and transverse muscles, but the direct form of hernia is not only very rare at any age, but, as has been said, does not occur congenitally. It is probable, however, that some undue weakness may exist which predisposes to rupture between the deep epigastric vessels and the cord resulting from the obliteration of the hypogastric artery, or between the latter and the border of the rectus and pyramidalis muscles. Such weakness may be caused by slight change of position of the cord of the hypogastric artery in its relation to the deep epigastric, as it clearly aids in the production of the pouch in the peritoneum easily demonstrated at this point. The course of the deep epigastric artery is always from the inner margin of the internal abdominal opening running between the extra-peritoneal fascia and the peritoneum towards the umbilicus. When it reaches the border of the rectus muscle it pierces it and passes upward to join with the internal mammary artery. In herniotomy, the constriction usually occurring at the inner opening, the incision should be made parallel to the course of the artery. The coverings to a hernia in the inguinal region are practically those of the testicles, and, in operating, the only one which is recognizable is the cremasteric fascia. Much undue stress has been laid upon the number and origins of these coverings. It would be more useful to master the exact positions of the openings, and to understand the construction of the inguinal tract.

THE PERITONEUM.

The folds of the peritoneum can best be understood by reference to the development of the alimentary canal. When the latter first assumes the tubular form it is a simple straight cylinder, placed in front of the vertebral column, attached to it and to the rest of the embryo by a membranous fold or rudimental mesentery. By degrees the intestine, growing in length, becomes looped at the centre and straight at its upper and lower ends, whilst the portion which is destined to become the stomach is dilated. This portion gradually turns over on its right side, so that the border which is connected to the spine by the membranous fold comes to be turned to the left. The stomach becoming further dilated is at first placed vertically, then obliquely, and then transversely, carrying with it in all its changes the membranous fold, from which the omenta are afterwards produced. In the early embryo the calibre of the upper part of the intestine is greater than that of the lower, and there is no distinction between the two until the formation of the cæcum, which is at first about the centre of the canal, and is a simple tubular diverticulum which later dwindles at its free part, becoming the vermiform appendix. After the appearance of the cæcum the primitive intestinal canal undergoes great changes: the lower portion increases in calibre, and the upper becomes more looped, sending some of its coils with the cæcum into the umbilical cord (see Fig. 24).

About the beginning of the third month the ileo-cæcal valve is discernible, and the colon, first lying to the left of the small intestines, gradually crosses over their upper part, and, steadily growing in length, assumes about the fifth month the normal position found in the adult. The curvatures of the stomach follow as a sequence upon its dilatation and change of position, and the mesial fold of the peritoneum surrounding it becomes the sac of the omentum, the portion covering the right side of the stomach being turned inward and the portion over the left side passing over the front wall of the stomach, so that its free edge becomes the anterior boundary of the foramen of Winslow, the opening between the lesser and greater involutions, or cavities of the peritoneum, as they are called. These involutions are partly due to the unequal constriction of the pyloric and œsophageal ends of the stomach and the beginning of the duodenum. The mesentery about the larger intestine is variously arranged and prolonged during the growth of the abdominal organs of the fœtus, and the many congenital defects and abnormal conditions offer the best means of explaining what are usually described as its normal relations. It has been suggested that an abnormally long mesentery may predispose to herniæ; and it is certain that the peritoneum will allow of very considerable stretching if it is gradually exerted.

THE STOMACH.

The stomach, as has been described, is originally placed vertically in the abdomen. This position may continue in adult life, and it probably does so more often than has been observed, but the author is firmly convinced, from numerous autopsies and dissections, that its normal position after birth is as seen in the illustration (see Fig. 25).

It is thought that the peculiar shape of the fundus of the organ is due to the muscular action of the layers of the stomach on the food, and in newly-born children the fundus does not exist. Its dimensions and relations are subject to constant alterations, principally due to the changes in the position of the diaphragm, to which it is closely attached, but also consequent upon its empty or distended state. It has been shown by Braune that the cardiac orifice of the stomach is of valvular construction, and so

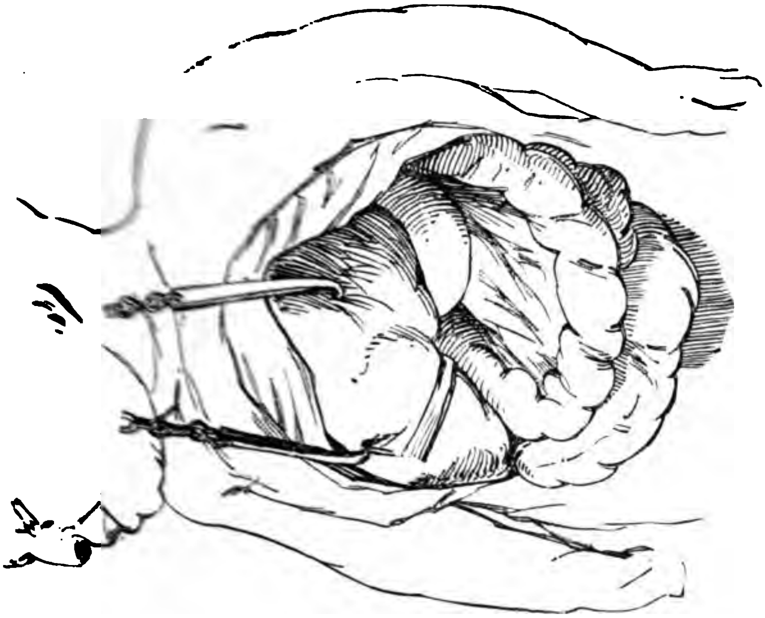
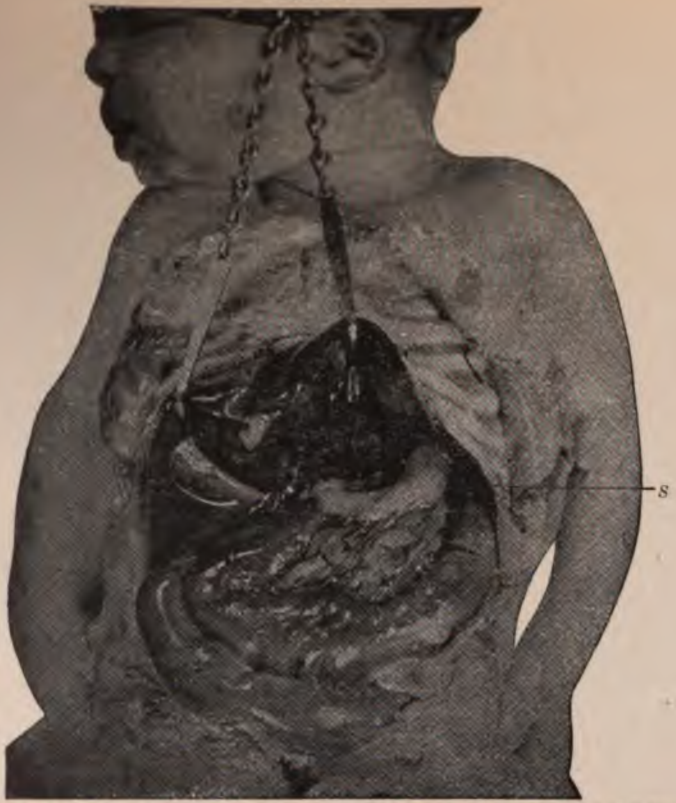
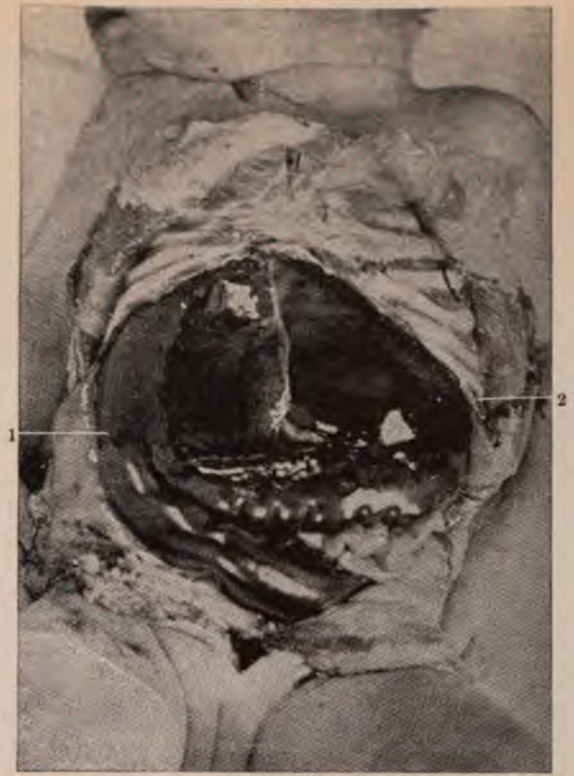


FIG. 25.



PHOTOGRAPH FROM A DISSECTION. LIVER RAISED TO SHOW THE POSITION AND SHAPE OF THE STOMACH, S, IN THE NEW-BORN INFANT. (By Dr. McClellan.)

FIG. 26.



PHOTOGRAPH FROM A DISSECTION, SHOWING THE USUAL SIZE OF THE LIVER IN THE NEW-BORN INFANT.—1, right lobe; 2, left lobe, entirely concealing the stomach. (By Dr. McClellan.)

arranged that regurgitation of fluids into the œsophagus is somewhat difficult. More recently Gubaroff has stated that this valvular arrangement is deficient in infants, and thus the facility with which they vomit may be accounted for.

THE SMALL INTESTINE.

The small intestine varies greatly in length. According to Treves, it measures at birth nine feet five inches, and it grows about four feet in the first two months. The divisions into jejunum and ileum are arbitrary, but the upper part of the small bowel usually occupies the left iliac fossa, and the lower the right. The ileum is that part of the intestine which is most frequently involved in external hernia. The duodenum contains the glands of Brunner, which are commonly the seat of perforating ulcer in cases of burn. They occupy principally the first part of the duodenum, which is covered by the peritoneum.

THE LARGE INTESTINE.

The large intestine from the cæcum in the right iliac fossa to the sigmoid flexure in the left is accessible to pressure through the abdominal walls, and in cases of collection of flatus giving rise to colicky pains, in young children, much relief can be easily afforded by gentle rubbing over the course of the colon. The usual course of the colon is upward from the right iliac fossa to the liver, where the bowel arches transversely across the abdomen to the spleen, forming, at the bends, the hepatic and splenic flexures, and thence downward to the left iliac fossa, where the sigmoid flexure usually occurs before the bowel terminates in the rectum. Very often the disposition of the colon varies, and it may lie diagonally across the abdomen, passing from the region of the liver to the left groin. In children it is frequently different from the condition usually found in the adult described above. Treves states that the cæcum and colon at birth measure one foot exclusive of the sigmoid flexure, which is about ten inches. During the first four months this portion of the bowel does not increase in length, but the sigmoid flexure diminishes, owing to a readjustment of the mesentery. Measurements of the bowel can only be approximative; for the longitudinal muscular fibres are easily damaged in removing the viscera from the body.

The cæcum has a large and loose mesentery in its early formation, and this condition sometimes persists during life. The cæcum is covered with peritoneum, except at its posterior surface, which is connected by a quantity of areolar tissue to the iliac fossa. Here perityphlitis manifests itself. This part is susceptible of great distention, and it is a frequent seat for concretions. The vermiform appendix usually lies behind the cæcum, directed upward. It can be readily reached by an incision made in the semilunar line upon the right side at a point midway between the umbilicus and the spine of the ilium.

The usual point of stricture of the colon is where the sigmoid flexure terminates in the rectum. It is probably due to the arrangement of the bands of peritoneum (sigmoid mesocolon), which brings the loops of the bowel so closely together that they are readily twisted upon their axes. The so-called sigmoid flexure usually consists of a large loop puckered up into folds, and occupies rather the pelvis than the iliac fossa. It is frequently misplaced in children (see Fig. 25). The sigmoid flexure passes directly over the left spermatic vein, and in consequence of constipation the pressure may produce varicocele on the left side in young adults.

The RECTUM is considered with the pelvis.

THE LIVER.

This important glandular organ begins to be formed at a very early period of fœtal life by a process from the intestinal tube. It grows very rapidly, so that at the third or fourth week in the fœtus the liver constitutes nearly one-half of the entire body-weight, almost filling the abdominal cavity. From that period towards birth it decreases proportionately, however, its relative weight to that of the body being then as one to eighteen. Its position in the abdomen in the fœtus is more symmetrical than that which it occupies later, owing to its right and left lobes being of nearly equal size (see Fig. 26). In infancy and early childhood the large size of the liver has much to do with the differences in the relations of the abdominal viscera as compared with adults. Its size is variable, even within the limits of health, and it is difficult to define its position at different periods of life, owing to its rising and falling with the diaphragm, to which it is attached by folds of the peritoneum, and also to the shape of the thorax (see Fig. 13). In young children in life it is generally found by percussion in the upright posture to be somewhat lower than would be supposed from the usual description in the text-books, its lower border reaching nearly to the crest of the ilium and its left lobe extending across to the costal cartilages of the left lower ribs (see Figs. 20, 21, 27, and 28). The liver is moulded to the arch of the diaphragm, which separates it from the thin margin of the base of the right lung, which descends in front of it. Ordinarily it extends to the left a little over an inch beyond the margin of the sternum. In the middle line the liver is in close relation to the skin in front of the stomach and reaches about half-way between the ensiform cartilage and the umbilicus. Its lower edge corresponds to a line drawn from the ninth right to the eighth left costal cartilage (Quain).

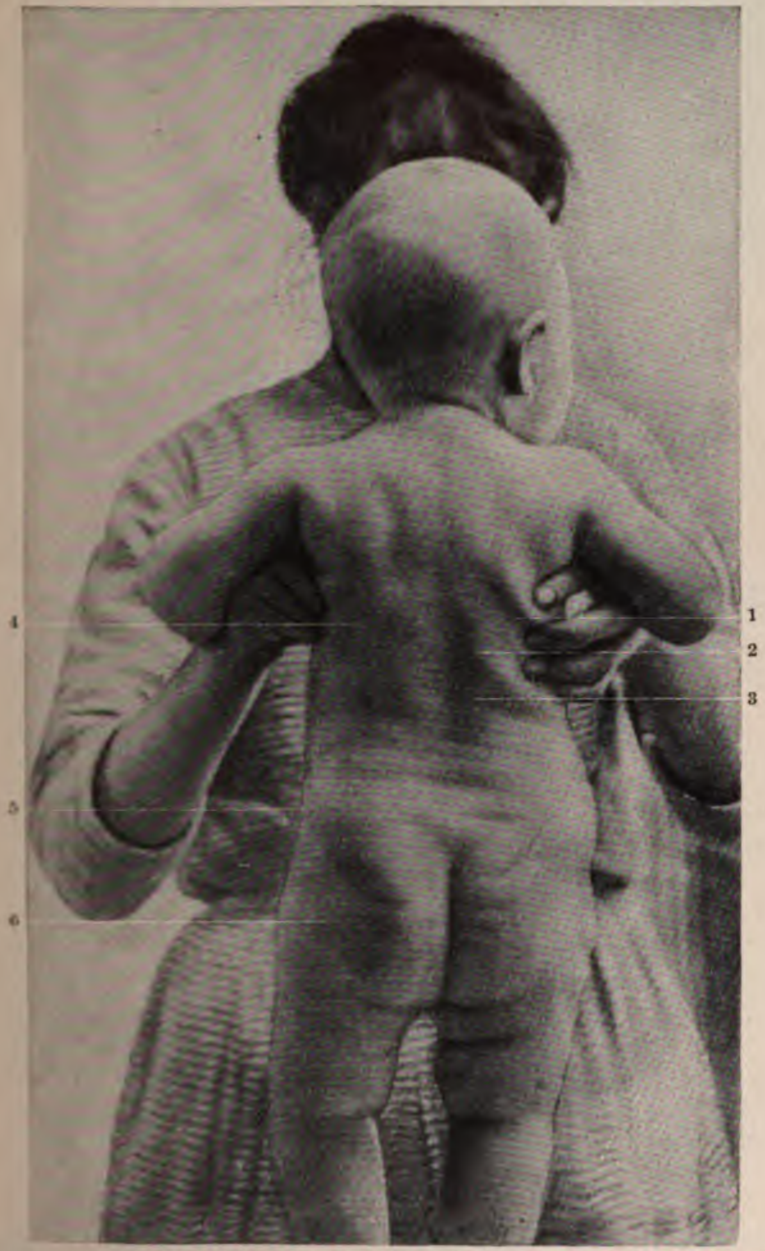
When the body is erect, the lower edge of the liver can be felt about half an inch below the costal cartilages. In the recumbent position it cannot be felt beyond the margins of the ribs in the adult.

The position of the fundus of the *gall-bladder* is in relation to the surface about that of the ninth costal cartilage near the border of the right rectus muscle.

THE SPLEEN.

The first appearance of the spleen in the fœtus is about the eighth week, on the left side of the dilated portion of the alimentary canal or stomach.

FIG. 27.



PHOTOGRAPH OF BABY SEVEN MONTHS OLD, SHOWING THE TOPOGRAPHICAL RELATIONS OF SOME OF THE ORGANS AND LANDMARKS BEHIND.—1, superior angle of right scapula; 2, lower point of liver-dulness; 3, position of right kidney; 4, inferior angle of left scapula; 5, left iliac crest; 6, position of great trochanter.

It increases rapidly in size after birth, and soon attains the same proportional weight to the body as in the adult. The spleen varies more than any other organ in the body according to the state of nutrition, being larger in well-nourished and smaller in wasted children. Its size always increases after digestion, and it is in this state that its substance is most likely to be injured by violence. It is a very vascular organ, but it contains most blood during digestion. The spleen is nearest the surface in the neighborhood of the tenth and eleventh ribs, above which it is overlapped by the edge of the lung. The diaphragm intervenes between it and the parietes everywhere. The splenic flexure of the colon and the stomach are in front of the organ. It is invested and suspended by the peritoneum, so that the normal organ is rarely injured. There is muscular tissue in the capsule of the spleen, to the contractile power of which is attributed the recovery of patients suffering from punctured or pistol-shot wounds. It is said that in children the spleen when enlarged encroaches more upon the thoracic cavity than in the adult, owing to the greater resistance offered by the costo-colic fold of the peritoneum upon which it rests.

THE PANCREAS.

The pancreas is well formed about the second month in the fœtus, about the same time as the salivary glands, which it resembles very much in arrangement and function. Its situation is in front of the first lumbar vertebra, behind the stomach, and corresponds to a point about a hand's-breadth above the umbilicus (see Fig. 13).

THE KIDNEYS.

It is a curious fact that the development of the urinary organs, as well as those of generation, is preceded by the formation and temporary existence of two glandular and vascular bodies, called Wolffian, after their discoverer. In the fœtus they exist only in the early stage of development, and reach their full size about the fifth week. They occupy the abdominal cavity upon either side of the vertebral column, and, after reaching their full size, speedily shrink into its lower part, soon becoming entirely wasted. They take no part in the formation of the kidneys or their overlying capsules, merely preceding and perhaps substituting them in embryonic life. The fœtal kidneys about the seventh week are found as two small oval masses behind the upper part of the Wolffian bodies, which completely cover them. They very soon become lobulated, and continue so until a little while after birth, when their lobulated condition disappears, being thereafter indicated by the pyramids of Malpighi. The kidneys of new-born infants are larger than those of adults, relatively speaking, and are situated lower down than in after-life (see Fig. 22). It should be remembered, however, that the lumbar part of the spine at birth is relatively small, and that on this account the kidneys appear to be lower in relation to the iliac crests than in adult life. A few years after birth the position and relations of the kidneys approximate those of the adult. The deep position of the kidneys and their relation to the spine where the flexion of the column is most acute rende them liable to injury when the back is struck with the spine bent forward Hæmaturia often follows such injuries, and may be thus explained. Th common description of the right kidney being somewhat lower than the lef owing to impingement of the liver has not been found to correspond wit observations made upon frozen sections of children's bodies. Even in case where the liver was greatly enlarged, the colon being pushed down by it the kidney has not been displaced but covered by the overlapping live (see Fig. 23). The kidneys, although having the peritoneum in front an on their external borders, lie behind it, embedded in a large quantity o loose fatty tissue. This fatty tissue is the chief support which holds thes organs in place, and if it is interfered with or absorbed, the kidneys ma be displaced very readily. In operating for any cause upon the kidneys, i the normal position, the patient's body should be extended over a pillo upon the unaffected side, so as to curve the spine laterally, and the incisio should be made along the outer margin of the quadratus lumborum muscle the patient's hand's-breadth from the spine. There are varieties in form position, size, and number of the kidneys, which are not incompatible wit the healthy performance of the function of the organ.

The only peculiarity of the *suprarenal capsules* in children is thei relatively large size. In new-born infants they generally quite cover th kidneys as well as surmount them.

THE PELVIS.

The pelvis of the fœtus and young child is of very small capacity proportionally to the size of the body, and its obliquity is considerably greate than in the adult (see Fig. 30). The alteration in form which the pelvi undergoes is in accordance with its adaptation to the transmission of th weight of the body both in the standing and sitting postures.

There are two arches available for these postures. In standing, th arch is represented by the sacrum and its junction with the two iliac bones, the acetabula, and the intervening masses of bone. In sitting, the arch consists of the sacrum and its iliac articulations, the tubera ischii, and the intervening masses of bone. These arches have been called the femoro-sacral and the ischio-sacral (Morris).

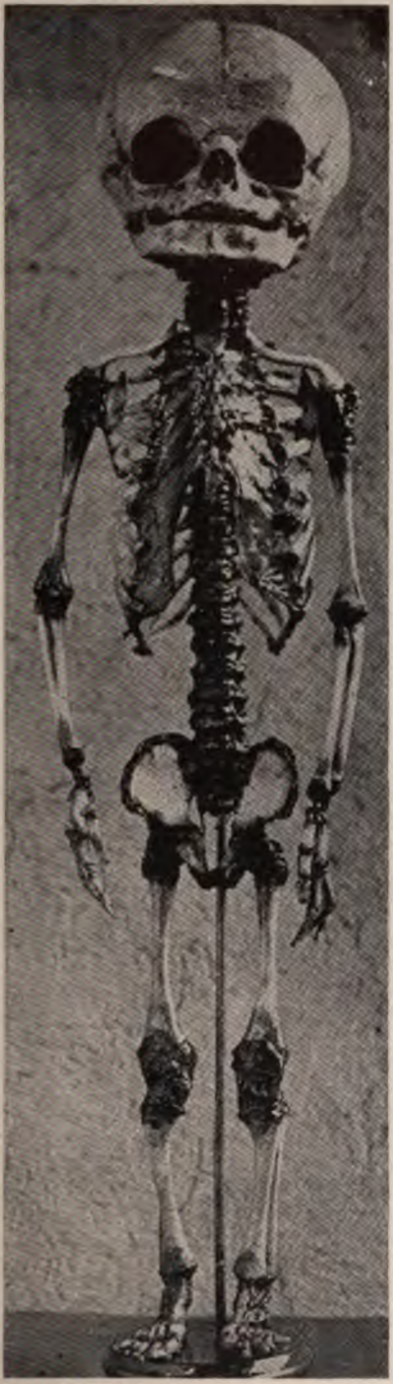
The sacrum and the symphysis pubis are common to both arches.

In rickety deformity of the pelvis, it yields in front at the symphysis, which is pushed forward, and the two acetabula approach each other, the cavity of the pelvis becoming greatly contracted. When this deformity occurs in young infants it has been ascribed to contraction of the ilio-psoas, erector spinæ, gluteus medius, and other muscles.

THE BLADDER.

The bladder is originally derived from the urachus, which is part of the membranous sac appended to the umbilicus in the early fœtal state, and

FIG. 30.



SKELETON OF CHILD ABOUT FIVE MONTHS OLD, SHOWING THE COMPARATIVELY LARGE SIZE OF THE CRANIAL BONES, THE GENERAL CARTILAGINOUS CONDITION OF THE EPIPHYSES, THE SEGMENTATION OF THE STERNUM, AND THE OBLIQUITY OF THE PELVIS. (Photographed by Dr. McClellan.)

called allantois. The allantois appears to be formed at first as a solid mass projecting from the posterior extremity of the body. Very soon this mass becomes hollowed into a vesicle covered with blood-vessels, and is connected with the intestine, which also begins to be formed about this time. This vesicle protrudes through the umbilicus, conveying vessels to the chorion and forming the fœtal part of the placenta. At a very early period the allantois closes beyond the umbilicus. The part within the abdomen widens, to form the bladder, and the tubular formation, the urachus, remains as a ligament on its anterior surface. The Wolffian bodies which precede the kidneys in their development furnish the ureters out of their efferent ducts to the rudimentary bladder.

From this brief account of the formation of the bladder it will be understood that that organ is at first an elongated tube situated in the lower part of the abdomen.

In the infant the bladder is not pyriform, as used to be described, but egg-shaped, having the larger end resting in the pelvis (see Figs. 13 and 22). The upper part is narrowed by the hypogastric arteries, which converge towards the umbilicus. There is no marked fundus or base to the bladder in the young child, and it is situated mainly in the abdomen, the pelvis being small and shallow (see Fig. 29). As the pelvic cavity increases in size, the bladder gradually descends into it, and, the infant about this time assuming the perpendicular attitude, it has been thought that the weight of the urine tends to make the lower part more capacious. Observations upon the dimensions and position of the bladder will naturally vary with the empty or distended state of the organ. Throughout childhood until towards puberty, when the organs of generation are developed and the neighboring parts assume their normal adult relations, the urinary bladder is always so loosely attached to the pelvic walls that, although it may have settled into the pelvis, it will require very little force to push it upward into the abdomen. This lax condition of the bladder-attachments is of great importance in the consideration of surgical interference in this region. In the young child the anterior wall of the abdomen, from the symphysis pubis almost to the umbilicus, is in close relation to the bladder, and the neck of the bladder and urethral orifice is about on a level with the upper border of the pubic symphysis (see Figs. 13 and 29).

The peritoneum is reflected entirely over the posterior surface of the bladder in the child, passing behind the urachus downward to the level of the neck of the bladder, and thence on to the upper part of the rectum. This latter fold (the recto-vesical pouch) usually embraces the prostatic region very closely, and is liable to injury in children during the operation of lithotomy, causing peritonitis, the most frequent fatal termination in that operation.

The anterior surface of the bladder is always uncovered by the peritoneum in children, and when the viscus is distended in adults it is also uncovered for the most part. The bladder is capable of very great distention in adult life,—in fact, after the age of fifteen; and cases have been reported where the summit of the organ reached to the umbilicus, and even to the ensiform cartilage. When it becomes so distended it presses against the anterior wall of the abdomen and dissects the serous membrane away from the parietes. In the adult, when the bladder is empty its anterior surface is covered by the peritoneum down to the symphysis pubis. The capacity of the bladder in infancy is smaller than in after-years, and this may account for the frequency with which young children micturate.

The *prostate gland* is very small in children. According to Sir Henry Thompson, this gland "at the age of seven years weighs only about thirty grains, and between eighteen and twenty years it weighs two hundred and fifty grains, or nearly nine times as much."

The *urethra* appears to increase slowly in length from birth until puberty is reached. Its canal is more dilatable than is supposed in both adults and children. The meatus is often constricted so that only a small-size catheter or sound can be introduced, but if the orifice is incised quite a large instrument will readily pass. The membranous part of the urethra in children is very long, owing to the smallness of the prostate gland at that period of life. In sounding the bladder in a child, it should be remembered that the urethra lies close to the rectum, and that its walls are exceedingly thin and delicate.

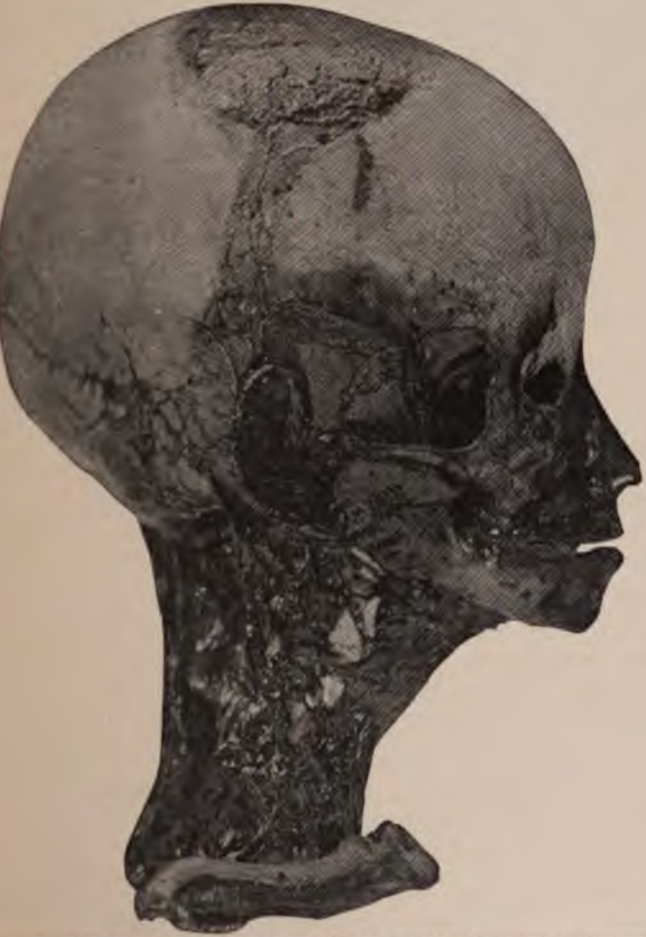
It has been pointed out by recent observers that the condition of the bladder and rectum influences the curvatures of the urethra. If the lower part of the rectum is distended, the prostatic part of the urethra is pushed upward and forward, as well as lengthened (Garson); and if the bladder is filled it is shortened (Symington). On the whole, the degree of curvature is greater in the child than in the adult, but there are variations in this respect naturally following, as do those pertaining to the contiguous parts, upon growth or immature development. The female urethra is embedded in the anterior wall of the vagina, which is sometimes of large size in childhood, and corresponds to the upper part of the prostatic part of the male passage. It is very distensible.

THE PENIS.

In children the skin over this organ is very thin and loose, as should be remembered in performing the operation of circumcision. The mucous membrane is very apt to become adherent to that over the glans penis, so that most male infants, if not properly attended to after birth, are liable to the condition of phimosis. This may give rise to accumulation of secretion beneath the prepuce, and cause vesical irritation, and other distressing symptoms, through reflex involvement of the prostatic nerve plexus, or by direct impression upon the pudic nerve.

The penis is often the seat of arrested development,—*hypospadias* being the condition when the interior wall of the urethra and relative part of the corpus spongiosum are wanting, and *epispadias* when there is a deficiency in the superior wall of the canal and adjacent parts of the corpora cavernosa.

Fig. 16.



Photographed from Preparation in Mütter Museum, College of Physicians of Philadelphia, showing Lymphatic Vessels of the Vertex of the Skull and of the Face and Neck, in a Child Six Years old.

The scrotum in children varies with the general condition of the health. It will be noticed to be smooth and pendulous in strumous and enfeebled states of the system, while in the healthy and vigorous the rugæ are pronounced, owing to the contractions of the muscle-fibres in the dartos.

THE RECTUM.

The lower part of the large intestine is situated in the abdomen in infancy, and its name *rectum* is more applicable then, for it is more truly straight at that period than it afterwards becomes on the full development of the pelvis and its other proper contents. To this vertical position of the rectum has been ascribed the frequency with which children suffer with prolapsus ani. The sacrum is nearly straight in children, and this probably has to do with the direction of the bowel and its liability to prolapse. Examinations of frozen sections of young children have revealed that the second portion of the rectum is very short, and it has been inferred that if this is distended the distinction between the upper two portions is obliterated and the whole of the rectum is comparatively straight (Cunningham).

The peritoneum is reflected over the upper portion of the rectum, and is relatively to the adult condition lower down in children. It is in all cases not so low behind as it is in front, where it forms the recto-vesical pouch. The attachments of the rectum to the surrounding parts do not extend so high in children as in older persons. As age advances, the three portions of the rectum approximate the conditions and relations found in the adult.

DEVELOPMENT OF THE ORGANS OF GENERATION.

The *rôle* which is played by the Wolffian bodies is one of the most interesting of the facts in embryology. In referring to the urinary organs it was noted that the Wolffian bodies take no part in their formation, except to contribute from their ducts the ureters. Prior to the appearance of the kidneys, two small oval masses of blastema are placed on the inner border of the Wolffian bodies. These are the rudimentary testes or ovaries, whichever they are to become; and as the kidneys are formed just above them and grow rapidly they are pushed outward. After the kidneys become established the Wolffian bodies shrink away, leaving only mere vestiges of their lower parts, which consist of two sets of canals known respectively as the Wolffian and Muellerian ducts. From the Wolffian ducts are formed the epididymis and vasa deferentia; and from the Muellerian ducts are produced the vagina, uterus, and Fallopian tubes. The sexual differentiation does not take place until about the fourteenth week of embryonic life, and depends upon the development of one or other of these sets of fœtal ducts.

THE TESTICLES.

It will be seen from the above account that these organs are formed within the abdomen in the lumbar region, and occupy the position below the kidneys about the seventh week of fœtal life. They lie behind the peritoneum, pouches of which pass down into the scrotum, as has been stated in the note upon inguinal hernia, and appear to lead the way for the testicles. These organs about the fifth month in the embryo begin to leave their original position, and gradually descend to opposite the internal inguinal opening in the extra-peritoneal fascia, which they reach about the seventh month. At the eighth month they are usually found within the scrotum, the peritoneal prolongations becoming their vaginal tunics already described. The testicles may be retained within the abdominal cavity, or may lodge for varying periods of time, or for life, in the inguinal tract.

THE OVARIES.

The ovaries, like the testicles, are at first situated in the abdomen, and in the process of development also descend, but only so far as the brim of the true pelvis in new-born infants. After birth they gradually reach the side wall of the pelvis and become relatively near to the external iliac arteries.

THE UTERUS.

Until the approach of puberty this organ exists as an undeveloped rudimentary body placed between the bladder and the rectum in the upper part of the pelvis (see Fig. 29). In the child it is wholly unlike what it becomes in the adult, not only in size, but also in its external and internal configuration. The cervix is longer, thicker, and firmer than that of the body. In truth, there can hardly be said to be any body to the organ in early life, for the arbor vitæ reaches to the top of the uterus, and there is no internal os. The upper portion is generally thinner and more flexible, and may be considered as representing the body. About the time of puberty the uterus undergoes rapid changes and acquires its adult character, the body growing faster than the cervix, together with the development of its appendages. The histology and physiology of this organ have been laboriously studied by numerous investigators, and the result of their views inclines to the belief that the uterus is normally anteflexed.

The state of the bladder and rectum will have much to do in determining the position of the uterus. The attachments of the uterus, consisting mainly of peritoneal folds to the rectum, bladder, and pelvic walls are so arranged as ordinarily to allow a greater mobility of the organ in girlhood. Observation made upon the cadaver with the bladder gradually distended demonstrates the manner in which the vesico-uterine fold of peritoneum acts upon the body of the uterus, raising up the fundus. In cases of acute retroflexion this fact may sometimes be of service. The mucous membrane lining the uterine cavity appears to resemble the so-called adenoid tissues, and has been carefully studied recently by Johnstone, who calls it the "menstrual organ" because of its proper function which is established at puberty.

THE PERINEUM.

In children the anatomy of this region varies from that of older persons mainly in the changes relative to the rectum and bladder and to the general looseness of the fasciæ at the outlet of the pelvis. The operator for stone in the bladder in childhood who selects this region should remember that the pelvis is narrower in them than in the adult; that the neck of the bladder is high up, and capable of being pushed up still higher; that the bulb of the spongy portion of the urethra is very small; and that the recto-vesical fold of the peritoneum embraces the prostate gland, which is only in a rudimentary state. From these considerations it is imperative that the knife should be entered close to the median raphé, if lateral lithotomy is chosen, upon a staff held closely under the pubes, and, with the rectum, previously emptied, pulled downward by the finger of the other hand, cut outward to a point midway between the anus and tuber ischii. In children it is better to cut well into the neck of the bladder, in order to avoid opening up the pelvic fascia by passing beyond the prostatic area. In cases where the stone proves large it is well to cut across the median line, keeping within the limits of the other prostate lobe. In all operations for stone in the perineum the bulbar artery and the rectum are the chief parts to avoid wounding.

THE SPINE.

In the embryo, the vertebral column begins to become cartilaginous in the sixth or seventh week. There are, generally speaking, three nuclei or centres of ossification in each typical vertebra,—one for the body and two for the arches or laminæ and transverse processes. The deposit of bone in the laminæ of the vertebræ commences above and proceeds gradually downward; hence we often meet with cases of spina bifida in the lower part of the column in consequence of arrest of development in the lumbar arches and upper part of the sacrum. Ossification of the bodies of the vertebræ, on the other hand, usually begins about the centre (the ninth dorsal vertebra), and extends upward and downward. A recent observer states that there are some fifteen segments in the coccyx of the fœtus from the fifth to the ninth week. These become subsequently absorbed or coalesce into the four permanent vertebræ belonging to the coccyx.

At birth the infant's spine is quite straight, serving merely to connect the head, limbs, and ribs, and as a protecting column to the spinal cord. It is very flexible at this time, and totally without the important factors of gravity and muscular contraction, which, as the child begins to sit and stand and walk, tend to produce the characteristic curvatures in the neck, back, and loins. These curvatures are not fully developed until adult life, and, as the spinal column owes to them its elasticity and power of withstanding various forces communicated to it, they are deserving of special attention. In the back of the young child, especially if it be delicate or the subject of rickets, there will always be noticed a general curving of the column backward. In fact, it may be said that this convex curvature of the back that which persons naturally assume when feeble or weary at any period o life, and habit or occupation often makes it very pronounced. The dors curvature and the pelvic curvature, made up of the sacro-coccygeal vertebr are the natural ones found in an infant. In the embryo, at the very begi ning of the formation of the column it assumes this dorsal convexity, an as soon as the sacral promontory is developed it is only modified by t addition of the sacral and coccygeal curves.

The normal curvatures of the spine are maintained to a great exte by the disks of intervertebral substance, which are most developed in t regions where most movement is allowed. The intervertebral substance composed at its circumference of fibrous tissue and fibro-cartilage, and its centre of a soft, pulpy matter. The disks act as buffers, and resi shocks to the spine. The natural curves are all antero-posterior, with very slight lateral one to the right in the dorsal region. The erector spin mass of muscles are inserted into the spines and transverse processes, a tend to establish equilibrium. The motions of which the spine is capab are lateral, antero-posterior, and rotatory. The greatest degree of rotati and lateral flexion is found in the neck and loins. Structural changes a unequal muscular exercise produce deformity. When the curvatures a exaggerated, they take the names of cyphosis, lordosis, and scoliosis, a cording as the convexity is directed backward, forward, or laterally. T first deformity is seen in rickets and in caries of the vertebræ. The lordos or saddle-back, is determined very often by inflammation of the hip (coxalgia), and the scoliosis, the most frequent, is generally met with amo young men and boys who assume vicious attitudes. It is an invariable ru with regard to spinal deformities that if we have a weakness occurring at point which occasions deviation there will arise compensating deviatio above and below it. In marked cases there will occur also a rotatory curv ture, caused by contraction of the slips from the longissimus dorsi whi are inserted into the angles of the ribs. This may be so powerful as place the transverse processes in the site of the spine.

Lateral curvatures in the dorsal region occur about the fourth or fif vertebra.

THE LIMBS.

The earliest traces of the limbs in the embryo are found about the fif week. They undergo rapid metamorphoses so that about the eighth we not only are there rudimentary arms and forearms, thighs and legs, but al a separation of digits for hands and feet. The lower limbs are a little lat than the upper in their formation.

Of the skeleton generally it may be said that the bones composing it not acquire their final complete development until adult age is reach During childhood there are marked changes occurring in the bony framewo at various periods.

The growth and development of bone constitute at all times an importa subject, which the surgeon may have to contemplate, but in no connection can they be more suitably considered than in the limbs of children. From the time of Berzelius the chemical composition of bone has been described as one-third animal matter and two-thirds earthy matter, but it has been found by recent observers that the relative proportion of these constituents differs in the different bones. There is a great difference of opinion about the variable quantity at different ages, but it has been pretty clearly demonstrated that there is no change in the proportion in the individual bones from infancy to old age, and that rickets and osteomalacia are due not to a change in the bony composition, but to an inherent diathesis, as scrofula or syphilis. In other words, it is rather the quality than the quantity of the constituents which occasions the peculiar characteristic lesions of bone in these affections. The proportion of animal matter is usually described as being in excess in the bones of children, and the earthy elements are supposed to increase with advancing age, but it should be recollected that the bones during infancy are exceedingly vascular, and that it is almost impossible thoroughly to remove the vessels in order to determine by experiment the exact proportion of the constituents of bone at that period of life.

In the fœtus the skeleton is mapped out for the cranial bones in membrane, and for the long bones of the limbs in cartilage. A long bone affords the best example of the process of ossification, for it may be said to depend upon both membranous and cartilaginous formation. The process begins in the centre of the shaft of the bone (diaphysis), and proceeds towards the extremities (epiphyses), which remain cartilaginous until some time later, when centres of ossification occur in them and the process involves them also. The extremities are separated from the shaft by a layer of epiphyseal cartilage until the growth of the bone is completed (see Fig. 30). Simultaneously with the ossific changes in the centre of the cartilage of the fœtal bone, a very vascular membrane is developed around the shaft. This is called the *periosteum*, and consists of two layers which serve as a nidus for the ramifications of vessels which pass from it into the bone. In young children it is thick and very vascular, and is only connected at the epiphyseal cartilages at either end of the shaft, being separated from the latter by a layer of soft blastema containing "osteoblasts," from which ossification proceeds on the surface of the growing bone. Green-stick or incomplete fractures, which occur sometimes in children, are probably due as much to this condition of the periosteum as to the apparent excess of animal matter in the bone itself. Later in life the periosteum is thinner, less vascular, and more adherent to the surface of the bone. The tendons and ligaments have firmer hold upon the bones, because they become incorporated and continuous with the periosteum at their attachment. Cartilage is now considered as only a temporary substitute for bone in the early stages of the formation of the skeleton.

Bones grow in length by deposition taking place from the ends towards the centre. The shaft increases in circumference by deposition from the periosteum on the external surface, while the medullary canal is produced by absorption from within. Owing to the ends of a long bone having separate centres of ossification, and the interposition of the layers of cartilage between them and the shaft until its full length is attained, the bone i indurated in the parts where the greatest strength is required, whilst th longitudinal growth is facilitated.

About the centre of the shaft of the long bones there is a large foramen leading obliquely into the medullary canal, which accommodates the medullary artery, usually a branch of the main artery of the part of the limb to which the bone belongs. This medullary artery sends branches upward and downward through the marrow, which are generally considered to anastomose with the arteries of the cancellous and compact tissues. Th veins emerge from the long bones by foramina at the extremities and upon the surface of the shaft and by the foramen for the medullary artery Owing to the veins being enclosed in the bony tissues, their coats are exceedingly thin, and consequently in cases of amputation where there i suppuration there is danger of purulent absorption. In children and young persons, where the periosteum is thick enough to admit of being dissected readily from the bone, it is advisable to make a flap of the periosteum to cover over the end of the bone before adjusting the skin and muscle flap of the stump.

The medullary canals of the long bones in the infant are filled with a reddish, oily fluid. By degrees this becomes transformed into a fatty substance of a yellow color.

THE UPPER EXTREMITY.

There is very little about the scapula, which, together with the clavicle the upper end of the humerus, and the structures surrounding them, form the region of the shoulder, that is peculiar to childhood, other than that th several centres of ossification from which it is developed are not completed in their office until about the seventeenth year. The acromion is rarely joined to the bone proper before the twenty-second year, and sometime there is only a fibrous connection between it and the spine, which may giv rise to supposed fracture. It is worthy of note that the latissimus dors muscle passes over the inferior angle of the scapula, and is usually attached to it, so that the bone is held in contact with the thorax. As a result from injury, the angle may slip from beneath the muscle and project under th skin. In poorly-nourished children, where the muscles are flabby, th scapulæ often jut like two prominent wings.

The clavicle is peculiar in that it is not only the first bone in the skeleton to ossify, but that ossification in it begins in its primary fibrous substanc before the deposition of cartilage. At birth the entire shaft is bony although the ends are cartilaginous. The sternal end is the sole epiphysi to the clavicle, and it is joined to the shaft about the twenty-fifth year. It i rarely separated from the shaft by accident, owing to the close ligamentou attachments of the sterno-clavicular joint, but the powerful pectoralis major muscle might produce displacement in a young person. This bone is frequently the seat of green-stick fracture, owing to the loose and exceedingly thick periosteum which surrounds it in the young, as well as its more early ossification. The presence of the protecting periosteum renders injury to the subjacent structures in case of fracture of the bone in children very improbable.

The humerus at birth is nearly ossified in its whole length, the extremities being entirely cartilaginous. Ossification does not commence in the head of the bone before the second year, and in the tuberosities in the third year. Generally there is one centre of ossification for the two tuberosities, but there may be one for each tuberosity. About the fifth year the centres for the head and tuberosities become joined, and form the upper epiphysis, which is not united to the shaft until the twentieth year, and sometimes later. The lower end of the humerus is developed by a centre of ossification in the radial portion of the articular surface about the third year, and another centre for the inner articular surface appearing as late as the twelfth year. The inner condyle is formed about the fifth year, and the outer condyle in the fourteenth year. In the sixteenth or seventeenth year the condyles and articular surfaces, having joined, unite with the shaft.

In early childhood the ligaments and tendons about the joints are stronger and more resisting than the contiguous bone. Falls upon the shoulder or upon the arm held close to the side of the body in children may result in detachment of the upper epiphysis of the humerus. In one case within the writer's practice, a little girl, aged five years, sustained a fracture of the shaft of the humerus, with separation of both the upper and lower epiphyses. This form of accident is commoner in childhood than fracture of the surgical neck of the humerus, and therefore the importance generally given to the distinction between this and the anatomical neck intended to indicate the danger of fracture of the former rather than of the latter is unnecessary, until after the consolidation of the shaft with the epiphysis at puberty. Arrest of development of the arm-bone may follow upon interference with either epiphyseal line, the upper one of which is at the base of the great tuberosity and the lower just above the condyles.

Lifting children by the arms is a hazardous proceeding, and the reason why it is so infrequently followed by dislocation or epiphyseal detachment is probably due to the ligamentous function of the long tendon of the biceps muscle and the protecting influence of the deltoid. There is a large bursa under the deltoid and over the insertions of the supra- and infra-spinatus muscles, which sometimes communicates with the shoulder-joint. Acute arthritis in infants may be due to strain or wrench involving the bursa or inciting epiphysitis, which involves the joint-structures. It is less common in the shoulder than in the hip.

The Elbow.—Owing to the cartilaginous condition of the olecranon process of the ulna, which forms a hinge with the lower end of the humerus, the elbow-joint in childhood owes its main strength to the lateral ligaments and the tendons of the muscles which pass over it. Just belo the external condyle of the humerus there is a pit or dimple in the skin the child when the elbow is extended. This pit is an important landmar as the head of the radius can be felt rolling in pronation and supination the forearm. There is a large bursa over the olecranon and another benea the insertion of the triceps, neither of which ordinarily communicates wi the joint. It is well to note that the epiphyses which meet at the elbo joint unite with their shafts earlier than those at the opposite ends of t bones; also that the foramina of the medullary arteries are directed *towar* the elbow.

The development of the ulna occurs from three centres,—one for t shaft and one for either end. At birth the ends are entirely cartilaginou The olecranon does not begin to ossify until the tenth year, and it is join to the shaft about the sixteenth year. The lower end ossifies in the four year, and joins the shaft in the twentieth year.

The radius is developed from three centres. The head is ossified in t fifth year, and joins the shaft about the eighteenth year. The lower end ossified during the second year, but does not unite with the shaft befo the twentieth year.

The Wrist.—The bones of the wrist are all cartilaginous at birth, a they become ossified at varying periods, as follows: the os magnum in t first year, the unciform in the second, the cuneiform in the third, the trap zium in the fourth, the semilunar in the fifth, the scaphoid in the sixth, t trapezoid in the seventh, and the pisiform not generally before the twelft year. The bursæ at the wrist do not connect with the joint, but are in rel tion with the adjacent tendons.

The metacarpal bones and the phalanges are usually composed of a sha and an upper epiphysis. The shafts are ossified soon after birth, and t epiphyses are all united about the twentieth year.

THE LOWER EXTREMITIES.

The hip-joint is so deeply placed and thickly covered by soft parts th it is more exempt from acute inflammation in childhood than other joint It follows, however, from its peculiar construction that when disease attack the hip-joint, and is attended with formation of pus, the destructive change are very great, because the pus is so pent up that it is long before it reache the surface. The capsule is thinnest in front and behind, and in cases o effusion into the joint the swelling first shows itself in these localities.

In chronic hip-disease in children the limb assumes certain false posi tions, which have been described by Treves as follows: first, the thigh i flexed, abducted, and a little everted; associated with this there is apparen lengthening of the limb, then lordosis of the spine occurs, then the thig becomes abducted and inverted, and incident to this there is apparent short ening of the limb; this is followed by real shortening of the limb.

The disease, when it begins in bone, usually involves the epiphyseal lin that unites the head of the femur to the neck. This epiphysis is joined to the shaft about the eighteenth or nineteenth year. In hip-disease pain is referred to the front of the knee by the anterior crural nerve, and to the back of the joint by the obturator and sciatic nerves, these nerves supplying both joints.

The three bones composing the acetabulum are united by a Y-shaped cartilage until the age of puberty, and in some cases of destructive hip-disease may become separated; and there is a large bursa between the front of the capsule and the tendon of the ilio-psoas muscle. It communicates with the joint. There are also bursæ between the great trochanter and the gluteus maximus muscle, and over the tuberosity of the ischium. Neither of these is normally connected with the joint.

The acetabulum is generally completely ossified by the eighteenth year.

The femur is developed by five centres, one for the shaft, one for each extremity, and one for each trochanter. It begins to ossify next after the clavicle in the fœtus. The centre for the lower end of the bone appears just before birth, and forms the condyles and tuberosities.

The head of the femur begins to ossify about the end of the first year, the great trochanter about the fourth year, and the lesser trochanter between the thirteenth and fourteenth years. The epiphyses are not joined to the shaft until after puberty,—the first being the lesser trochanter, the next the greater trochanter, the next the head, the lower extremity (which is the first to show ossification) not consolidating with the shaft before the twentieth year. On account of the length of time which the lower epiphysis takes to ossify, the growth of the femur is greatest in the lower part of the shaft. In operating for knock-knee or bowed-leg, it is well to avoid interfering with the epiphyseal cartilage, so as not to modify the growth of the limb.

The tibia and fibula, like the bones of the forearm, have three centres of ossification. The centre for the upper end of the tibia appears at birth, and is completed and joined to the shaft about the twentieth year. The lower end shows ossification in the second year, and joins the shaft in the eighteenth year. The upper end of the fibula begins to show ossification about the fourth year, and unites about the twenty-fifth year. The lower end appears in the second year, and unites about the twentieth year.

About the knee there are many bursæ; in fact, they are placed between the bony prominences and tendons of all the muscles attached in the neighborhood of this joint. Some of these communicate with the articulation, but those which most frequently do so are the bursæ which are placed between the quadriceps and the femur and between the ligamentum patellæ and the tubercle of the tibia. The first of these is just above the pouch of synovial membrane which extends above the patella, beneath the extensor tendon. Joint-disease is very apt to affect the knee, owing to its exposed position. In neglected cases, the hamstring muscles are frequently contracted, owing to their receiving branches from the great sciatic nerve, which also supplies the joint. In some cases of progressive disease, the contraction of these muscles will produce a partial luxation by drawing the tibia ba ward.

When effusion takes place in the ankle-joint, the swelling first appe in front beneath the extensor tendon, and afterwards behind on either s of the tendo Achillis. The lateral ligaments are too strong to be affec by effusion within the joint.

The Foot.—The normal arch of the foot is in great measure maintai by the central part of the plantar fascia. It is exaggerated by mar contraction of this fascia in club-foot, such as congenital varus and tali equinus. In order to divide the membrane, the knife should be introdu from the inner side, so as to avoid the external plantar artery, abou finger's-breadth from the os calcis.

In dividing the tendo Achillis, the knife should be introduced a finge breadth above its insertion.

The posterior tibial vessel runs midway between the os calcis and internal malleolus. The tibialis posticus tendon may be easily divi above the base of the inner malleolus. It can be cut also on the side the foot between the annular ligament and the scaphoid bone. The tend of the tibialis anticus may be divided in front of the ankle or at its insert into the internal cuneiform bone.

The tarsal bones, excepting the os calcis, all have one centre of ossifi tion. The os calcis has an epiphysis which appears during the tenth y and unites with the rest of the bone after puberty. The order in wh the ankle-bones ossify is as follows: the body of the os calcis in the si month of fœtal life, the astragalus in the seventh month, the cuboid in ninth month, the external cuneiform in the first year, the internal cuneifo in the third year, the middle cuneiform in the fourth year. The metatar bones and phalanges have each one centre for their shafts and one for th epiphyses. The epiphyses appear about the sixth year, and become uni between the eighteenth and twentieth years.

In conclusion, the author ventures to state that the illustrations, m of which are original, are adapted from his forthcoming work on Anato The photographs of living children, indicating topographically the positi of the thoracic and abdominal organs and the important surgical landmar are intended to serve as diagrams. The rise and fall of the diaphragm a the distention of the stomach during digestion render it difficult, howev to map out with accuracy the position of the viscera on the living body.

THE PHYSIOLOGY OF INFANCY.

By ANGEL MONEY, M.D.

PRELIMINARY REMARKS.

The more one studies the literature of the physiology of infancy and childhood, the more the truth is forced home on one that but little that can be of value in practice is known.

Nor are we likely much to advance in our really useful knowledge until further steps in the direction of a molecular physiology have been made.

Few of the text-books upon children's diseases have much space allotted to the study of physiology. The growth and development of the infant are subjects of importance; the chemistry of its secretions bears upon the great question of dietetics; the study of its excretions gives us an index to its tissue-changes. The guide to the maintenance of its health is a knowledge of its physiology.

Whilst there is much that is of absorbing interest to the student of biology in what has already been ascertained of the physiology of infancy, most of it is of a theoretical or not clearly applicable nature, and moreover would require a large volume to expound. In this work nothing but what has practical bearings or actual utility can be given, for the author prefers to present a clear if meagre outline rather than a confused picture of details.

GROWTH.

The new-born male child measures on an average 50 centimetres, and the female 49. The rate of growth in length has been determined by Liharzik to follow a certain law, increasing seven and a half centimetres in certain spaces of time, following an arithmetical series, so that at the end of the first month the body measures 57½ cm. in length; at the third month, 65 cm.; at the sixth month, 72½ cm.; at the tenth month, 80 cm.; at the fifteenth month, 87½ cm.; at the twenty-first month, 95 cm. During the next arithmetical series, from 21 to 276 months, the increase is at the rate of five centimetres per interval, the number of intervals being seventeen. The interval required for the added unit of growth is seen to increase in arithmetical progression.

Growth in Weight.—Three thousand two hundred grammes is gi as an average weight of the full-term male child, and two thousand n hundred for the female. But this seven pounds is often exceeded. natural loss of weight occurs for three or four days after birth, being rat more than six per cent. of the body-weight. In grammes the total loss been estimated by Haake, Winckel, and Quetelet, at 222. Then follo an increase in weight which appears to vary greatly; for some assert t the growth is gradual and regular, whilst others have observed it to t place by leaps intermittently. Sometimes much flesh is added during second month of life; but the fourth month often witnesses the great rate of increase in weight.

Russow noticed a considerable difference in the increase in weight infants according to the mode of rearing them: in the breast-fed growth is more steady and always transcends that of the hand-fed.

Speaking generally, the weight of the body is doubled by the fi month and trebled by the twelfth; Russow avers that hand-fed babes not treble their birth-weight till the second year of life; the same differe obtains in after-years, so that a child four years old which was suckled the mother (for the usual period) weighed generally two thousand gram more than a child who was artificially fed from birth.

In weighing infants, allowance must be made for the passage of ur and fæces, so that a difference of 20 to 30 grammes counts for nothing.

Whilst using weight as a guide for appraising the welfare of childr it should be remembered that rickety and scrofulous children are of heavy from a richness in fat: we should therefore compare the growth weight with that in stature.

Estimating the weight at birth as three thousand one hundred gramm Hähner's investigations gave the following results showing the daily a monthly rate of increase:

1st	month,	3835	grammes,	showing an	increase of	735	at the daily	rate of	24.5
2d	"	4930	"	"	"	1095	"	"	36.5
3d	"	5540	"	"	"	610	"	"	20.3
4th	"	6010	"	"	"	470	"	"	15.6
5th	"	6680	"	"	"	670	"	"	22.3
6th	"	7005	"	"	"	325	"	"	10.8
7th	"	7680	"	"	"	675	"	"	22.5
8th	"	8100	"	"	"	420	"	"	14.0
9th	'	8370	"	"	"	270	"	"	9.0
10th	"	8680	"	"	"	310	"	"	10.3
11th	"	9170	"	"	"	490	"	"	16.3
12th	"	9470	"	"	"	300	"	"	10.0

Zeising's table shows that the absolute growth in length of the wh of the members of the body is greatest during the first triennium; t smallest growth in length of most parts of the trunk occurs in the thi triennium. The increase in length of all parts in the first fifteen years much greater than the further growth of the body up to its completi

A comparison between the gain in weight and the growth in length is instructive, since these increments seldom proceed in close relationship to each other, though a certain parallelism between the two should be observed. An increase in weight often precedes further growth, and rapid growth is often interrupted by an increase in flesh.

Generally the gain in weight after birth is more evident than the increase in length; these two elements of growth have no very definite proportion to each other, but a certain relation between them may be observed. A vigorous child generally gains four pounds in weight in three months, and an increase of a pound every month causes the child to double its birth-weight in five months, and treble it in twelve. After the fourth day the body grows in weight at the rate of three ounces for the second week, four ounces for the third, five ounces for the fourth, and during the second month an ounce a day is about the right quota of growth in weight. In the third and fourth months about five ounces a week is the amount; this drops in the next three months to an average of three ounces a week; then about the teething period a slight pause in growth and weight may be noted. Growth in length may be accompanied by a slight falling off in weight. In spring and early summer the length of the body increases often in a marked manner, recalling something of a similar process in the vegetable world; in northern latitudes, growth in children seems to occur at these periods only.

A child in health generally gains twenty pounds in weight and ten inches in height in the first two years of life; in the third year four pounds and four inches are about the usual additions to the weight and stature. During the next six years the body increases by annual increments of four pounds in weight and two or three inches in height. After ten years the body puts on flesh at the rate of eight pounds a year.

AVERAGES OF HEIGHT AND WEIGHT OF BOYS AND GIRLS OF ENGLISH-SPEAKING RACES; CALCULATED BY DR. WILLIAM STEPHENSON FROM TOTALS OF BRITISH AND AMERICAN STATISTICS.

BOYS.

Age. Years.	Height in Inches.	Gain in Height.	Weight in Pounds.	Gain in Weight.
5	41.30	. .	40.49	. .
6	43.88	2.58	44.79	4.30
7	45.86	1.98	49.39	4.60
8	47.41	1.55	54.41	5.02
9	49.69	2.28	59.82	5.41
10	51.76	2.07	66.40	6.58
11	53.47	1.71	71.09	4.69
12	55.05	1.58	76.81	5.72
13	57.06	2.01	83.72	6.91
14	59.60	2.54	93.46	9.74
15	62.27	2.67	104.90	11.44
16	64.66	2.39	120.00	15.10
17	66.20	1.54	129.19	9.19
18	66.81	.61	134.97	5.78

GIRLS.

Age. Years.	Height in Inches.	Gain in Height.	Weight in Pounds.	Gain in Weight.
5	41.05	. .	39.63	. .
6	42.99	1.94	42.84	3.21
7	44.98	1.99	47.08	4.24
8	47.09	2.11	52.12	5.04
9	49.05	1.96	56.28	4.16
10	51.19	2.14	62.17	5.89
11	53.26	2.07	68.47	6.30
12	55.77	2.51	77.35	8.88
13	57.96	2.19	87.82	10.47
14	59.87	1.91	97.56	9.74
15	61.01	1.14	105.44	7.88
16	61.67	.66	112.36	6.92
17	62.22	.55	115.21	2.85
18	62.19	. .	116.43	1.22

About the ninth year in girls and the eleventh in boys there is a perio of diminution in the rate of growth, and in the thirteenth year in girls an the sixteenth in boys the activity of growth is at its greatest, correspondin to the assumption of womanhood and manhood by the processes associate with puberty. Dr. Stephenson's article in the *Lancet*, September 22, 1888 gives interesting information of the relation of weight to height during adolescence. Stature increases steadily with age, but not at a uniform rate, an the weight also increases with age, but not uniformly or in arithmetical proportion to the height. The ratio of weight to height increases with age, s that whilst between five and six years the weight of the inch is one pound at eighteen it is two pounds. After various workings the following law wa found to hold good: between five and eighteen years inclusive the weigh varies directly as the height squared, and inversely as the amount by whic the age falls short of a certain number that can be easily ascertained.

Let H represent height, W weight, n age, and m the ascertained number The formula is $\frac{H^2}{m-n}=W$. As the rate of growth is not uniform, th value of m is not constant. It can be readily found for each year from th typical standard of the formula $\frac{H^2}{W}=m-n$.

Reclus and others have observed febrile appearances during rapid growt of the body, but it must be questionable whether there was not some obscur disturbance exciting the fever, for Bouilly states that pains about the growing ends of the bones attend the rapid rate of growth in the limbs. Nevertheless the facts deserve attention, as does also the interesting growth of th body in length during acute febrile processes which cause wasting: perhap the actual pyrexia stimulates the growing ends of the bones to increase activity, and the increase in length of single bones from chronic inflammation deserves consideration in this connection.

The richness of the muscles and other tissues in glycogen grow rapidly less after birth. The muscles of the infant are richer in water and poorer in myosin than in adults, there being more extractive matters, fats, and inorganic constituents. The muscles are very poorly developed in the new-born, so that great absolute and relative increase takes place during childhood and youth. Fatigue is more easily induced in children than in adults. This may be due to a greater production of metabolic products. Ranke believes that sarcolactic acid is the material on which the tired feeling depends; the reaction of working muscles should be less alkaline than in adults, owing to the greater production of acid products. The elasticity of muscle should also be less; muscular activity produces relatively more heat and less mechanical motion; rigor mortis occurs and disappears more rapidly.

The circumference of the head of the new-born child averages 35 cm. for the male and 34 for the female. Again Liharzik's law of growth steps in, and informs us that for each period the rate of growth to the twenty-first month is $2\frac{1}{2}$ cm.; during the second arithmetical series the rate being $\frac{11}{14}$ cm. It may be calculated that at the twenty-first month, or after six arithmetical intervals, the circumference of the head measures 50 cm.

Elsässer showed that the anterior fontanel normally grows in size till the ninth month, and in general it then begins to close, being ossified from the borders and thus filled in on an average at about the age of eighteen months.

The circumference of the thorax of the new-born child averages 31 cm., and the rate of increase follows the same arithmetical progression, the unit of addition being $3\frac{9}{34}$ cm. for six intervals, so that at the twenty-first month the circumference measures about 42 cm.; from this age to the one hundred and fifty-third month the increment is $1\frac{5}{34}$ for each interval, so that the circumference would then be about 55 cm. Then at puberty the rate of increase is $5\frac{3}{4}$ up to the end of the increase in growth. It will be seen that the chest-circumference increases only moderately until the thirteenth year, and then very rapidly, chiefly in correspondence with the development and growth of the lungs and heart.

In growth it will be perceived that the chest-circumference races that of the head, so that normally it beats the latter in the second year, and it is a sign of constitutional disease if the circumference of the thorax does not exceed that of the head in the third year of life.

The numbers thus given must only be taken as approximately accurate, since nationality, individual peculiarity, climate, and food have considerable influence on the process of growth.

Russow showed that the breast-fed infant grew some two to eight centimetres more during the first year of life than the hand-fed.

Fröbelius maintains that the circumference of the chest of the normal new-born must be at least 2.5 cm. less than the head-circumference, and that half the length of the body must exceed the circumference of the chest by 7 centimetres. In both sexes the antero-posterior chest-diameter is equal to the antero-posterior pelvic diameter.

Most parts of the body in the new-born are proportionally wider tl
in the adult.

THE NERVOUS SYSTEM.

In order to study the development of the will in children, we m
observe carefully the movements of the new-born and of the infant.

The congenital movements are purely involuntary and impulsive, be
pure reflex or automatic acts. The incessant small movements of vari
parts of the body, especially of the face,—grimaces (microkinesis),—bel
to this category. They are due to nerve-discharges in the spinal mo
centres, which either spontaneously explode or are made to discharge
insignificant irritations either of their afferent nerves or perhaps by var
tions in the vascular conditions on which their functions depend.

More obvious *reflex* acts exist also, as when movements result from we
marked peripheral impressions,—light, sound, and touch. These acts oc
just as in brainless animals, and indeed are altogether comparable to
movements of a decapitated frog.

The time required for the performance of these reflex movements see
to be somewhat longer than after they have been frequently repeated. It
probable that some reflexes are more perfectly developed at birth than othe
In the first days of life, reflex movements can be obtained from all t
sense-organs,—optic nerve, auditory, olfactory, taste, trigeminal, and cu
neous nerves of the surface of the body. But the stimulation must eitl
be stronger in intensity or affect wider areas than in later life: in the sk
and retina, at all events, a larger number of nerve-fibres must be simu
taneously stimulated in order to obtain an undoubted reflex. The refl
excitability of the skin of the face is relatively greater than that of oth
parts of the cutaneous surface.

A third variety of congenital movements is the *instinctive*, which occu
it is true, after irritation of sensory peripheral nerves, but not with t
machine-like regularity of reflex movements. Laughing as the result
tickling the sole of the foot does not always occur. Sucking is perha
the best example of an instinctive movement. The act of licking is al
similar.

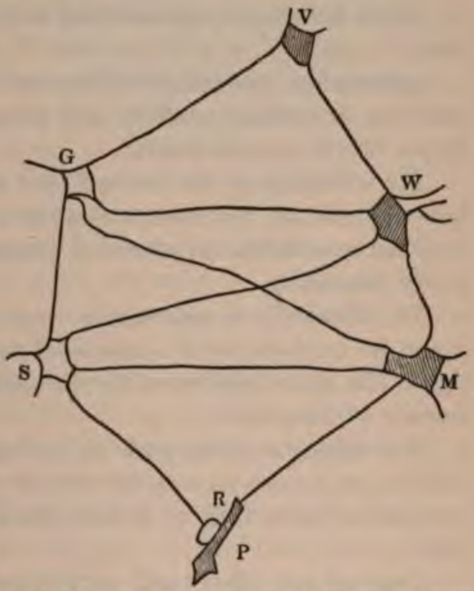
Probably, at birth a pure act of volition cannot occur either in man
in animal.

Willed movements can take place only when the development of tl
senses has gone so far that not only are the qualities of individual sens
tions clearly recognized, but also complete *perception* has become established
by which we must be understood to mean the power of comparing sensatior
and of referring them to their proper causes. Without the power of idea
tion there can be no will, without sense-activity there can be no ideation
so that the will is inseparably interwoven with and dependent on the sense
Only after the first three months of life have passed does evidence exist o
the possession of a will; and then the acquisition does not come suddenly
but gradually.

CLASSIFICATION OF MOVEMENTS IN CHILDREN.

The following schema (after Preyer) will help the study of the various movements:

Impulsive movements involve action only in *M* and *P*, the lowest parts of the motor nervous apparatus, and it is supposed that no recognizable sensory stimulus is required; such movements are also entirely unconscious. In reflex movements the parts involved are those designated by the letters *R S M P*. Instinctive movements necessitate the action of certain sensory impressions and of at least three centres which stand in morphological relation with one another. Lower sensory, higher sensory, and lower motor centres must concert in order to produce the simplest instinctive movement, *R S G M P*, in which first a sensory impression calls forth a sensation, and thence ensues the simple instinctive or reflex movement. It will be perceived that the highest sensory, or rather the perceptive, centre need not be in action, as in sucking, which most probably is entirely unconscious.



R, the termination of the sensory nerves; *S*, the lowest sensory centre; *G*, the highest sensory centre in the cortex; *V*, the perceptive centre in the cortex; *W*, the higher motor centre; *M*, the lower motor centre; *P*, the terminations of the motor nerves.

In more complex movements requiring the aid of consciousness the whole of the centres and paths will be in action, as in imitative movements. It will be inferred also that the highest centres may act as the result of alterations occurring in them without actual impressions from without; and this corresponds to complex movements involving memory and comparison of perceptions.

The following impulsive movements may be observed in the new-born:

Stretching and bending of the arms and legs, like those occurring during intra-uterine life, and recalling the movements of animals awakening from hibernation; they occur also during sleep.

Straightening of the legs immediately after waking, seen repeatedly in the second week; it remains unchanged all through, and may be seen to occur even without waking. Ocular movements occurring before the opening of the eyes when waking are impulsive, and may be seen in adults.

The movements of the new-born in a bath of the same temperature as the uterus can hardly be simply reflex. These movements may be di expressive of pleasure, but they remain even for four months as purpose and as asymmetrical as on the first day of life.

The body may exhibit to-and-fro pushing and drawing movements the second month ; sometimes this movement becomes so constant and l ing as to constitute disease. I have been consulted several times on acco of this movement, which suggested genital irritation.

Facial movements, asymmetrical or symmetrical, may also be seen dur sleep.

Independent movements of the nose—apart from the action of the nasi seen in sucking, snuffling, and deep breathing—were not observed Preyer till the seventh month.

The wrinkling of the forehead and the closure of the eyes in the fi hours of life are not always impulsive, but sometimes of reflex orig Only the remarkably asymmetrical grimaces of the new-born are probal purely impulsive.

The bilaterally symmetrical movements of the face and arm of refl source are much earlier developed and more differentiated than those of t legs. The same holds good for the adduction, abduction, supination, a rotation of the arms.

The waking suckling performs during the third quarter of the first ye striking purposeless movements with the arms, whereas in the legs, wheth the child is in the bath or in bed, alternating extension and flexion is t rule.

Crowing and other vocal sounds are during the first year due to di charges in the motor nerve cells of impulsive, not reflex, kind, just as a the squeaking of new-born animals and the piping of chickens st unhatched.

Certain associated movements require notice because of their seeming involuntary and impulsive character. The extension of the little fing whilst carrying a spoon to the mouth is very common in infants even up the age of three years ; and this may persist till much later. Other ass ciated movements may be observed in the last months of the first year life, and also later.

We all have witnessed the involuntary movements of the hands an fingers which may accompany the act of feeding, and which give the chil the air of eagerness to get food ; the movements involuntarily evoked b music are probably of the same order.

Although at birth many *reflex* movements obtain, yet the reflex excit bility of the new-born is less marked than a little later in life. During th last months of fœtal life reflex activity must be rapidly increasing. Preyer experiments on unhatched chicks, and on immature fœtuses of rabbits an dogs, prove that many reflexes, such as swallowing, and even inspiratio exist before birth. The human fœtus undoubtedly can swallow, since it i clear that the meconium contains some of the contents of the liquor amni in the form of epithelial scales and fine hairs and other parts of the vernix caseosa. At birth a new reflex (automatic?) movement commences with the establishment of respiration. Perhaps the first forcible expiration causing a cry is purely reflex in origin. Sometimes instead of crying a definite sneeze takes place, as Darwin pointed out. In sneezing, as in swallowing, the reflex action involves the co-ordination of many muscles, demonstrating the perfection of development thus early at birth of certain parts of the nervous centres and paths. In sneezing, the eyes of infants are closed every time, just as happens in apes according to Darwin. Donders showed that the filling of the ocular vessels was lessened by shutting the eyes; and thus this reflex act seems somewhat purposive. Champneys noticed flexion and extension movements of the limbs to accompany the act of sneezing in his infant during the first nine months of life.

Sniffing, yawning, and coughing may be noted in the first days of life; snorting was noted on the twenty-fourth day by Preyer; hawking or expectoration, however, has to be acquired, and often its appearance is very late; but involuntary coughing even as early as the fourth day of life has been accidentally attended with expectoration.

The involuntary tossing out of the nipple accomplished by the tongue is a much more skilful movement than is the spitting out of the skin of a grape, though the latter is a voluntary act of later acquisition, which becomes perfected about the nineteenth month.

Sobbing and sighing, which in later life have psychical associations, have in infants not any such meaning; sighing may be noted first in the seventh month, especially on raising the infant from the recumbent into the half-sitting posture.

In early life the respiratory movements have no relations whatever to the emotions: the heaving of the breast in passion, the holding of the breath from attention, and such-like movements, are not to be observed at that period. Indeed, the respiration in the first weeks is very irregular: now stormy, next feeble, and then ceasing, the respiratory rhythm of the newly-born exhibits all the possible physiological and pathological variations. Vomiting easily induced, choking, and hiccough are well-developed reflexes even at birth. Choking movements may be evoked by tickling the palate and root of the tongue, or by applying bitter stuff to the same parts, or by moistening the upper lip with bad-smelling substances. Vomiting may be induced by filling the stomach with water, or by tickling the throat, even in the youngest infants, and eructations from the stomach are quite common. Hiccough is very common during the first three months of life; it has been noticed within twenty hours of birth: even when bad it may be arrested by a teaspoonful of lukewarm sugared water.

Other movements than the above are, however, of far greater psychological importance,—the reflex movements of the eyes and the movements of the head and limbs following cutaneous stimulation. According to Pflüger, if one side of the head be tickled whilst the infant is asleep, the hand of the same side will be raised towards the tickled spot, just as in t case of a decapitated frog stimulating with acetic acid the inner part the thigh causes attempts on the part of the same foot to remove the ir tant. Preyer tried the experiment on his infant at the age of fourteen da and afterwards: sometimes the law of Pflüger held good, but by no mea always, for the right hand might move at the irritation of the left tem (or left side of the nose), and sometimes no response followed. Duri sleep Rosenbach has observed an absence of the abdominal, cremasteric, a patellar reflexes; but this is certainly not customary, though an inequali of the superficial reflexes may obtain on the two sides during sleep. T knee-jerk is almost invariably more ready during sleep, and it may obtained immediately after birth.

The rapid closing and shutting of the eyes at the age of two months a sign of perfected vision, because it shows a perception of rapid moveme on the part of the child. Agreeable sensations tend to cause the eyes to opened wider than under uncomfortable impressions.

The movements of the eyes of the new-born are not properly c ordinated: each eyeball may in the first days of life move independently the other, so that squinting may occur. Perhaps this immaturity is t reason why ento- or ecto-peripheral irritation may cause squinting; for it certain that very slight causes may disturb the motor equilibrium of a nervous centre in infants.

It must take some time before the infant learns to fix an object and exercise accommodation for near vision fully. The young infant may co verge the eyeballs whilst the pupils remain dilated. The function of a commodation, involving convergence and narrowing of the pupil, is n congenital, and many weeks may elapse before these associated moveme are perfected.

Touching the tip of the nose of the new-born child causes both eyes be screwed up; if one nostril only is touched, the eye of the same side on is closed, but if the irritant is stronger both eyes tighten and the head thrown back. In the new-born, touching the conjunctiva, the cornea, or eyelash causes shutting of the eyes; but the irritation of the eyelashes a lids does not always cause this reflex, and these parts are certainly le sensitive than the cornea. If a stream of air is blown upon the face t eyes shut, but only if the cornea, conjunctiva, or eyelids are affected, and t eye most blown upon shuts more quickly and more tightly than the oth From researches on new-born chicks and guinea-pigs, Preyer finds that t closing of the eyes from irritation occurs less promptly than later on. I the suckling eleven days old he noticed that the reflex closure of the li was perceptibly slower than in the grown-up. On the fiftieth day t slightest touch of an eyelid caused rapid shutting. The sensitivity is contrast with the observation that in the first weeks of life, the infant bei in the bath, some lukewarm water may fall on the conjunctiva of the op eye without causing closure.

All new-born birds and mammals appear not to have the power of holding up the head; the newly-hatched chick learns to do this in a few hours, but the human infant takes many weeks before it can perform this act. Since powerful movements may be seen in the neck-muscles in the first and second weeks, it is most probable that the congenital disability to balance the head on the neck is not due to muscular weakness, but simply to a want of co-ordinating power which doubtless has its structural equivalent in the motor centres of the brain proper. Even in the twelfth week the head is not properly balanced, but falls sometimes forwards, backwards, or sideways when the infant is set up. About the sixteenth week the head may be held up in a well-balanced position. That the act is dependent upon an effort, however slight, of the will seems clear from the observation that in sleep the head lolls on one side even in adults. In children of poor development the date of acquisition of this function of balancing the head is postponed by one or more months.

The first attempt to sit up may be noticed about the sixteenth week, but it may be earlier or much later than this; the successful fulfilment of this function also varies, the fortieth week of life being a fairly early date. When first learned, this act, like others, is not very stable, so that slight influences may cause its failure, and a really firm seat does not obtain generally till the tenth or eleventh month. In sitting upright, it may be noticed that the soles of the feet are turned towards each other, recalling the posture of the ape and that obtaining in utero; and even long after birth, when the babe is free from impediments, the intra-uterine position of the limbs may be noted in the adducted legs and flexed and adducted arms.

Attempts to stand may be made as early as the thirty-eighth week of life, but its successful accomplishment is often much later, and varies with the strength and rate of development of the child; by the eleventh or twelfth month standing should be an accomplished fact. With support, attempts to stand may be found as early as the twentieth week, and perhaps earlier (Sigismund).

A few children never creep on the floor: the date of appearance of creeping varies much in different children even in the same family; and the mode of creeping differs in different nations, since in crawling one or both knees may be used. Forward progression on hands and knees does not occur until some time after walking. The movements of putting one leg in front of another are instinctive, and may be witnessed in infants long before they can stand, by holding them out naked, the hands of the holder supporting the child under the armpits.

Walking may be developed as early as the eighth month, but usually it is the twelfth, and it may be much later than this. At the age of nine months most children begin to crawl, and from six to eight months later they will be able to walk by themselves.

The idiosyncrasies of children and families must be remembered; for some children walk long before others can stand, and some run before others walk. Dr. Champneys's child stood upright with some axillary support, soles touching the ground, in the nineteenth week, and it then moved its f forward without irregularity, though the feet were sometimes raised high.

There can scarcely be any doubt that the acts of sitting, standing, cra ing, running, walking, jumping, climbing, and throwing are instinctive origin and do not have to be learned by imitation, the truth being that su acts are not performed at birth simply because of the want of developm of those parts of the brain on which such acts depend.

The muscular sense begins to develop itself probably before birth wh fœtal movements commence, but becomes perfected only with the perf development of the various movements of the body.

A few hours after birth new-born infants react to strong impressio made on the olfactory organs. Although before birth hearing does not exi yet several hours after reflex movements can be obtained as the effect of lo sounds: the imperfection of hearing in the new-born is partly to be account for by the horizontal position of the membrana tympani and by the circu stance that the tympanic cavity is full of a gummy fluid. Immediate after birth the reactions obtained by giving a baby sweet substances are qu different from those following the administration of bitters.

Sight in the proper sense of the word does not exist for the new-bor which can, however, differentiate light from darkness, but only when a co siderable portion of the field of vision is illuminated or darkened. If t brightness is very much stronger than its surroundings, as, for example, bright flame in a dark room, then even in the first weeks of life it is reco nized. The separation of colors is very incomplete during the first months life, and perhaps is limited to the appreciation of unequal intensity of ligh

Yellow, red, and the pure white, gray, and black, are the first to recognized, whereas green and blue are not perceived till much late Probably even when a year old considerable difficulty exists in separatin green and blue from gray; and whilst during the second year of life a chil may name correctly the four primary colors, yet any normal child of fou without special training would find it easier to name them than to recogni mixed tints.

The rapid closure of the eyelids due to the rapid approach of any obje towards the eye is not present during the first weeks of life, it being a refle movement having a defensive object which first originates in consequence o an unpleasant feeling due to changes in the field of vision.

New-born infants possess but feeble perception of light. Exposing babe to the action of twilight five minutes after birth, Preyer observed th eyes to open and shut so that the palpebral fissure at times measured fiv millimetres, and a little later the eyes were noticed to be wide open and th forehead wrinkled. Before the end of the first day it was evident from th play of the features that a difference in the intensity of light was appreciate by the babe. On the second day the eyes rapidly closed on bringing candle-flame near; and on the ninth day the head was energetically turned away from the flame and the eyes tightly closed. The sensitiveness to light was greater in the waking state than immediately after sleep, so that the same object which at one time caused dislike at another excited pleasure. On the eleventh day the infant showed signs of pleasure at the sight of a burning candle and also of a bright curtain-holder. On the tenth day it was noted that the throwing of a strong light on the eyes of a sleeping infant caused contraction of the orbicularis palpebrarum. The pupils of new-born infants soon react to light, but are apt to vary much in size; they may contract to the diameter of two millimetres soon after birth. At the age of two months bright objects excite signs indicative of mirth.

In normal infants by the end of the first week the closure of the eyes as the effect of sudden loud sound may be observed. Preyer finds that it takes at least three-quarters of a year before a child recognizes the tones of a harp; and it is questionable whether it can differentiate the tones properly before the second year. Nevertheless, many children sing before they speak, and they distinguish the noise and sounds of speech long before they can reproduce them. The direction from which a sound comes may be determined by an infant two or three months old.

The sense of touch during the first hours of life is feebly developed, and the sense of temperature is said not to exist, owing to the surface having been kept at a uniform temperature. The new-born is not very sensitive to painful stimulation affecting only a few skin-nerves (as a pinch), yet it cannot be doubted that it knows thoroughly the difference between pleasurable and painful sensations. These defects of sensation are to be ascribed to defective development, not only of the skin, but also of the brain.

Taste is well developed in the new-born, since sweet, bitter, acid, and salty things give different reactions: in this respect the human new-born is better off than many animals. Although this distinguishing power obtains, yet there is but small capacity for perceiving differences in intensity.

Immediately after birth the new-born cannot smell, probably as the effect of the liquor amnii having filled the nasal passages; but a little later agreeable smells can be separated from disagreeable ones.

In the first three months of life the number of pleasurable feelings is not numerous: the staying of hunger, the enjoyment of sucking, the sweet taste of the milk, the pleasure of the warm bath, the joy at beholding bright masses of light, and somewhat later that dependent on the movement of objects before the eyes, the pleasure of undress which provokes lively movements, and also the comfort of being dried after the bath. Acoustic impressions increase the sense of joy in the second month of life. It cannot be till the third month that the infant derives pleasure from recognizing his mother. The first period of life is one of the least pleasurable, since the number of enjoyments and the capacity for the same are of the smallest. With attempts at grasping the infant finds a new source of pleasure in the second quarter of life.

The most powerful factor in the development of the understanding is the capacity the infant has for astonishment and its related emotion, fear.

THE CIRCULATION.

The changes of the fœtal circulation which follow the expansion of the lungs are too well known to need repetition here. There are other facts of difference between the infantile and the adult circulation which are of great physiological and pathological importance. The relation between the size of the heart and the width of the arteries in children is, roughly speaking, the very reverse of what obtains in the adult. Expressed in numbers, the volume of the heart to the width of the ascending aorta is in infants as 25 : 20. Before the onset of puberty the ratio is approximately 140 : 50; after puberty, 290 : 61. From these facts it follows that the blood-pressure in the systemic arteries is much less in children than in adults. The case is very different in the pulmonary circulation. With the cessation of the fœtal circulation there commences a slow widening of the aorta.

In childhood the ascending aorta is relatively to the pulmonary artery much narrower than in later life; the circumference of the pulmonary artery is to that of the ascending aorta, reckoning the length of the body at 100 centimetres, at the end of the first year of life as 46 is to 40; in the adult, as 35.9 is to 36.2. It is held to follow from this difference that the blood-pressure is higher in the child's than in the adult's lungs. Beneke has shown that at puberty the heart rapidly increases in size, so that the aorta becomes relatively narrow and the blood-pressure in the systemic circulation greatly raised. The annual increase in the size of the heart between seven and fourteen years of age is only eight per cent.; whilst during the development of puberty the rate of growth is nearly one hundred per cent. It is interesting to regard this great and rapid growth with corresponding increase in blood-pressure side by side with the great mental, muscular, and sexual changes: it seems clear that the remarkable increase in the activity of man's highest functions demands a more efficient circulation conducted at higher pressure. That the liability of infants "to take cold" and their proneness to collapse from suddenly-acting causes are largely to be attributed to their relatively feeble heart and circulation is highly probable.

THE BLOOD.

The longer the new-born infant remains attached to the placenta of the mother, the larger will be the quantity of blood which passes into its circulation; and indeed it seems that its vessels may become overfull. A considerable disturbance in the distribution of blood occurs after the separation of the child from the mother, and it appears as though the circulatory system did not easily become accustomed to the great alterations of pressure and redistribution of labor: sometimes this natural oscillation passes beyond normal bounds, and hemorrhage from the various surfaces or into the various tissues ensues. When nothing alarming happens, still the researches of

Cohnheim, Zuntz, and others have shown that remarkable alterations occur. During the first days of life some of the fluid of the blood is excreted, so that its mass becomes less, a sort of thickening or concentration taking place. The total quantity of blood in an infant is in relation to the body-weight most probably somewhat less than in adults: this has been variously estimated by different observers. Roughly, the weight of the blood compared with that of the body in the newly-born is about five per cent., or 1 : 19.5, whereas in adults it is about eight per cent., or 1 : 13. The specific gravity of the child's blood is somewhat lower, also, as may be represented by the numbers 1048 : 1055, and this corresponds with a smaller percentage of salts, hæmoglobin (except in the newly-born), albumen, and fibrin. The red blood-corpuscles are found to exist in greater numbers on the first day of life than on the fourth and succeeding days, the numbers being six to seven millions per cubic centimetre, as against four to five millions. Silbermann and Ponfick have discovered many forms or *shadows* of red blood-corpuscles from which the hæmoglobin had been robbed: they are often difficult to see, but there can be no doubt of these delicate ring-like shapes being the stroma of red blood-disks. Silbermann observed them to be more numerous the more deranged was the child's condition during the first few days of life. Hofmeier noted that the red blood-corpuscles varied greatly in size, were more spherical than in the blood of adults, and showed little tendency to run into rouleaux. The white blood-corpuscles are generally more numerous than in the blood of adults, more prone to run together in masses, more viscid, deliquescent, and less stable. These changes are of great pathological importance, and may explain much that is obscure in our knowledge of icterus neonatorum. There evidently is a great destruction of red blood-corpuscles leading to hæmoglobinæmia; and then the circulating blood, as Schmidt maintains, may hold a large quantity of fibrin-ferment resulting from the destruction of both kinds of blood-corpuscle. Silbermann injected hæmoglobin into the blood of frogs, dogs, and puppies, and then found that the state of their blood, urine, and liver had a close similarity to the conditions found in the first days of life. Silbermann therefore urges that the state of blood of the newly-born is such as to predispose them to disease. But it seems doubtful, according to others, whether the blood has any increased tendency to clot in the vessels as the consequence of the alleged increase in the fibrin-ferment. Relatively, the number of red blood-corpuscles is said to be greater in children even after the first days of life than in adults, but later still a falling off has been observed. According to Demme, there are on an average 135 to 210 colored to one colorless corpuscle during the period comprised between twelve hours and one hundred and fifty days of age,—as compared with the numbers in the adult, 330 to 350 red to one white. Slight fluctuations are met with according to whether the enumeration is made before or after meals. The nature of the nourishment also has an influence, breast-fed infants having relatively more red blood-disks than children brought up by hand. Although so much hæmoglobin exists at first only to be destroyed in the first days of life, still at th end of the first week a rise, though not to the original figure, is to be note The physiological loss of weight noted soon after birth (see page 52) probably intimately connected with, if it be not due to, the remarkab changes in the blood and probably other tissues; the variations in the tem perature of the body should perhaps also be considered in the same light.

THE PULSE.

The pulse of infants and children is very irritable, variable, and irregul in rhythm, the nervous centres on which it is dependent being very unst ble: perhaps the great frequency at birth and during the early months due to a want of development of the physiological inhibition which ma show itself in actual structural imperfection of the nerves. This suggestic appears to receive confirmation from experimental researches on some your animals; for some observers have found that stimulation of the vagus ner and centre has not that restraining influence which later in life it comes possess.

Any slight influence, even physiological, such as crying or suckin often so perturbs the pulse-rate that in infants the mere pulse loses muc of its pathological significance. During fever, if the child is asleep drowsy and not disturbed by external circumstances, the pulse-rate is four to be raised; and this is proportional to the rise in body-temperature.

The normal frequency of the pulse during the first weeks of life ma fluctuate between 150 and 120, being rather more frequent in females ar in smaller infants. It does not appear to vary with the posture of th sucking infant: later in infancy posture exercises its usual influence. the end of the first year of life the rate varies from 100 to 120. After th the pulse beats about 100, and tends to get less frequent, till at five yea the normal frequency may be reckoned at 90. The researches of Rameau Volkmann, and others have shown that a certain relation exists between th length of the body and the rate of the pulse, but in clinical work this inte esting physiological information is of no value.

RESPIRATION.

The narrowness of the nasal respiratory passages, the smallness o the nasal cavities, and the slight development of the Roman arch of th pharynx and of the cavities opening into the nose must be considered i discussing the physiology of infantile respiration. The lungs, originall small, grow very rapidly during the first months of life; nevertheless the remain during childhood, as Beneke showed, relatively to the weight an length of the body, less than in adults. The breathing of male and femal infants and children is mostly effected by the diaphragm and lower chest, in the adult male. The rhythm of breathing, like that of the circulatio is very irregular and variable, especially at birth and for the next fe weeks; and this has to be remembered in estimating the nature of an disease from which infants may suffer. After the first half-year the rhythm should be fairly regular, whilst during the first months an approach to the Cheyne-Stokes rhythm, if not normal, may at least be induced by very trivial pathological causes. Breathing is effected through the nasal passages, so that considerable swelling of their linings and of the naso-pharynx may cause much suffering and shortness of breath, since the habit of breathing through the mouth has not been acquired, and, indeed, seems somewhat difficult of acquirement.

The number of respirations per minute is greater than in the grown-up, and varies in the newly-born between 30 and 50, being fewer during sleep and then also more regular; in the first year of life it ranges between 25 and 35; but these numbers are increased by crying and laughing and diminished by fixing the infant's attention. Not uncommonly in young children during physical examination of the lungs long pauses between expiration and inspiration may be observed, and the breathing may be stopped for many seconds. The increased frequency of respiration has been ascribed to the smallness of their lungs and to their great need of oxygen, whilst the loudness of their breath-sounds (puerile respiration) may be explained by the force of their inspiration and expiration, the frequency of their breathing, and the narrowness of their air-passages. The epiglottis being folded on itself, like a leaf on its midrib, is an anatomical fact on which sufficient stress has not been laid; and this anatomical feature is the more marked the younger is the infant.[1]

Von Pettenkofer has estimated that a child produces in proportion to its body-weight nearly twice as much carbonic acid as an adult.

THE DIGESTIVE SYSTEM.

The relative dryness of the cavity of the mouth in the first months of life is attributed by Korowin, Zweifel, and others to the slightness of the salivary secretion, which, however, begins to increase towards the end of the second month of life. Although the salivary secretion at birth, slight as it is, does possess some power of transforming starch into sugar (probably maltose, not glucose), yet this power is only properly gained when the secretion becomes freer. The same is held to be the case with the pancreatic diastatic ferment, and the pancreatic power of digesting fat appears also to be almost in abeyance for the first months of life: nevertheless the observations of Kramstyk show that the alimentary tract possesses great facility for the absorption of fatty particles.

According to Korowin's experiments, the pancreatic juice does not attain its full powers for converting starch into sugar until the end of the first year of life, although a trace of diastatic ferment exists in the second month, and this is increased in the third month. Zweifel found that in strong children the pancreatic extract digested albumen in the first month.

---

[1] See the author's Treatment of Disease in Children, Lewis, London, 1887.

The stomach of the child is undeveloped in many ways, and its capacit is said to be not greater than 35 to 45 cubic centimetres at birth, b rapidly becomes greater, so that at the end of a fortnight Beneke's mea urements were 153 to 160 c.cm., and at the age of two years 740 c.c Langendorf and others seem to have demonstrated that the gastric jui of infants contains pepsin and hydrochloric acid and to possess the usu digestive properties on proteids. The alimentary tract of the infant is, rel tively to the length of the body, longer than that of the adult. Beneke numbers in the new-born are 570 : 100; in the second year, 660 : 100; i the seventh, 510 : 100; and in the thirtieth, 470 : 100; and Forster us these facts to account for the greater ease with which children appropria a milk diet. The muscular structure of the stomach and intestines is b feebly developed; Brunner's and Lieberkühn's glands are only developin during the first periods of life, whilst a great richness of lymphoid tiss in the solitary and agminate follicles perhaps accounts for the readiness wit which fat is absorbed, since it is held by some that the lymphoid corpuscl are the carriers of fat directly from the alimentary tract into the lacteal The physiological act of vomiting is easier in an infant than in the adul doubtless from the anatomical peculiarities of the stomach, its long ax being almost in a direct line with that of the gullet.

The liver of the new-born infant is relatively large and very rich i blood; it is larger than both lungs put together, and this proportion, Beneke showed, is not reversed till puberty. The increased size is probabl in harmony with the great nutritive and metabolic activities required f the processes of growth and development, especially of the neuro-muscul apparatus.

The bile of children, recently investigated by Jacubowitsch, is distin guished by its poverty in inorganic salts, with the exception, however, o iron salts, its poverty in cholesterin, lecithin, and fat, and particularly it smaller percentage of the special bile acids, and the glycocholic is in com paratively less amount than the taurocholic acid. It is supposed that thi smaller percentage of bile acids is a favorable fact, on the ground that th bile acids hinder the peptic and pancreatic digestion, the active ferments o which processes are supposed to possess feebler powers than in the adul On the other hand, the poorness in bile salts is considered to be a reason fo the alleged incomplete assimilation of fat by the infantile alimentary juice and agencies, since the bile acids emulsify the fat in the intestines, glyceri and fatty acids resulting. Some believe that the indigestibility of fatt human milk and of cow's milk may be ascribed to the above-mentione peculiarities of the bile. It must be obvious, however, that much more ha to be learned before physiology and clinical medicine can be brought int harmony with each other.

Escherich has carefully studied the digestion of milk in infants. Th fæces of infants fed purely on milk have a golden tint, a soft lard-like consistence, a feebly acid reaction, and contain from eighty-four to eighty-si per cent. of water. The digestion and absorption of proteids in the alimentary canal are so efficient that but little passes on in the fæces. In fact, the whitish flakes and clots nearly always seen in the fæces are composed almost entirely of fat, fatty and lactic acids in combination with lime, whilst cholesterin and traces of bilirubin, intestinal epithelia, and mucus may also be detected. In addition, large quantities of bacteria are always present,—a fine slender bacillus, named by Escherich the bacterium lactis aërogenes, and the polymorphic bacterium coli commune, which often takes the form of cocci, being the two chief kinds. Of other varieties of bacteria, as of the proteolytic cocci which fluidify gelatin, torulæ, and mycelial fungi, there are almost none in the normal milk-fæces. But these varieties may be discovered in pathological conditions, as also when the diet is a mixed one or contains much meat. The simplicity of the micro-organisms found in the milk-fæces is doubtless in correspondence with the absence of the products of albuminous decomposition, such as tyrosin, indol, phenol, and skatol; in the milk-fæces, however, milk acids are always present, and to their presence should be attributed the acid reaction. Fermentation of milk-sugar leads to the development of carbonic acid and hydrogen, which are the principal gases in the intestinal tract of healthy infants fed purely on milk, foul-smelling gases being conspicuous by their absence.

Though the quantity of fæces varies much in sucklings, yet three grammes for every hundred grammes of milk ingested may be given as the average proportion.

Certain peculiarities exist in the stools of the new-born, the so-called meconium being odorless, tenacious, and viscous, of greenish color and weak acid reaction. It contains constituents—epidermic cells and hairs—derived by swallowing from the liquor amnii, also cholesterin and intestinal epithelium. Normal meconium is free from products of putrefaction, phenol and acids of the benzoic series being absent. Moreover, immediately after birth it does not contain bacteria, but in a short time numerous and various micro-organisms make their appearance. Probably some entered by the anus; and some may have gained entrance by the mouth and respiratory passages. An investigation on the new-born might throw some light on the vexed question whether healthy blood and tissues do contain micro-organisms. According to Escherich, even these bacteria disappear, or are replaced by the two varieties above mentioned, when a pure milk diet is commenced.

DENTITION.

The eruption of the teeth is undoubtedly influenced by the constitution and the nutrition of the infant, for in the well-nourished children of healthy parents the teeth are cut earlier and more regularly than in others. The first lower middle incisors may be cut between the third and the tenth month, the average time being the seventh; the corresponding uppers may be cut a month or so later; the lateral incisors may appear about the same time, or a month later; then the first molars should be cut at about twelve months or so, the canines about the eighteenth, and the four remaining molars at abo two years. Exceedingly variable in their date of appearance, much allo ance must be made for individual and racial peculiarities. This differen may be noted, however, between the eruption of rachitic and healthy, bo that the former, besides being delayed in their coming, also come irregular and out of the ordinary series, whilst in the healthy the number of teeth c is usually even, two of a like sort being cut nearly simultaneously, and t proper order of the series—viz., middle, lateral incisors, premolars, canin second molars—is maintained. As a general rule, the lower teeth are c before the upper. The permanent teeth begin to appear about the seven year, the first to come being the first molars; then follow the central inciso at eight, the lateral incisors at nine, the first and second bicuspids at ten a eleven respectively, the canines at twelve, the second molars at fourteen, a the wisdom teeth in adult life; for twenty milk there are substituted thirt two permanent teeth.

THE URINE.

Relatively large at birth, the kidneys do not increase so much in si during childhood as do the heart and the lungs,—the adult lungs bei from twenty to twenty-five times as large as at birth, whilst the kidne only grow till they become twelve times heavier than at birth. The fun tional activity of the kidneys reaches its height even at birth, and in t pyramidal portions of the kidney of the new-born it is quite common to s reddish or yellowish deposits, or even brownish streaks, which microscopic examination shows to be uric acid crystals blocking the straight urina tubules: so common are these uric-acid infarcts (Virchow) that they mu be considered to be of no pathological significance.

The quantity of urine increases rapidly during the first ten days life, but during the next week more slowly. Cruse has estimated the dai quantity to be about 130 to 417 cubic centimetres, but there is no dou that it varies within very wide limits: its rapid increase must be attribut chiefly to increased ingestion. During the second year of life the dai quantity discharged is estimated at 500 to 600 cubic centimetres, and twi as much is said to be passed in one day during the fourth year.

The specific gravity of the urine increases rapidly from the fifth to t tenth day of life, then diminishes; but, according to Cruse, the phosphor acid increases. The average density is given as 1005 to 1010.

During the first days of life the urine is generally turbid, dark, an acid, but later becomes clear, straw-yellow, and generally neutral. T kind and quantity of food influence its characters and composition ver materially. The first act of micturition may often be delayed for twent four hours or more, and this is probably the reason for the urine bein dark, turbid, and acid, it having undergone concentration in the bladd owing to absorption of water. If the urine is passed immediately aft birth, it is clear, nearly neutral, and very pale. These facts prove th the kidneys have been at work during intra-uterine life, and they also see to show that the nervous functions of micturition chiefly dependent on the lumbar enlargement of the spinal cord are in a fair state of perfection.

According to some, the discharge of nitrogen in the urine of infants is relatively smaller than in the adult; and the same has been stated of the discharge of phosphoric acid and chloride of sodium; and to account for this it is supposed that more of the ingested nitrogenous stuff is retained for building-purposes. Others, probably with more truth, assert that, the metabolism of foods and tissues being in greater excess, the urea-discharge is also in excess in children as compared with adults.

During the first days of life the normal urine is maintained by some to contain a trace of albumen, which, however, soon disappears, and according to others albuminuria is not normal even at any time.

THE TEMPERATURE.

Always a subject of much interest, the temperature of the body before, during, and after birth has been much investigated. A recent monograph by Raudnitz gives a good *résumé* of our information on this subject. Immediately after birth the temperature falls as much as 1.7° C., the average minimum temperature being, according to Eröss, 35.84°, whilst the average normal temperature of the new-born is given as 37.6° C. After this preliminary fall a slow rise to the normal occurs, and a temperature of 37.8° to 38° C. on the fourth or fifth day is, according to Eröss, a sign of abnormality.

The temperature of young children undergoes many fluctuations at the dictation of slight influences which should be ascribed to the instability of the heat-regulating centres, just as we witness the same irregularity, irritability, and variability in the respiratory and circulatory centres. Demme has noted a fall in the temperature of children experimentally kept in dark rooms. Increased ingestion of food, and much crying or struggling, raise the temperature, whilst inactivity and sleep lower it. A high temperature, like a high pulse or a high rate of breathing, has more significance if observed during the sleeping state of an infant.

Many observers attest the influence of the law of temperature being manifested even in infants, for they have observed the normal daily variation. As a rule, the temperature begins to rise in the morning, reaches its highest point in the evening about six, and then slowly sinks to its lowest point in the first hours of the morning. It is noteworthy that new-born infants may under the excitement of inflammatory affection yield a temperature as high as 41° C.=105.5° F. In fever the differences between the morning and the evening temperature are greater than in adults, and the influence of antipyretics on the fever-heat is more considerable in them than in the grown-up.

THE SKIN.

Coated with a layer of sebum, shed epithelium, and hairs, called the vernix caseosa, the skin of the healthy new-born infant after being cleansed is found to be much redder and more tender than in later life, and is covered with fine down called lanugo. In the first few weeks of life a fair amount of desquamation occurs, and the fine hairs also fall out. During this period the sweat-glands do not make much perspiration, but the sebaceous glands are much more active, especially on the scalp, where flakes of fatty matter may accumulate and be shed with hairs stuck thereto.

SUMMARY.

It will be seen that the subject of the physiology of infancy is a comparatively unworked field, but a most interesting one nevertheless.

As anatomy and histology form the basis from which are to be studied developmental and pathological differences, so physiology is the standard by which the clinician judges of the value of the symptoms resulting from functional disturbances.

DIAGNOSIS.

By JAMES FINLAYSON, M.D.

A SPECIAL chapter on the Diagnosis of the Diseases of Children might be regarded as very important if the subject could possibly be dealt with in this way; but diagnosis is coextensive with the whole range of this work, and in point of fact it will consume a very large proportion of its various sections and different volumes. Why, then, it may be asked, have such a chapter at all? Are not the diseases of children, apart from malformations and perhaps one or two comparatively rare affections, essentially the same diseases that affect adults? Are not the facts of disease the same whatever the age? Are not the principles and methods of physical diagnosis practically identical for all periods of life? The diseases are essentially the same; the methods are in no sense very different; and the reader of this chapter will be supposed to bring to his aid a moderately extensive knowledge of disease as it exists in adults, and likewise a certain familiarity with the methods of diagnosis and with the signs of disease as observed there. What more, then, is wanted? Why have such a chapter? Nay, more, why have special treatises on the diseases of childhood at all?

A student confronted with a sick child may be moderately well acquainted with diagnosis as practised in our general hospitals, but may feel as if all his knowledge and all his methods had suddenly failed him: he may experience the same sense of helplessness which a traveller will have when suddenly cast adrift in a strange land, of whose customs he is ignorant and whose language he has not yet learned;[1] in proportion as he is intelligent, and practised in travelling at home, will he experience the vexation of seeing the same kind of things—the means of locomotion, the places of rest, the various forms of food—and yet be unable to understand how to avail himself of them all; or, perhaps, misguided by some spurious resemblance, or misled by some opposite custom, he may find himself injured by the very knowledge which would otherwise be useful.

We usually begin by asking our adult patient how he feels, or where he has pain, if any be present; but our little patients may be too young to speak, or, if they do speak, the pains and discomforts may be referr to in a misleading manner: thus, it is common for a child with a pa originating in the chest to refer it to the stomach or belly, and this n merely in words, but actually by direct signs. All the information v are in the habit of getting from the patient's description of his discomfo may thus utterly fail us; the distress may be as great, or even greater, b the "infant crying in the night," however definite, however obscure, hov ever complex, or however varied the nature of his misery may be, has " language but a cry."

---

[1] See Dr. West's Lectures on the Diseases of Infancy and Childhood, 7th edition, London, 1884, p. 3.

Baffled in this direction, the student bethinks himself of the well-know physical signs of disease, for here at least we are independent of articula signs, and "there is no speech nor language where their voice is not heard He tries the pulse, but his approach and the excitement of crying have se it up to a preternatural height, with numbers uncountable or at least wi no meaning. He tries the temperature, but the child resents having a the mometer put into the axilla; or in other cases the arm is so thin and scragg and the covering of soft parts so imperfect and so difficult to keep in app sition that, even if feverish, the readings may come out as normal or sul normal, and so mislead instead of helping us. He now contents himsel with feeling the skin with his hand, well knowing how fallacious this even in adults, and proceeds, after allowing the child to settle a little, to tl examination of the chest, beginning, as is common, with percussion und the clavicles; of all things, this is one which a young child objects to, c account of the pain so apt to be caused, and on account of the source c new terror in each successive stroke being so visible; the infant agai begins to cry or scream, drowning the relative percussion-sounds sougl for, or allowing only of some "chinking" or "cracked metal" percussior note being caught amidst the din, and so giving rise to all sorts of erroneou visions of cavities in the apex. Auscultation fares no better; the child ol jects, in its own language, to a hard stethoscope pressed with a heavy hea on its tender ribs, and sees new dangers in the close proximity of the ol server's head. But even if more successful, the student is accustomed t rely on the patient's taking long breaths, when asked to do so, in order t educe the crepitant râles or other abnormal sounds; or to hold the breatl to get clear of this complication in auscultating the heart; but it is ofter if not usually, utterly vain to expect any such assistance from a child o tender years, while the hope of assistance from vocal resonance and fremitu has to be equally abandoned.

At any rate, the student hopes he may find evidence of chest-disease i the cough or expectoration; but he learns with astonishment, although th cough may be very severe, that there is no expectoration at all; and by an by he finds that the study of expectoration in the lung-diseases of childhoo is not of the same importance as in adults, for the good reason that there i usually none brought up; on consulting his books he finds that even in pronounced pneumonia rusty sputa are almost unknown, and that the chil may cough but little, or may only begin to have any noticeable cough when the worst of the disease seems over.

Defeated in the region of the chest, the abdomen is not likely to give more chance of success: the muscles are on the strain from pain, or from fright, or from crying; and of course palpation and percussion require the most favorable conditions of the abdominal walls to afford any information. Or he may find what he thinks is an enlarged liver or spleen from mere displacement of these organs in the lax abdomen of the child, and the enlarged and displaced spleen of rickets may never have been heard of, although he may have visions of enlarged spleen from typhoid fever, ague, or leucocythæmia.

The examination of the urine, at least, might be supposed to be a matter familiar enough to a well-trained student, affording some information or at least negative data. But very likely the urine cannot be saved, being passed, of course, by young children without warning even when well, and very often passed in this way during an illness by those who are old enough to give notice at other times. But the mother or nurse may have information to give about its appearance, or a stray and scanty specimen may be shown confirming their description of its being "as white as milk" when passed,—a characteristic usually pointing in an adult to the admixture of pus, and so constituting a grave symptom of urinary disorder, but, as a rule, dependent in the child on the presence of white urates or some other trifling peculiarity.

If the beginner now turns to the history of the illness, he may be equally perplexed. The rigors which form so leading a feature in the initial stages of acute inflammations and fevers in the adult are almost invariably absent in the young. The symptoms often come on either with such suddenness as to leave no chance of tracing their progress, or so insidiously and indefinitely that often even a careful mother can only tell us that the child "is not well," or that he is cross and fretful, or pining, or wasting, without definite indications of the reason why such conditions exist.

The family history is frequently rendered vague and indefinite on account of the youthful age of the patient, for the evidence of the inheritance of disease on which we rely most confidently is furnished by the life- or health-history of the brothers and sisters of our adult patients; but these brothers and sisters of our infantile patients are not, very likely, as yet born, or at least may not have had time to show their morbid tendencies; the parents also, even if they actually die young, may not, at the time of the inquiry, have developed the evidences of their fatal diseases.

DIAGNOSIS MADE EASY—TEETHING.

Such are some of the difficulties which beset the study of children's ailments, even by those who are, so far, familiar with the diagnosis of disease. The difficulties are no doubt great,—often, indeed, insuperable,—and we have frequently to be content with vague results. But at this point an insidiou temptation is presented to the beginner, decked out, it may be, in the allurin apparel of speculative science falsely so called. The illnesses of young children are cleverly described as being largely connected with, and essentiall due to, the process of dentition. The gradual evolution of the first teet constitutes a striking and most important feature of early life. The proces goes on in all young children, and so is available as a universal explanation a few exceptional cases usually present sufficient evidences of disease t show that the *absence* of dentition is also potent as a cause of bad healt With the growth of the theory of reflex action and irritation, in the earl part of this century, the newest views of the nervous system were used t clothe afresh this old and specious doctrine, so that what might have die out as an old-world superstition was made to appear as the most advance scientific doctrine and the most modern practical application of a recen brilliant discovery. This convenient theory of the dependence of infantil disease on the process of dentition is, of course, now exploded, and i might be supposed that it needed no notice in a work like this. Bu superstitions are difficult to kill; although exploded in one country, or i one time, they linger on to haunt the members of each new generation and temptations rejected as unworthy by the common good sense of community remain to allure each set of new individuals as they grow up So it is with teething. Every young practitioner, often baffled by the intricacies and difficulties of infantile disorders, is led, as it were, to some hig mountain and made to survey the wide realm of infantile disease, with al the manifold forms and degrees of suffering or illness to which the youn are subject, and he is made to feel that all this dominion can at once b made over to him if he will but fall down and worship this fetich calle "Teething." It matters nothing that at the moment no teeth may b coming through; they *are* coming; the "breeding" of the teeth is eve more serious (because deeper and more unseen) than the mere piercin of the gum. It matters not that the symptoms persist after the gum i pierced; others have yet to come. It matters not that the coming toot (really retarded by the illness) shows no signs of coming through to confirm the diagnosis, for it is ingeniously contended that it is this very delay in reaching the surface which accounts for the prolonged and serious illness. Even after the whole of the milk-teeth have appeared, we can easily speculate that the "breeding" of their larger successors must give rise to even more serious perils than those which the child has just come through.

The diversities of age and the diversities in the period of development of the teeth are thus easily bridged over, but the diversities in the manifestations of disorder are no less ingeniously met. The varied forms of nervous disturbance—pain, restlessness, convulsions, tremors, twitchings, and spasms—are conveniently ascribed to the irritation of the fifth nerve: is not this an afferent and sensory nerve? are there not reflex actions? What can be plainer! Young children are affected with a form of paralysis so peculiar to them that it has been termed "infantile paralysis;" but the process of teething is also peculiar to them: has not "reflex paralysis" been described? Why, then, refuse to assent to the name of "dental paralysis"? Teething children have their mouths dribbling with saliva. If their bowels are confined and the motions hard and dry, who can doubt that dentition, by draining away the fluids to the mouth, gives rise to this disagreeable symptom? Of course, equally, if the bowels are loose, this same saliva will naturally explain the diarrhœa of infants from its being swallowed. Violent choleraic attacks, convulsive seizures, or any terrible disaster may be easily traced to a poisoned state of the saliva: who has not heard of the poisonous saliva of a mad dog? But the irritation of teething may equally disturb the bronchial mucous membrane: is it not continuous with that of the mouth? Bronchitis and catarrh can thus be ascribed to teething without supposing any exposure to chills or any error in clothing. If the mucous membranes suffer, why not the skin? A "tooth-rash" is a splendid safety-valve; and when it resists our best efforts at treatment, we can explain how dangerous it is to cure a rash in a teething child, in case of its driving in the disease to some internal organ! The affections of the eyes and ears are too obvious to require explanation: do we not speak of the "eye-teeth"? and who has not felt pain in his ears from a bad molar?

The popularity of this doctrine depends partly on its saving a world of trouble to the doctor, but also on its meeting the views of the parents. We all like to have things made clear; and if "the doctor explained" the interesting connections referred to above, we can easily understand how much cleverer both doctor and parents would appear in the eyes of the latter.

But there is a subtler explanation of this popularity of teething with parents and nurses. The human mind resents the idea of our transmitting anything but good qualities to our children, or, indeed, of anything bad having been transmitted in *our* especial families. A mother's death from consumption and a sister's illness from the same cause can be explained away to the physician so as to lead to the well-known summary of the whole matter, that there is "nothing like hereditary consumption in the family." How much more natural to try to explain away hereditary scrofulous disease, for example, transmitted to our own children, and to account for swollen glands in the neck, or the results of a similar mass in the abdomen, by the natural effects of the process of teething!

But, if we desire to minimize the bad effects of an inherited taint thus transmitted to a new generation, it is even more likely that parents and nurses should try to minimize the evil results of want of care, or of errors in diet, clothing, and hygiene, as regards the young children under their charge. A diarrhœa due to a wrong style of feeding is a slur on their character and discretion; a diarrhœa from teething is what might be called (as in the marine insurance policies) "the act of God." A wasting due to prolonged starvation of the child (it may be in the midst of plenty) is a serious matter to be faced by the attendants; a pining away from a pro-

longed and troublesome dentition is sad, but not blameworthy. A conv
sion due to the cutting of a tooth is alarming and frightsome, but not wi
out its pleasurable excitement when combated promptly and successfully
the domestic remedy of the warm bath and the timely scarification of
gums by the doctor; but to ascribe the convulsion less to the teething th
to the state we call "rickets," raises the awkward subject of what rick
depends on; and questions of proper feeding, of good air, and of who
some bedding, when raised by the doctor, disturb the *entente cordiale* foste
by the other view.

If the practitioner wishes to avoid trouble in the diagnosis and trou
with the attendants, the comfortable diagnosis of teething is most attr
tive. Indeed, if it were only a matter of speculative or pathological
terest, perhaps it might be legitimate to allow the mothers thus to soo
their minds, amidst the distresses of their children, without raising a
unpleasant doubts. But here, as everywhere in medicine, diagnosis lies
the root of intelligent practice. The process of teething is inevitable a
universal, and is but little under medical control of any kind; on the ot
hand, the processes leading up to rickets, for example, are largely un
control, even in the case of those who are comparatively poor. The dia
nosis of teething diverts the mind of every one concerned from the vi
points of food, air, and hygiene. Many a teething child has been allow
to go on indefinitely, to a hopeless extent, with a diarrhœa which, far fr
being attended to, was regarded with complacency as a beneficial outlet
the dangers of dentition, the stopping of it being looked upon as little l
than deadly; for the constipation so often associated with tubercular meni
gitis, and the convulsions attending it, were regarded as more dangero
complications of teething,—diarrhœa on the one hand, and constipation a
convulsions on the other, being both erroneously ascribed to this proce
instead of putting the diarrhœa down to bad and indiscriminate feedin
and the meningitis, with its attendant constipation and convulsions, to
inherited tubercular constitution.

It is this practical consideration—the intelligent and prompt treatme
of sick children—which makes one protest against the doctrine of dentiti
as a cause of disease. It may still be a moot point how far a child is ma
ill by teething; but if the beginner is ever to make any progress in t
diagnosis and treatment of the diseases of infancy he must take up t
attitude of refusing to believe that any child is ever *seriously ill* fro
teething: a restless night, a little disturbance of the stomach and bowel
may occur, but whenever a real illness appears, whenever a prolonged di
turbance of the digestive or other system has declared itself, then we m
be sure there is something else at work, some fault to be corrected, or son
more grave disorder impending. If the beginner resolutely determines
find out what this fault or this disorder really is, he will, with increasin
experience, fall back on teething less and less, even for trifling ailment
and so by his counsels may prevent the development of more serious mi

chief. If for "teething" we read "stomach and feeding," and if we always consider whether these are at fault, we might, although proving disagreeable and troublesome at times to the mothers and nurses, do more good service to the suffering infants.[1]

METHOD OF EXAMINING SICK CHILDREN.

The method of examining any sick person must be determined by the actual condition at the time, whatever plan may be in the mind of the physician, or whatever may be the views of doctrinaires. In the case of a sick child this is pre-eminently true. Urgent symptoms, like fits of any kind, or obvious features, like the appearance of an eruption, demand, of course, direct attention without much preliminary inquiry. In ordinary cases it is well, as a rule, to hear from those in immediate charge of the child a full and connected account of the illness and its supposed cause, taking special note of the exact dates on which the various events occurred, as this precision as to time often leads the narrator to correct or modify or expand the original statement. Sometimes this preliminary narrative is best obtained in the sick-room; the physician can then sit down without attracting the child's attention to the visit's having any direct reference to him; or the child may, at times, go to sleep during the narrative, and so afford a chance for seeing the effect of this state. More often it is best to get all this account in another room, out of the hearing of the child, unless very young; but in any case it is important that the examination should not be begun until after the physician has a pretty clear view of what points may come up for his investigation.

It is usually desirable to ascertain by definite and categorical questions whether the illness, as now existing, appeared to come on in the midst of perfect health; and, if not, to ascertain with precision up to what time the child might be regarded as perfectly healthy. Unless this is put to the mother as a definite question, much confusion is liable to creep into the narrative. In very young children it is usually best to hear the whole medical history of the infant, with dates of weaning, teething, walking, etc., connecting thus the past history of the child with his present illness. Any previous illnesses of the child should also be fully considered, as they often have a direct bearing on the case, even when the previous illnesses may seem of an accidental character, like measles or whooping-cough; and this is all the more important when the illness investigated is chronic, and perhaps of an obscure and indefinite character. The obtaining of a connected account of the child's illness is a matter of no small difficulty, confused as it is apt to be by different persons being in charge, and by the minds of the attendants and their ideas of time being rather muddled from natural anxiety

---

[1] This subject is more fully discussed by the writer, from the historical point of view, in a series of papers "On the Dangers of Dentition," in the *Obstetrical Journal of Great Britain and Ireland*, October, 1873, to February, 1874; or, in a shorter form, in the *British Medical Journal*, September 19, 1874.

and want of rest. The greatest patience and forbearance must be show
women worn out in thus watching the young. It is usually much the
way to let them tell their story in their own way, as this satisfies their mi
and supplementary information can be gained by questioning : if the
dency is to prolixity and irrelevancy, the narrator can be guided and k
to the point by sticking to the dates, day after day being taken up,
the rambling thus materially lessened.

The beginner will do well to listen with respectful attention to
accounts of an illness given by the mother or by a faithful nurse. T
familiarity with the ways of the child may make them feel that there
something wrong, although their powers of observation and description
not enable them to carry conviction to the minds of others; their const
handling of the child enables them to detect a diminution in the firmn
of the child's flesh, or a failure in his strength, which may readily esc
the attention of any one else. When a woman of sense in attendance o
child alleges that he is ill, or that he is worse, the chances are that she
right, even although the proofs she may adduce may seem trivial or er
neous. It is in their interpretations and theoretical ideas that mothers
so apt to be wrong, and their wild speculations on the "liver," the "hive
and the "nerves" tend to bring their opinions generally into unmerited c
tempt. Of course some women have the power of arguing clearly enou
up to the level of their knowledge; and the writer can recall a case
intussusception in an infant, where the mother urged that there must
"some obstruction between the stomach and the outlet of the bowe
although the doctor in attendance had evidently never thought of the c
in this light.

Men much versed in the treatment of diseases of children are alwa
chary of setting aside the opinions of the mothers and nurses when th
differ from their own as to the relative state of the infant's actual conditi
unless these opinions are clearly found to be based on some erroneous int
pretation of the symptoms present. While estimating lightly all their the
retical views, we should weigh seriously all their statements and opinions
to the actual facts of the illness, and especially as to the general condition.

For the personal examination of the child, there should be the greate
flexibility of plan, and a ready promptitude in taking advantage of eve
chance which may arise, and in deciding at once which points are of t
greatest immediate importance in the case. Thus, if the child be asle
advantage may be taken of this to get the pulse and respirations counte
the general character of the breathing observed, and the color of the fa
noticed; even auscultation may to some extent be possible. On the oth
hand, if the case seems to be one of abdominal disease, this same state ma
afford a golden opportunity for examining the belly, slipping the warm han
under the clothes of the sleeping child, and ascertaining the condition o
the walls and of the internal organs, before crying, or fright, or pain, ma
render the parts so tense as to baffle the observer. At times, by sittin

down and taking the temperature in the axilla, holding the arm to the side, or getting the nurse or mother to do so, we may allow time for the agitation and fright at the sight of a stranger to subside, and the child may even go to sleep in the process, allowing some part of the examination to be made in this state. Or the delay in taking the temperature may sometimes be utilized for hearing the history, if this has not been fully gone into, or for filling in details or clearing up confusion in the narrative.[1]

In proceeding with the examination, the guiding principle is to avoid sudden or abrupt methods; preliminary manœuvres, and even a little playfulness, often help to establish friendly relationships which facilitate the work; but no definite rules can be given. The patience and good temper of the physician must be inexhaustible, and when these are combined with a genuine desire to benefit the child, and a *bona fide* love of little children, there are no limits to what may be done. Here, as in many other things,—

"It is the heart, and not the brain,
That to the highest doth attain."

It is idle to deny, however, that at times the greatest patience and tact seem alike thrown away, and the examination must remain very incomplete; or perhaps special parts of it, if of extreme importance, may have to be carried through by main force. Usually this depends less on the nature of the illness than on the habitually bad moral training of the child on the part of the parents; or it may depend on the medical examination or treatment in this or in some previous illness having been of a harsh or at least disagreeable character. One part of the examination has often to be conducted, so far, by main force, viz., the examination of the throat, and for this reason it is usually kept to the last. Some young children occasionally give us every facility, and, by getting them to open their mouths widely and to draw a deep breath, we may see the fauces well enough; or we may require to aid the view by a gentle depression of the tongue with the tip of the finger or the end of a spoon. When such methods are not available, or fail to suffice, the best way is make every preparation for securing proper light from windows, lamps, candles, or tapers, and to have adequate assistance for holding the child firmly during the examination, and for controlling the arms, which are often best kept out of the way by a blanket or sheet held tightly round the front of the chest so as to include them. When all is ready, the mouth may have to be opened by main force, and even the nostrils held in separating the lips and teeth, and then, with the handle of a teaspoon, a bone spoon, a spatula, or a tongue-depressor, we hold down the tongue and turn the head so as to see both sides of the fauces in a g light: we are often aided in this view by the gasping breathing of the ch or even by the efforts at vomiting. From want of proper arrangem before beginning, the hands of the child may tear away the spatula or candle, and all the annoyance has to be gone over again, under worse c ditions than at first.

---

[1] Of late years some mothers take the temperature of their sick children before sending for medical advice, and can tell with accuracy the degree of fever present: indeed, they are sometimes guided in sending for the medical attendant by these observations. In such cases there is none of the delay referred to, and the temperature found comes in as part of the history narrated.

Young children (under five or six years) are usually examined best their mother's knee; if in bed, they can be lifted out with one of th blankets, this change often helping to pacify them if fretful. Soft sha or thin blankets, previously warmed, are very useful in covering up the ch while successive portions of the body are being exposed for examinati thus, the shawl may be tucked round the loins while the back of the ch is being examined; or over the shoulders, or over the abdomen, as the may be. Exposure of a small part of the surface of the body for a sh time, with the adjoining parts covered over with warm shawls, has seld any injurious influence: a large surface uncovered is, however, a very dif ent matter, and with the lower part of the back uncovered we are apt have the cold air extending also round to the abdomen and even to lower part of the chest in front,—a most undesirable occurrence, parti larly if the examination happens to be a little protracted and the skin from sweating or from the application of poultices; when thus cove with moisture the skin should first be dried with warm towels.

With some tact on the part of the nurse, the back of the child, w thus seated on her knee, may often be pretty well examined before the ch realizes that anything except rearranging the clothes is being attemp For the observer keeps literally as well as figuratively in the backgrou and some one may perhaps divert the child's attention in front by show some bright object, as a lighted taper, trying, of course, to avoid as much possible any distracting sounds in carrying out this diversion. In very yo children, and even in some others, the back of the chest can often be b examined by laying the child on his abdomen on the nurse's knees and th uncovering the back; the child is often pacified, for a time, by this cha of position, to which, of course, he is accustomed during the process dressing and renewing the napkins. A similar benefit is often obtained getting the nurse to hold the young child close to her breast, with the f of the infant towards either shoulder, as if looking over it, and when child's vision is thus directed away from the physician the back may be part uncovered for the purpose of examination.

The examination of the chest is usually best begun in one of these wa for the *back* of the chest is usually the most important, as the signs of br chitis and pneumonia are often most marked there; moreover, this part the examination is sometimes all that the child will permit. Auscultat is first practised, because it is less disagreeable to the child and requi greater quietness, and on the whole it is more important than percussi It is best done, as a rule, by listening directly with the ear to the chest-w as this is less irritating than applying a stethoscope: moreover, the head of the observer, closely applied, follows more readily any wriggling movements of the child. Circumstances will determine whether the skin should be completely bared or whether some thin garment may be left; of course nothing thick should intervene. A thin towel or napkin or handkerchief, previously heated, may be interposed between the ear of the physician and the skin, for the head of the observer is often much colder than the child's skin, and so is apt to irritate; in any case we prefer to have only one ply between the ear and the skin; at times we can listen with advantage with the ear on the naked chest. After auscultation of the back of the chest is completed, and any observations made on this part by eye or hand, it is usually well to practise percussion. Of course we generally prefer the finger as a pleximeter to judge of the resistance as well as the sound. The strokes are made lightly and rapidly, and it is often well to make a mental estimate of the average or mean sound educed by the *series of strokes* obtained in varying states of inspiration and expiration, as the breathing is often so rapid that no other basis of comparison can be obtained. Having in this way made observations on the percussion-sounds, on the two sides at various levels, from top to bottom, we can then lay the child down or turn him over on his back, having finished our examination of this region. When the child is laid flat, the front of the chest may be examined; this is generally the best position for young children; older ones may often sit up with advantage, having the back supported by the mother's arms. Auscultation of the front of the chest is usually best accomplished by means of a stethoscope, although the direct method also can often be practised with advantage in this situation, and sometimes we are glad to try both methods if we fail at first. In using the stethoscope it is well to put it in position first, or even to allow the child to play for a little with the "trumpet," so that he may feel that it is nothing terrible, before bringing down one's head with what is apt to be, even with care, somewhat uncomfortable pressure. By short successive applications and giving plenty of time, the use of the stethoscope may often be practised successfully; whereas if stethoscope and head are abruptly applied simultaneously, even apart from the tender chest being hurt by the hard instrument, the child is apt to be greatly terrified. While auscultating we lie in wait, as it were, for the occasional deep or even sighing respirations, so common in childhood, to reveal the râles developed only under such circumstances. Great patience and yet promptitude are required, as the child often holds the breath almost entirely, and then has a series of quick shallow respirations; but usually we can also catch an occasional deep breath. When the child is old enough, and an estimation of the resonance and fremitus of the voice seems important, it is often possible to get the child to answer some question put by the mother during the examination.

The examination of the abdomen may often with advantage be taken before proceeding to the front of the chest: the relative importance of the two parts of the examination as judged from the history of the illness must guide us. If the child is lying quietly, we may be able to palpate the abdomen and determine the position of the organs, or the presence of glandular or other swellings, before attempting the examination of the chest; for this, of course, however carefully performed, may lead to a fit of crying. Particularly is this apt to occur with percussion in front, even when gently done, so that we often leave this to the very last when any disturbance from crying will be less vexatious, even if it occurs. The percussion of the front of the chest is almost always best done with the finger as a pleximeter. Care must be taken to put the finger in exactly similar relative positions on the two sides; and, as before, we often aim at getting an average sound out of successive strokes. The lateral regions are often of great importance in cases of pulmonary collapse. The percussion under the left clavicle seems often a little duller than the right, in young infants, apart from any disease, probably owing to the proximity of the heart impairing the sound. A slight relative dulness at the *right* apex counts, therefore, for more than a similar dulness at the left in very young children.

The exploration of the abdomen is often most important, but not seldom the difficulties are extreme. For this reason it has been suggested above that, where this part of the case seems of primary importance, no chance should be missed of examining it during a quiet period, perhaps while the child is asleep, perhaps before risking an upset from the examination of the chest, perhaps in the midst of this part of the process. On the other hand, we may with equal propriety postpone the examination of the abdomen to the last, if the child is cross, in hope of a better chance arising. Too often in young children we are confronted with the difficulty of extreme tension of the abdominal walls, with resistance and screaming and kicking, making palpation or percussion equally useless. Sometimes by keeping the hand lightly applied under the clothes till the child is settled, we may be able, without arousing his fears, to feel the state of matters as regards laxity, tenderness, or tumor.

In palpation, we must see that the hand is warm, and that it is applied, in the first instance, gently and lightly, carefully avoiding any sudden jerks with the fingers, but feeling with the whole hand so as to avoid exciting the muscles to resistance. For similar reasons we, of course, begin with light palpation, before risking the irritation of deep palpation or of percussion, and the results of palpation frequently guide the exploration by percussion. The presence or absence of tension of the walls is important; we often find them tense in inflammatory affections of the bowels and of the peritoneum, even apart from effusions; and if we can press down a lax abdominal wall without eliciting signs of pain, we may almost presume that there is no peritonitis. The mere inability to do this counts for little, unless circumstances favor the examination, as the least fright may render the abdominal muscles extremely tense, and pressure then readily causes pain and further resistance.

The next point is to determine the position of the organs and the presence of any solid tumor. The liver can usually be felt, but it is often erroneously supposed to be enlarged when but little changed in size. It must be remembered that the liver is relatively large in young children, and that it is also relatively prominent in them below the ribs. Moreover, in rickets and other distortions of the chest the liver is displaced so as to simulate a great enlargement. Indeed, the whole belly is often very prominent and distended in rickets, and the inexperienced finding an apparent enlargement of the liver may erroneously suspect the presence of dropsy; even the percussion-note, perhaps from great tension of the gas in the bowel, or from other causes, may seem to be duller than a tympanites could give; in such cases also the air-filled bowels often give a spurious kind of fluctuation, which may confirm the deception, and so we occasionally see futile attempts at removing fluid from the abdomen by tapping in cases where none could possibly be obtained, because there is none present. The spleen may also be found very readily at times in rickety children, partly from enlargement, partly from displacement. By inserting one hand behind the left false ribs, and pressing with the other in front, we can often feel the spleen; when felt very distinctly we are seldom wrong in presuming that it is more or less enlarged. In marked enlargement its notch can be easily felt, and sometimes it extends away down to the pubic bones, and even turns there towards the right of the middle line. The spleen is often enlarged in rickets, usually only to a moderate extent; it may be found enlarged in scrofulous children, with, it may be, albuminuria or other signs of amyloid disease: occasionally the enlargement is associated with leukæmia: ague must also be inquired for: concurrent disease of the liver may suggest the cause of splenic enlargement: embolism has also been known by the writer to produce very palpable increase in the size of a boy's spleen. Occasionally, however, no satisfactory explanation can be found of the tumor, which may disappear as unaccountably as it grew.

For enlargement of the kidney and for other abdominal tumors the reader is referred to special sections of this book.

There is often great importance to be attached to finding little tumors or lumps in the abdomen in cases of suspected tubercular peritonitis and tabes mesenterica. In searching for these, the full breadth of the hand should be applied at first to the wall, and if the child is quiet this is worked about in various ways and pressed down, at first gently, and then, as the child gets accustomed to the manipulation, more deeply, in search of the hard masses: at this stage the fingers have to be made to dip down, and we often feel more security in the diagnosis when the lump can be caught between the finger and thumb or between the two fingers. It is always well to go back upon a suspected tumor, after it is found, so as to feel sure that the sensation is not due to any accidental cause.

During this part of the examination the hand may experience a distinct sensation of rubbing or friction in the abdomen: this may be made out more plainly, at times, by wriggling movements of the hand, or by tryi to rub the abdominal wall against deeper parts; or we may bring it out making the patient breathe deeply while we feel carefully in different par as this makes some of the abdominal organs move with the diaphragm.

Any pain or tenderness elicited during such manipulations should course be noted: the beginner must, however, be constantly on his gua against being misled by the absence of tenderness into supposing that the is no peritonitis, as erroneous ideas on this subject have been too oft gathered from systematic lectures or from descriptions of "typical cases The fact is that in a very large number of cases of tubercular peritonitis children there is no tenderness on pressure at all, and even in some cases acute peritonitis of a fatal nature it may seem to be absent or to be merg in a general uneasiness on handling the abdomen.

The prominent belly, contrasting strongly with the wasted state of th chest and of the thighs, is a familiar appearance in tubercular disease the abdominal organs, even in cases where there may be little or no flu present; and an even more striking degree of the same thing may be foun in cases of malignant tumors of the abdomen. In both of these conditio we may see more or less distention of the superficial veins, and at tim they may be enlarged to an enormous extent. The uniform distention o the abdomen from fluid in the peritoneum often contrasts with more loca ized swellings from tumors there; the discrimination must be made by pe cussion and palpation, as in the case of adults. Fluid in the abdomen i as already stated, often due to tubercular disease, although of course it ma also be due to the existence of dropsy from disease of the heart and kidne but in such cases we have almost invariably more or less dropsy elsewher A suddenly-developed dropsy, localized in the abdomen, may depend, as i the adult, on disease of the liver,—not usually on cirrhosis of the live although this is not entirely unknown, but perhaps due to thrombosis o the portal vein: in such cases we try to make out an enlarged spleen, an we inquire for hemorrhages from the stomach or bowels; we also, of cours examine for jaundice or other signs of hepatic disorder; but the diagnosi must usually remain very doubtful during life, unless the fluid quickl disappears. In bad peritoneal dropsy from any cause we may see hernia protrusions, with fluid in them communicating with the general abdomina effusion.

The chest is, of course, best surveyed when both chest and abdome are completely uncovered; but the actual state of the child must determin whether it is wise to have it so. The appearance of marked wasting wit great distinctness of the ribs; the existence of any of the forms of "pigeon-breast" with prominence of the sternum and an accentuated transvers groove above the liver; the presence of the so-called "beading of the ribs" (or the "rachitic rosary," as it is sometimes termed), consisting in visible and palpable swellings at the ends of the ribs where they join the cartilages; various bad conformations of the chest, whether with depression of the lower end of the sternum or with unilateral distortions interfering with the symmetry of the chest; bulging forward of the sternum, with a tendency to the circular form of chest, indicative of emphysema in older children, as in adults;—all these structural peculiarities can often be sufficiently appreciated at a glance.

But we must likewise notice the chest in action. The awful dyspnœa of croupy attacks, with powerful action of the muscles of the neck and sudden elevation of the upper part of the sternum and ribs, almost in a mass, coupled with recession of the ribs in the lateral region and sucking in of the lower part of the flexible sternum, tells at once of the urgent need for air experienced by the child, and of the mechanical interference with its entry into the lungs. An excited action of the accessory muscles of respiration, with panting and heaving of the chest, but without the recession movements just described, characterizes the attacks of spasmodic action in the child, as in the adult; for, although not very common under twelve or fifteen years, genuine spasmodic asthma in children is not so very infrequent as is often supposed. Marked unilateral respiration, with one side heaving rapidly and the other motionless, is very suggestive of a large pleuritic effusion, and this is rendered almost certain if we detect, on getting a fair view of the chest, that the motionless side is larger and fuller than the other, with obliteration of the intercostal spaces: some rearrangement of the position of the child may be required to ascertain this, as the decubitus is invariably in such cases on the affected side. Marked unilateral retraction and immobility at once suggest, in a child, the results of an old pleurisy or empyema, although of course it may occasionally depend on long-standing pulmonary excavation or on the contraction of a fibroid phthisis. Moderate flattening and retraction under the clavicle, or impaired movements there, fulness over the pericardial area from effusion, general bulging of the tissues of the chest and neck, with crackling on touching it, due to subcutaneous emphysema,—all require detailed examination, and cannot be recognized the moment the chest is seen, as in many of the conditions already mentioned.

STATE OF GENERAL DEVELOPMENT.

Weight—Dentition—Walking.

While the child is being uncovered, for the purpose of having the chest and abdomen examined, an opportunity is afforded of judging of the general development, and this survey must be supplemented by an examination of the limbs and of the head. The large head, prominent belly, and distorted chest may at once fix in our minds the idea of rickets, even apart from any deformity in the limbs; but usually, even in children who have never walked, we may see evidences of rickets in the great prominence of the curvatures of the clavicles,—appearing as if they had undergone repair from fracture,—and in the curved arm and forearm resulting from resting the weight of the body on the upper limbs while sitting up in bed;

enlarged wrists and ankles, and open or soft fontanels, come to our aid confirmations. The wasted appearance of the chest and limbs, contrasti with a great prominence of the abdomen (with or without the presence fluid), has already been referred to as strongly suggestive of tubercul disease in the peritoneum or mesenteric glands, constituting an affection special importance in childhood, as it is relatively frequent at this period life. It is best spoken of as "abdominal phthisis," owing to the frequent uncertain and mixed character of the pathological conditions actually prese The general aspect of the child with such disease is only referred to here, it will require very special consideration in other sections of this work.

Of course in phthisical disease, whether in the chest or the abdomen, may have a wasting which involves the belly also in the general atroph the whole child presenting a uniformly shrunken appearance. But, qui apart from affections of this kind, the whole body may be pretty equal atrophied in a multitude of cases of wasting disease arising from maln trition, due to improper feeding or diarrhœa, even apart from any tuberc lar tendencies. The patient's face is small, and assumes the appearance, many ways, of that of an old man. A good place to judge of the extent the wasting in a child is in the upper part of the thigh in the region of t great adductor muscles. We often see this part hollow and with the sk lying in loose folds. We may test with advantage the tone of the tissu by pinching up the skin here, the raised fold thus made taking a long tin to efface itself in cases of wasting and debility; even the skin pinched on the abdomen may linger as a visible fold to a striking extent.

Along with signs of general wasting we have often badly-formed nail with longitudinal marks amounting almost to cracks; or we may find t curving and clubbing familiar to us in phthisical adults. Frequently alo the spine, and extending towards the scapulæ, we see long soft hairs in weak children; but the importance of this sign must not be exaggerated, as may be found sometimes in those who are fairly strong.

A most important point in the estimation of the development and actu condition of children consists in weighing them. Unfortunately, accura details as to what may be regarded as normal weights for the various ag and heights, in both sexes, are not yet ascertained on a sufficiently extensi scale to guide us in estimating the value of one individual weighing, althoug numerous observations are to be found compiled by Quetelet, Roberts, Bow ditch, and Vierordt. It must be remembered that the normal weight vari relatively for the sexes at different periods of life; that in both sexes varies, of course, with the height; and that with the same sex and the sam height it will vary with the age of the child. Probably also the nationalit leads to variation in weight, as between, for example, American and Britis children. The social position of the children weighed for the purpose o ascertaining averages likewise determines a greater weight for the "mos favored classes" of society. In the case of very young children, the influ ence of nourishment by breast-milk determines for the more favored class in this respect an increased growth and weight in the early part of life; and it can even be traced as exerting an influence for some years after birth. This difference as to children nursed at the breast and those brought up artificially applies chiefly, if not exclusively, to the poorer grades of the community; at least the evidence, so far as statistical data are concerned, applies to these classes only, as the others scarcely come within the chance of such observations being made on a large scale.[1]

---

[1] MEAN HEIGHT AND WEIGHT OF 10,904 GIRLS IN THE UNITED STATES OF AMERICA. (Including 3681 American, 3623 Irish, 585 German, and 1397 Mixed English, Irish, and American Parentage.) Dr. Bowditch.

Abstract from Roberts's "Anthropometry."

AGE LAST BIRTHDAY.	HEIGHT, WITHOUT SHOES.	WEIGHT, INCLUDING CLOTHES.
5 years.	41.0 inches.	40 lbs.
6 "	43.5 "	44 "
7 "	45.5 "	48 "
8 "	47.5 "	52 "
9 "	49.5 "	56 "
10 "	51.5 "	60 "
11 "	53.5 "	66 "
12 "	56.0 "	76 "
13 "	58.0 "	88 "
14 "	60.0 "	96 "
15 "	61.0 "	104 "
16 "	61.5 "	110 "
17 "	62.0 "	112 "
18 "	62.0 "	114 "

MEAN HEIGHT AND WEIGHT OF BOYS AND MEN

Between 4 and 50 Years. English Artisan Class. (13,931 Observations.)

Abstract from Roberts's "Anthropometry."

AGE LAST BIRTHDAY.	HEIGHT, WITHOUT SHOES.	WEIGHT, INCLUDING CLOTHES = 7 AND 10 LBS.
4 years.	38.50 inches.	44 lbs.
5 "	41.00 "	50 "
6 "	43.00 "	54 "
7 "	45.00 "	57 "
8 "	47.00 "	59 "
9 "	49.00 "	62 "
10 "	50.50 "	66 "
11 "	51.50 "	70 "
12 "	53.50 "	74 "
13 "	55.50 "	78 "
14 "	58.00 "	84 "
15 "	60.50 "	94 "
16 "	63.00 "	106 "
17 "	64.50 "	116 "
18 "	65.50 "	122 "
19 "	66.00 "	128 "
20 "	66.25 "	132 "
21 "	......	.....
22 "	66.50 "	136 "
23–30 "	66.50 "	138 "
23–50 "	66.50 "	140 "

But, whatever difficulties beset the estimate of a child's weight as compared with any absolute standard, the relative weight of the child from time

---

HEIGHT AND WEIGHT OF BOYS BETWEEN 13 AND 20 YEARS.

(3695 Boys in Telegraph Service in England.)

Abstract from Roberts's "Anthropometry."

Average Weight in Lbs., without Coat, Hat, and Shoes.

HEIGHT.	AGE AT LAST BIRTHDAY.							AVERAGE IN LBS.	HEIGHT IN INCHES.
	13	14	15	16	17	18	19		
	lbs.	lbs.	lbs.	lbs.	lbs.	lbs.	lbs.		
4 ft. 3	62	......	......	......	......	......	......	62.0	51
" 4	73	70	......	67	......	......	......	70.0	52
" 5	72	69	......	......	......	......	......	70.5	53
" 6	74	74	74	67	......	......	......	72.2	54
" 7	75	76	77	76	......	76	......	76.0	55
" 8	78	80	78	87	81	......	......	80.8	56
" 9	82	83	83	86	......	......	......	83.5	57
" 10	86	86	87	88	94	......	......	88.2	58
" 11	87	89	91	93	93	91	......	90.7	59
5 ft.	90	93	92	96	101	109	......	97.0	60
" 1	96	98	99	101	106	102	119	103.0	61
" 2	96	101	104	106	109	111	113	105.7	62
" 3	103	108	108	110	115	117	117	111.1	63
" 4	111	112	115	115	116	115	120	115.0	64
" 5	......	107	117	115	120	127	121	118.0	65
" 6	......	119	117	122	126	126	130	123.3	66
" 7	......	123	120	125	132	129	138	128.0	67
" 8	......	......	131	126	131	142	144	134.8	68
" 9	......	129	142	132	138	144	140	139.4	69
" 10	......	......	136	144	147	151	137	143.0	70
" 11	......	......	......	......	149	129	150	142.6	71

This table shows not only the average weight and average height of boys, but also the variation of weight in boys of the same height, *according to age*. For example, there is a steady progression with age at the height of 5 ft. 2, boys of 13 being only 96 lbs. and those of 19 being 113,—a range of 8 or 9 lbs. above and below the mean of 105.7.

Although applying to those somewhat older than "children," the inter-relationship of height, weight, and age is well shown, and no similar data for earlier ages are known to the writer.

WEIGHT AND LENGTH OF INFANTS ACCORDING TO THEIR AGE AND THE CHARACTER OF THEIR NOURISHMENT.

(Russow: from Gerhardt's "Handbuch der Kinderkrankheiten.")

CLASS I.—CHILDREN OF AVERAGE WEIGHT AND UPWARDS.								
NOURISHMENT.	WEIGHT IN GRAMMES.					LENGTH IN CENTIMETRES.		
	15 Days.	3 Mos.	6 Mos.	9 Mos.	12 Mos.	15 Days.	6 Mos.	12 Mos.
Breast-milk	3594	5701	7072	8401	9930	51	67	73
Breast-milk, with some cow's milk and starchy food . .	3525	5310	6317	7916	8480	49	64	69

to time is a more definite matter. The weight of a child is so small that great care is required in regard to the estimate of the clothing. The best way in routine practice seems to be to take the weight in the clothes the child wears while in-doors, as this leaves the variation from time to time but trifling, although heavier underclothing and heavier shoes make a little difference. A system of frequent weighing (two or three times a day) has lately been advocated, and has been alleged to bring out some curious results, showing more definitely the somewhat fitful manner in which increase of weight occurs at different parts of the year, and its relationship to increase in height. Such a system of regular weighings of school-children, even if recorded only once a week, might give useful warnings to parents and teachers. Of course the conditions would require to be the same as regards the time of day when weighed and the clothing worn. We might then learn whether the strain of education or the influence of some latent disease was telling on the general nutrition before the deterioration had advanced too far.

In actual practice, however, we have seldom much assistance given us in diagnosis by the records of previous weights in children, although in the course of treatment we often avail ourselves of the balance to judge of the results in this respect and to guide the prognosis. It is in such matters as estimating a general falling off in the nutrition and weight that the opinion of careful mothers and nurses is so valuable, particularly in young children: even when the weight might show but little change, their estimate of the softness of their muscles, or, on the other hand, of their increasing firmness, indicates with considerable certainty the tendency of the case in either direction. The importance of a gradual falling off for weeks or months before the onset of dubious cerebral symptoms is well known in the diagnosis of tubercular meningitis, although in not a few cases this dreadful disease may

---

CLASS II.—CHILDREN UNDER THE AVERAGE WEIGHT.

NOURISHMENT.	WEIGHT IN GRAMMES.					LENGTH IN CENTIMETRES.		
	15 Days.	3 Mos.	6 Mos.	9 Mos.	12 Mos.	15 Days.	6 Mos.	11 Mos.
Breast-milk	3027	4225	5775	6490	7910	49	59	69
Breast-milk, with cow's milk and starchy food	2928	4143	5598	5932	6823	43	55	63
Cow's milk and starchy food exclusively	2900	4089	4744	5254	6128			

The original figures are here given as indicating the relative variations very clearly. For those not quite familiar with the Metric System the following figures will serve as a guide: 1000 grammes, or 1 kilogramme, =2.2 lbs. avoirdupois (nearly); and 10 lbs. =4536 grammes: 1 centimetre =0.3937 inch; 50 centimetres =19.685 inches. The artificial feeding here referred to concerned the poorer classes of society. Relatively better results might be obtained from more careful and scientific substitutes.

seem to surprise the child before any falling off had occurred. Likewis in other obscure affections of a tubercular or scrofulous nature, whether i lungs, bronchial glands, abdomen, or brain, this preliminary deterioratio before pronounced symptoms had appeared often constitutes a point o capital importance in the diagnosis.

A valuable means of estimating the development of children is afforde by the state of their dentition, and the age at which they may have begu to walk, for these are points on which the mothers can usually suppl information.

Particulars as to the order and date of the eruption of the milk-teet will be given in other chapters, so far as these can be reduced to a rule Speaking generally, we may say that in rickets, and all forms of bad an retarded development, the dentition is later in being completed; but i happens not infrequently that in such cases there may be an early start,—perhaps an unusually early start,—followed by a prolonged pause after th first few teeth appear. In not a few cases of bad rickets or of syphilis ther may, indeed, be a complete absence of the teeth even when the child is of a age when all the milk-teeth should have appeared. A backward state of th teething, so that the rickety child at two years may have only as many teet as a healthy child at ten or twelve months, is a common and very suggestiv fact. Very suggestive also is the arrest in the natural progress of dentitio often brought about by some intercurrent illness even in those who have bee healthy. Occasionally, no doubt, the appearance of new teeth in the ver midst of some serious illness is observed; but this is entirely exceptional.

The date of walking varies much in perfectly healthy children. An precocity in this respect is in no way desirable, and no anxiety should b expressed with regard to it if the child seems otherwise quite strong and healthy, unless the period goes beyond the fourteenth or fifteenth month although children in good condition usually walk a month or two earlier When, however, we find a child unable to walk at eighteen months, th chance of this delay being due to rickets is very great, if there are n obvious localized defects in the limbs from paralysis, joint-mischief, etc. Occasionally, indeed, the inability to walk depends on a general deficiency of the development of the whole nervous system, including a mental defect, to which even when very notable the mother is apt to be singularly, or perhaps wilfully, blind, enlarging, it may be, on the remarkable acuteness of her offspring.

As in the case of dentition, the child's progress in standing and walking is often arrested, after a fair start had been made, by the supervention of rickets. The child is then said "to have been taken off his feet," a report which must always suggest the idea of rickets to the physician. Of course any acute illness may operate in the same way, so that after recovery from measles, a bad bronchitis, or a diarrhœa, for example, the child may be found to have lost the power of walking, only to be regained slowly, so that he may appear to be several months behind others of his own age in this respect.

In cases of inability to walk, when brought for advice, we must ascertain by a local examination whether there is pain, or dislocation, or any mechanical defect interfering with the process; we also examine for atrophy, coldness, spasm, and other signs of paralysis in the limb, ascertaining if the child when sitting or lying can move the legs freely; we must also examine the back for curvatures or other deformities; we likewise search, as already stated, for any signs of rickets or for indications of mental defect. The case is often made clearer if we can ascertain that the child has ever walked, even for a month or two, or if any acute attack of illness was followed immediately by the loss of power complained of.

Precocious development of the sexual organs, or signs of premature puberty, are occasionally seen in children, in both sexes. When such are noticed, we must inquire for any unnatural excitation of the parts by the patient or the nurse, or for any evidences of masturbation, which of course at that age may assume very unusual forms if present. In young girls the premature signs of puberty may depend on some ovarian tumor. The writer had recently a child of three and one-half years under his care, with an abdominal swelling, where the well-developed mammæ, the presence of hairs on the labia pudendi, which were unduly full, and the occasional discharge of a material like butter from the vulva, enabled the diagnosis of an ovarian tumor to be made with a considerable degree of certainty. The tumor proved to be a very large solid ovarian tumor; but the shock of the very serious operation demanded for the removal of such a large solid mass was too much for the child.

EXAMINATION OF THE HEAD.

The development of the child as regards the bony system has been alluded to incidentally in connection with the distortions of rickets; but special attention must often be directed in various cases to the state of the head. The size of the head varies enormously, and it is not possible to give absolute measurements of any great diagnostic value. We can often judge of it best by a comparison, by means of the eye, with other portions of the body, particularly with the chest and abdomen; sometimes we can with advantage take measurements of the circumference of these three parts, particularly in watching their relative growth, in young children, over a period of months. The circumference of the head taken at the occipital protuberance and the prominent part of the brow is greater at birth than the circumference of the chest, which is best measured a little below the nipples to escape the angles of the scapulæ. This difference is maintained for some months after birth, but in well-developed children the measurements, as taken above, become nearly equal between the ages of one and two years, and after four years the chest rapidly increases as compared with the skull.[1] In badly-developed children the chest is much longer in asse ing its equal or superior size, this being delayed in many such cases till t age of five years.

The size of the head depends, of course, very much on the parentage the child; but it is often too large and sometimes unduly small in disea In rickets the head looks large and the face small; the top of the head usually rather flat, and sometimes gives the idea of a square shape. T fontanels are often much wider than usual for the age, and may inde remain unclosed, or only covered by a soft membrane, for a year or tv after they should be closed: the measurement of the fontanels, particular the anterior, can often be made with advantage in inches (both antero-pc teriorly and transversely) from time to time in watching a case, so as judge of its progress towards closure. The sutures in rickets can often felt with undue facility, but they are seldom actually separated and bulgi as in hydrocephalus; the thickened edges of the bones can often be felt the sutures, which assume in this way an undue prominence. Here a there at the sides, and in the occipital region particularly, the point of t finger may feel the soft spots of cranio-tabes. Whenever such characte are found in the skull we search for other signs of rickets: in the chest f the characteristic distortion and the so-called "beading" of the ribs; f curves in the long bones of both the upper and the lower extremities; mor over, the actual state and history of dentition, the date of walking or giving over walking, if the child is sufficiently old for this to come up, a the presence or history of tenderness in the bones on handling the child, a come in to help the diagnosis. Rickets has such wide-reaching effects, a has, in particular, so important an influence on nervous disorders, that t large head may readily lead the inexperienced to ascribe laryngeal spas and general convulsions to some grave disease of the brain, while really t case is essentially due to rickets and perhaps readily curable.

In examining the skull we may find thickened masses or bosses arou the fontanel especially, or, on the other hand, thinned portions of bone, so

---

[1] See "Third Report of the Clinical Hospital, Manchester," by Dr. James Whitehea London, 1859. The following table may be useful for reference, relating to *children of go development.*

NO. OF CASES.	AGE.	HEAD, INCHES.	CHEST, INCHES.	DIFFERENCE BETWEEN HEAD AN CHEST.
100	One day.	13.75	12.94	Head more than chest 0.81
66	6 to 12 weeks.	15.25	14.25	" " " 1.00
75	6 to 8 months.	16.68	15.58	" " " 1.10
71	11 to 13 "	17.80	17.20	" " " 0.60
67	21 to 24 "	18.38	17.85	" " " 0.53
50	34 to 36 "	18.70	18.61	" " " 0.09
60	4 to 4½ years.	19.20	19.72	Chest more than head 0.50
46	6 to 6½ "	19.51	20.76	" " " 1.25
40	9 to 10 "	19.56	21.31	" " " 1.75
31	11 to 12 "	20.00	23.46	" " " 3.46

or almost approaching to the character of holes (cranio-tabes). Both conditions have been described by Parrot as occurring in rickets; but, as he considers this disease as a manifestation of syphilis, we require to remember this in connection with the detection of similar conditions in congenital syphilis.

The enlarged head of hydrocephalus usually differs from that of rickets so clearly that mistakes do not often arise after a careful examination. The upper part of the head is not flat, but often arched or vaulted. The fontanel is not merely wide or unclosed, but often prominent and tense; the sutures issuing from it are frequently wide, with a protrusion between the edges of the bone. The face looks small in comparison with the head, and there is a peculiar downward look of the eyeballs, with an unduly large part of the white sclerotic visible in the upper segment, from the same cause. The enlarged head sometimes remains as a permanent record of the occurrence of hydrocephalus in the past, the illness having run its course, the sutures and fontanels being all firmly closed. In such cases the intellect may be defective, presenting the form of idiocy called macrocephalic; but enlargement of the head in this way by no means involves mental deficiency as a necessary consequence.

It was hoped at one time that auscultation of the head would supply important diagnostic or differential data in doubtful forms of enlargement; but this hope has scarcely been realized. No doubt, on listening over the unclosed fontanel of hydrocephalus we may frequently hear a whiffing sound synchronous with the pulse; but similar sounds may at times be heard in cases of rickets also, so that probably the open fontanel has more to do with the phenomenon than any changes within the skull. Auscultation may be practised either with a stethoscope held lightly over the fontanel, or, better perhaps, by direct application of the ear to the infant's head, with the interposition of a thin napkin or handkerchief if desired. During the auscultation very loud and at first unintelligible rushing sounds are heard, complicating any whiff or murmur which may be present: these are due to breath-sounds resonated in the nasal passages and cavities; they have, of course, no significance, and may be heard in healthy children also, but, as we seldom auscultate the head except when it is under suspicion of disease, they are apt to puzzle the beginner.

Percussion of the skull has been practised of late years in children, as in adults, in connection with eliciting localized tenderness due to superficial lesions in the brain or its membranes. Various parts of the skull are struck with the point of the finger; by questioning the patient, or by watching the expression of his face, we judge if any one spot seems specially tender. We must, of course, compare corresponding parts on the two sides of the skull, and if we detect a seemingly tender spot we come back upon it once or twice, to see that the difference did not arise from any mere accidental variation in the strength of stroke or the irritability of the child. Of course, if tenderness is made out, we must see that no mere abrasion or

similar state of the scalp is responsible for the difference. When clear
marked, the results of this method may have a certain value in fixi
the position of a lesion indicated otherwise by localized convulsions
paralysis.

A further application of percussion to the skull consists in a study
the sounds thus educed. Although promulgated more than thirty yea
ago,[1] this method has not been much practised. Of late a colleague of t
writer's (Dr. William MacEwen) has called his attention to a peculi
change in the percussion-sound found in the case of children affected wi
accumulations of fluid of various kinds within the skull: the change
percussion-sound is difficult of expression, but it is in the direction
undue resonance or of a tympanitic quality. Dr. MacEwen thinks th
the sound may even vary on the two sides of the skull according to t
position of the fluid. Certainly a peculiar quality of the percussion-sou
has been demonstrated by Dr. MacEwen in various cases, and has be
verified by the writer in certain children with cerebral tumors and effusi
into the ventricles of the brain; but as yet it seems scarcely possible
formulate the conditions which give rise to this change. In addition to t
difference between dull and clear or tympanitic sounds, we occasional
obtain a curious cracked sound on percussion over the parietal regio
suggesting the idea of the sutures shaking against each other, although th
may be found not only in young infants with unclosed fontanels, but also
those with firmly-united sutures. In the further study of this matter t
following points are suggested as worthy of careful consideration. 1. T
physical surroundings of the head of the child: thus, the percussion-sou
may be found very different according as the head is allowed to lie on
pillow or is supported on the lap of the nurse, and equally according as t
child lies or sits up. What may seem a highly tympanitic note while t
head rests on pillows may lose all this quality on a change of positio
2. The state of the fontanels and sutures, as to closure, must be kept
view as probably influencing the sounds. 3. Variations in the state of t
bones as regards thinness or thickness over the closed fontanels or arou
them, and in the region of the squamous portion of the temporal bor
are probably very potent in affecting the sound produced by percussio
4. The position of the head as held erect, or to one side or the other,
directed downward, or lying flat with the face upward or downwar
should also be kept in view, in connection with the idea that the presen
of fluid and its varying levels within the skull may determine a chan
of note.

Smallness of the head is no less serious a sign than enlargement, ar
when extreme it is often associated with idiocy (the "microcephalic" for
of some writers). Moderate degrees of smallness must not be judged

---

[1] Betz, Ueber Perkussion, insbesondere über Perkussion des Schädels, *Schmidt's Jah*
bücher, Bd. lxxxvi., 1855.

rashly, for, if the development and shape are otherwise good, this may result from family peculiarities of little import. With the microcephalic idiots we often find obvious deviations from the ordinary contour, sometimes giving a peculiar bird-like aspect to the head.

Occasionally unilateral alterations in the skull are detected as connected with obvious or obscure disease in the central nervous system; while unilateral atrophy, or more rarely unilateral hypertrophy,[1] may lead to a want of symmetry dating, it may be, from birth. Another form of want of development of the side of the head and face arises in connection with long-standing wry-neck in early life, and a slighter form has been ascribed to injurious modes of carrying the infant, so as to hinder free movements in all directions.

The occurrence of the "blood-tumor" called cephalhæmatoma, appearing soon after birth in the scalp of the infant, can usually be easily distinguished from the much more serious disorder due to defect in the bones of the skull, with protrusion of the brain substance or membranes, to which the name encephalocele is applied. These important subjects will be discussed in other sections of this work; but it may be worth while to mention the occurrence of another form of "blood-tumor" or hæmatoma in the pericranium, which may give rise to discoloration of the eyelids (as in cases of hemorrhage there from fracture of the skull): the writer has seen such an accumulation of blood in this situation, so extensive and so uniformly diffused as to simulate, at the first glance, a case of hydrocephalus. On examining with the hand for the sutures, however, the whole surface of the scalp was soft and fluctuating, and the bones of the skull could be felt only on dipping the fingers down suddenly through the fluid.

EXAMINATION OF THE BACK.

An examination of the spine of children reveals at times the two well-known forms of curvature with which we are familiar in the adult. Acute or angular curvature, described by Pott, occurs indeed with special frequency in early life, and its appearance is so characteristic as to require little notice here.

The lateral curvature is, of course, much less common in children than in girls at or a little beyond the age of puberty; but a typical lateral curvature may occur even in young babies, and in such cases we must see whether there is any error in habitually carrying the child so as to look in one direction only. Very often the lateral curvatures in children are merely secondary results of serious antecedent disorders. A pleurisy followed by retraction of the side, an infantile paralysis, grave hip-joint disease, fractures or dislocations in the leg or thigh, and indeed anything which shortens one of the lower limbs as compared with the other,—all these give ris lateral curvatures.

---

[1] See on Atrophy a very good article in Pepper's "System of Practical Medicine," vol. v., by Dr. Charles K. Mills. For Unilateral Hypertrophy, bibliographical references will be found in a paper by the writer in the *Glasgow Medical Journal*, November, 1884.

A very common curvature found in young children may mislead beginner by suggesting the presence of caries with Pott's curvature, w all that exists is simple softness of the bones and muscular weakness, s as occur so frequently in rickets. In these cases the back in the lower do and lumbar regions is found to bulge or curve backward when the chil made to sit; there is no true angular projection, and on taking the wei off the spine by the recumbent posture the curvature disappears.

An opposite curvature of the lower part of the column, with a holl instead of a projection, gives the spine a "saddle-back" appearance in t situation; there is a projection backward of the upper part of the sp about the scapulæ, and the name "lordosis" has been applied to this vari The deformity is due to paralysis or weakness of the muscles of the ba and it acquires special significance in the diseases of childhood from t "saddle-back" constituting one of the most striking features of the "pseu hypertrophic muscular paralysis," an affection which we may say is limi to children. The same condition of the back, however, may exist in cl dren as a more isolated affection and entirely independently of this form paralysis.

The peculiar fixity of the head and neck found in occipito-atlant disease needs only to be noticed in a word: it occurs with relative frequer in childhood.

The congenital malformation termed spina bifida requires also to mentioned here: its presence may account for paralysis and convulsions infancy. The gravity of the condition turns in part on the level at whi the tumor exists in the spine, and in part on the nature of the contents, matters which are fully discussed in special chapters.

TEMPERATURE—THERMOMETRY.

In all acute illnesses, and in very many of the chronic ailments, children, the determination of the presence or absence of pyrexia, and estimation of the degree and persistence of this pyrexia if present, co stitute points of capital importance in the diagnosis; very often t same data guide the prognosis and treatment. Formerly this was dete mined in part by the hand applied to the child's skin, say over the abdome and in part by the counting of the pulse. The number of pulse-beats in child, particularly if very young, is but a poor guide in the estimation fever. The pulse is made to run up by so many causes that it is only wh we feel satisfied that we have counted it in a period of quiescence, as duri sleep, that the numbers have any great meaning. The hand applied to tl skin is confessedly a rough method, but when the sense of increased heat very notable, one of experience may get considerable assistance from it. is when the skin feels to the hand little if at all hotter than normal that w may all make grave mistakes if we trust to such a method: again and agai even those of experience have supposed a child to show no increase of heat, when it has been found that the temperature was 3° or 4° F. higher than normal, or perhaps even more than this.

The introduction of the thermometer into regular clinical work has been of signal service, but in no department has it been of more practical value than in dealing with children. In using a thermometer it behooves us to know clearly what we are aiming at. It has already been admitted that the application of the hand may teach us much; and if all we desire is to record, as it were, in figures such information as the hand might give, then almost any method of using the instrument, however faulty, may serve such a purpose. But in dealing with instruments of precision, such as good thermometers, we are exposed to new fallacies if we do not use them properly. It is all very well, and perfectly fair, to say that the child does not seem hot as judged by the hand; we know, so far, the value of such a rough statement; but to say that the thermometer shows that there is *no pyrexia* implies something very different; and if the statement is founded on an error, we have through this fallacy not only lost what guidance the true temperature might have given, but we are actually liable to be put wrong much further than if no observations had been made with the instrument at all. While, therefore, not disparaging the practical assistance which rough and rapidly-taken observations may yield, we must see that we understand their real value. A child with a measly or a scarlet rash may be found to have, on some rough observation, a temperature of 102° F.; this may be enough for the purposes of diagnosis, showing that with the rash there *is* a distinct degree of fever; and if the child does not seem very ill, it may really matter but little whether the temperature is 102° F. or whether, if properly taken, it might come out as 103° F. or 103½° F. Of course a very great elevation (106° F. or 107° F.) might mean something very different; but in such a case the obvious state of the child would likewise be different.

It is when, perhaps, with a low surface-temperature there is a very distinct increase of the internal heat that errors from faulty observations become positively misleading. When, for example, we may be dealing with a case of enteric fever towards the end of the first week, and the thermometer, badly applied, shows a maximum of only 100° F. instead of 102° F., we might here almost infer that enteric fever was excluded by such an observation, if we trusted to the "instrument of precision." Or the hectic fever of obscure phthisis may be present, but missed by faulty use of the instrument; and so we might be led, if trusting to the record, to set aside the diagnosis of phthisis as unlikely, owing to the supposed absence of the fever which we had really failed to discover. Such mistakes are constantly being made, and the educational effect on those who make them is towards carelessness, inaccuracy, and confusion.

When the writer had to take charge on one occasion of patients in a children's hospital newly opened, he was confronted by the striking fact that nearly every child in the ward had subnormal temperatures, some o them to a considerable extent. It was evident at once to him that the fau lay not in the children's temperatures, but in the taking of the observation He explained to the head-nurse in charge that the temperatures were sul normal because they were badly taken, that probably not one of the childre had subnormal temperatures, and that very likely several of them we more or less feverish. The astonishment of the nurse was mingled wit indignation, for she had just taken her nurse's certificate with distinctio and, to do her justice, probably could take temperatures as well as mo practitioners of medicine. First by taking the temperature in the rectu it was shown to her that the heat of the child was *not* subnormal there, a then by great care in the taking of the axillary temperature it was show that there also the mercury rose to the normal. How, then, had the mistak arisen? By trusting to the rules given for inserting the thermometer in the axilla and leaving it for a stated time, regardless of the progress of t mercury upward, and reading it off then.

A child's axilla is often a very small affair, and especially when muc wasted there is scant covering even for so small a thing as the bulb of thermometer; the instrument readily slips out behind, or falls down, or t arm ceases to be applied; the regulation number of minutes allowed fin the mercury indicating the temperature of the night-dress or the adjace air, but in no sense the temperature of the *closed axilla*. To obtain thi we must see that the axilla *is* closed; and in young children this usuall means that the observers must hold the arm to the side themselves. B the temperature of the axilla, as open, is often very different from its te perature after it is closed; this, indeed, is a gradually increasing quantit as the surfaces of the skin applied to each other recover from the coolin influences of the previously interposed air when separated. All these poin are easily learned by a few observations with thermometers long enough t project, so as to be easily read from minute to minute, and without an maximum-registering index. With such an instrument, we often see th with the lapse of minutes the mercury falls instead of going on steadil rising; we learn at once, under such circumstances, that it must have slippe out in some way; if properly applied, we can read its gradual ascent, fro minute to minute, till it attains a stationary point; only when *this stationar level is maintained for four or five minutes* do we feel sure that the maximu is reached. By selecting different cases we may see the greatest diversit in the rapidity of the rise of the mercury; and it is this that has confuse many. At times the maximum is reached in one minute or so, and by wai ing four minutes longer we have the security of this being the maximu and so it may be rashly said that the temperature can be taken with absolut certainty in five minutes; and so it can, under such circumstances. Ver likely in an intense scarlet fever, with deep injection of the skin, we get rapidly-attained maximum in the axilla, particularly if the arm happens t have been close to the side, as by the child's lying on it; but if the ar has been separated from the side, or tossed about so as to take up colder air frequently into it,—if the child is wasted to "skin and bone,"—if with the feverishness there is a certain tendency to collapse,—then we have to wait till the influence of the cooling air on the skin is neutralized by the increased cutaneous circulation, favored by the apposition of the arm to the side; in this way a long time may be required for the observation, not from any want of sensitiveness in the instrument, but because the tissues of the armpit have been cooled and we must wait till the axilla itself is warmed up to its maximum heat, for it is only then that it is in any sense an index of the heat of the blood, which such observations really aim at estimating. Experience teaches us that when the thermometer in the axilla remains stationary for five minutes the maximum for that particular time of the day is attained with practical certainty. Under the adverse conditions referred to above, we often have to wait ten, fifteen, or twenty minutes before the stationary point is maintained for the requisite period of five minutes; hence fifteen to twenty-five minutes may be required in such a case, instead of five minutes as in the other. All this would have been readily learned by any of those really interested in the taking of temperatures, if the introduction of short thermometers had not made readings *in situ* very difficult, and if maximum-registering indexes had not misled the observers very often; for of course the index remains say at 99° F. for many minutes, or indefinitely, even although the mercury has really fallen to 95° F. owing to the bulb having slipped out behind into the child's night-dress; whereas if no index existed, this fall in the thermometer from such an accident would immediately have arrested attention whenever the reading was next made, and so have led to a correction of the faulty position of the instrument.

On account of the great delay often inevitable in cases of wasted children, and of the uncertainty as to the results unless the observation is supervised with the utmost closeness, axillary observations, especially in infancy, have been almost discarded by some, particularly as many young children resist and resent the introduction of a thermometer into their axillæ, so as to make its retention there for ten or fifteen minutes almost impossible. Temperature-observations taken in the rectum can be made rapidly and with great precision in three or four minutes (for, the cavity being *always closed*, there is no time required to counteract the cooling influence of the air, as in the mouth or the axilla); young children often object less to the introduction of the thermometer there, where it is unseen, than to its being placed in the axilla; and even if there is a little resistance, the period is so much shorter that with care this can usually be overcome. The child is placed on the left side in bed, or on the nurse's knee, the buttocks can be loosely covered with a blanket or shawl, and the thermometer allowed to project, after the bulb, properly oiled, has been introduced into the bowel, for a couple of inches: the observer should hold the thermometer and place the hand on the pelvis, to guard against sudden movements displacing the instrument. The mercury rises rapidly, and when stationary for one minute, in this situation, we know that the maximum has been reached; this always occurs within three or four minutes.[1] But few fallacies beset this method, the introduction of the bulb within a mass of hard fæces being one of the chief, although even then the error is probably trifling. Of course if the bulb were very large a certain time would be required to allow the blood to warm up the mucous membrane after parting with its heat to the mercury; but the bulbs of clinical thermometers are usually so small as to reduce this loss of heat to insignificance, in view of the mucous surface being richly supplied with rapidly-circulating blood.

Another source of serious mistake, in connection with temperature observations, arises from judging as to the pyrexia from one observation and particularly in concluding as to its absence in the case from isolated records. It constantly happens that a child's temperature is practically normal during the early part of the day, although by night it may be highly feverish. We must, therefore, consider whether the time of day has been favorable for catching the evidence of pyrexia. But even when fever is present we must constantly remember that in the feverish child, to a much more marked extent than in the healthy,[2] there is a *daily curve of temperature*, and that, to be even roughly comparable, the hours of observation from day to day must be approximately similar. In serious cases, or in connection with therapeutic measures, we may wish to know how far the high temperature is continuous, or to what extent remissions occur from hour to hour. The only way to secure a fair idea of the character and severity of the pyrexia is by reducing frequent observations (say every two hours) to the form of a curve; otherwise we are constantly liable to be misled by having our observations complicated by accidental elevations or depressions of the temperature, which may really be pursuing a practically unaltered course.

It is amusing in one sense, in another somewhat painful, to hear at times certain practitioners speaking complacently of their remedies—perhaps a little digitalis or aconite, or a grain or two of quinine—"bringing down the temperature" to the extent of a degree Fahr. or perhaps something less, when we know that no special care has been taken to have the temperature accurately taken at either observation, and that the difference they pride themselves on is probably the balance of errors,—that even if the observations were both quite accurate, the time of day very likely accounts for the diminution, for these remedies are often given at night, when the fever is high, and the improvement is found in the morning, when the pyrexia naturally falls,—and that, even if an attempt has been made to compare similar periods of the day, these isolated observations may give a misleading conception of the course of the pyrexia in the intervals. Even more extraordinary is it to see the influence of iced applications to the bare chest and abdomen, in reducing the internal temperature in fever during their use, gravely proved by reading a thermometer put into the axilla, under such circumstances, five minutes after its introduction! The writer had this influence demonstrated to him at a well-known hospital, where those in charge seemed highly pleased with their scientific methods!

---

[1] This method was advocated strongly by Ziemssen; see his "Pleuritis und Pneumonia im Kindesalter," Berlin, 1862. It is referred to by the writer in his paper on Normal Temperature in Children, *Glasgow Medical Journal*, February, 1869, and more specially in his paper on the Temperature of Children in Phthisis, *Glasgow Medical Journal*, November, 1869.

[2] The writer's opinion is that the temperature in healthy children cannot be correctly spoken of as either higher or lower than in adults; it is in a sense both; the daily range is greater, amounting to about 2° F. or even 3° F. The minimum in health is attained shortly after midnight, and the maximum in the afternoon; the temperature falls rapidly in the evening, about the time the child goes to sleep. It may range from 97° F. to 100° F. in the rectum in healthy children.

Of course the physician must judge which cases are those calling for frequent and scrupulously accurate observations on the temperature, and he must judge how far children should be disturbed in this way. The practice of medicine is an art, not a science, and we must often be content with approximate and roughly accurate methods; but let us understand clearly when we are scientifically accurate and when roughly practical: we are bound to be more rigidly exact in our proofs when we are vaunting the success of some troublesome, disagreeable, or dangerous method of treatment.

The various degrees of temperature may be regarded, so far, as having the same significance as in adults; but in the child the temperature is more mobile, and trivial disturbances of the digestive system may make the thermometer run up very quickly to a great height. In the interpretation of the records we require to be guided by the results of actual experience in clinical thermometry, and we must not conclude as to their significance from *a priori* reasoning. The following table, given by the writer in his "Clinical Manual," may be found useful as a guide:

Below	35° Cent.	= 95° Fahr.	Very low or collapse temperatures.
	36° Cent.	= 96.8° Fahr.	
About	36½° Cent.	= 97.7° Fahr.	Subnormal temperatures.
Normal,	37° Cent.	= 98.6° Fahr.	Normal temperatures.
About	37½° Cent.	= 99.5° Fahr.	Slightly above normal, or sub-febrile temperatures.
	38° Cent.	= 100.4° Fahr.	
	38½° Cent.	= 101.3° Fahr.	
About	39° Cent.	= 102.2° Fahr.	Moderately febrile temperatures.
	39½° Cent.	= 103.1° Fahr.	
About	40° Cent.	= 104° Fahr.	Highly febrile temperatures.
	40½° Cent.	= 104.9° Fahr.	
Above	41° Cent.	= 105.8° Fahr.	Hyperpyretic temperatures.

Very high temperatures (106° F. or more) and very low temperatures (under 96° F.) are necessarily fraught with danger; but a *sudden* rise of temperature (to 104° or 105° F.) may sometimes give ground for hoping that we are dealing with a trifling febricula; on the other hand, a moderate temperature (102° to 103° F.) with cerebral symptoms may, just because of its moderation, give rise to the most grave apprehensions of a deadly meningitis, whereas with a higher temperature (say 105° or 105½° F.) v might hope that the cerebral symptoms were dependent on an incipie pneumonia or some less fatal disorder. The figures must be interpreted n only in view of the other symptoms, but also in view of the known facts medical thermometry.

PULSE.

The pulse in childhood has ceased to be regarded as any great criteri of the degree of fever, having been superseded to a great extent by the u of the thermometer; but its value in many other ways is still very grea The strength of the pulse in childhood affords, as in the adult, one of o best guides in estimating the general strength of the patient, although both ages we are liable to many mistakes and surprises in relying undu on this sign. The correlation of the pulse and temperature is often ve suggestive. At the beginning of enteric fever we may have a pretty hig degree of pyrexia (say 102° or 103° F.) with almost no elevation of t pulse-rate, a combination always suggestive, when ascertained; very ofte however, the low pulse and the apparently cool state of the skin may le the physician to omit taking the temperature at all. Towards the end such a fever the pulse may be higher in proportion than the temperatur and it may continue, probably through weakness, to be very high even aft the defervescence is complete.

A slowness in the pulse has often great significance in the diagnosis o cerebral affections, and especially of meningitis. At the beginning of t illness, with distinct elevation of the temperature, we may find the pul rapid, but with the advance of the disease the temperature may fall to som extent, and with more or less drowsiness the pulse usually falls, and ma even become extremely slow (say about sixty beats per minute): this always of evil omen and aids the diagnosis of this disastrous disease. Wit the advance of the disease, after the temperature has become almost norma we may find the pulse running up to an almost uncountable height a da or two before death.

Closely allied to slowness is irregularity and intermission in the puls This also, occurring with headache, sickness, moderate fever, or other sign of meningitis, is always of grave import. The irregularity is of two kind and both are found in meningitis. We may have the pulse pretty regul as to rate, but with a succession of a few beats now and then having dis tinctly less strength; or we may have the irregularity in the rate of a fe beats, quite irrespective of any influence of the respiration-movement which we must remember have some effect on the pulse-rate even in health Or again, with or perhaps without much irregularity, we may have inter mission in the pulse, a beat being lost every five or ten or twenty beats This also occurs in brain-disease of various kinds.

Irregularity and intermission of the pulse occur in other than brain-dis orders, notably in cases of pericarditis in its early stage, and also in acut endocarditis: probably on this account we have irregularity in the pulse not uncommonly, in chorea. Of course it is often present, as in the adult, in valvular disease of the heart. In the extreme stage of feverish illness, flickering or irregular or intermittent pulse indicates the grave condition of the patient, but in such cases the diagnosis is already made, as a rule.

Much unnecessary alarm has sometimes been caused by detecting an intermission in the pulse-beats in the case of children not obviously ill, to any serious extent at least, particularly during sleep, or it may be in the evening before going to bed. With medical parents this discovery is apt to excite alarming ideas of incipient meningitis; but it is to be construed only as an indication of a certain amount of weakness, perhaps of a merely temporary character. It is similar in kind to the intermission in the pulse often found in children after their recovery from enteric fever, during their sleep at nights: if the general indications are favorable, no stress should be placed on these intermissions.

The "pulsus bigeminus," the "pulse of high tension" in renal disease, the "aortic regurgitant pulse," the "dicrotic pulse," and other peculiarities best brought out by the sphygmograph, all occur in childhood; but their significance is similar to that in the adult, and need not detain us here.

PHYSIOGNOMY.

The idea of defining special temperaments and diatheses from the general aspect of the patient is now abandoned by most: at present it is felt that the necessary precision is unattainable for any useful classification of temperaments.[1] And yet, every one of experience can recognize certain peculiarities at a glance, bearing vitally on the diagnosis; not always so much of a build and complexion indicative in themselves of danger, as of evidences of past or present disorders which from their constitutional character may throw light on the future. Such are the evidences of glandular enlargements in the neck or old scars there or elsewhere from scrofulous disease; the evidences of old ulcerations of the cornea; the sunken nose and notched teeth of syphilis or the mucous patches or eruptions at the child's lips or anus dependent on the same disease. At times, also, the peculiar complexion, with much ruddy color, may suggest the inheritance of a scrofulous tendency, although this may appear, at the time of the observation, in any overt manner at least, only in other members of the family. The delicate build, with fine eager features and look of premature beauty, may suggest fears of tubercular tendencies in one; while coarse features with thick lips and reddish hair may give rise to forebodings of a similar kind in another. These very different types of two classes frequently affected with pulmonary consumption have been made the subject of experimental researches by means of composite photography;[2] and it is possible that the growing application of photography in the study of children's diseases may lead in the future to some more clear apprehension of the rather indefinable, but still very important, impressions gathered from the aspect of a child by one of experience.

---

[1] Jonathan Hutchinson, The Pedigree of Disease, London, 1884.

[2] Mahomed and Galton, Guy's Hospital Reports for 1881.

Coming to pallor of the complexion, we are in presence of something more definite. Its significance as a sign of anæmia is recognized, as in the adult, by a comparison of the color of the mucous membranes, or, if need be, by actual testing of the blood-color, and by ascertaining if the general symptoms of anæmia, such as breathlessness, giddiness, etc., are present, or by a physical examination of the veins and heart for anæmic murmurs.

Occasionally, although very rarely in childhood, we have the combination of anæmia with the bronzing of Addison's disease. The presence of freckles in abundance on the face, and of pigmentation about the cheek-bones and brow, is sometimes, apparently, connected with the existence of pulmonary consumption.[1]

The presence of jaundice in children is recognized as in the adult, and special sections of this work deal with several peculiar forms found in early life and with the spurious jaundice of new-born children.

But, apart from obvious jaundice, we sometimes see a dark complexion, somewhat allied to it, leading one to feel that those with this "bilious temperament," as it is often called, are specially liable to digestive disorders, with a tendency to great feverishness and headache in such attacks; while with a blond or florid complexion we often see that children are specially liable to great cerebral excitement and delirium from very trivial ailments.

The appearance of flushing in feverish illnesses of all kinds; the flushed cheek or cheeks in the early stages of cerebral inflammations; the combination of flushing and duskiness in many pulmonary inflammations; the more pure duskiness of suffocative bronchitis, advancing through various degrees to alarming lividity; the combination of duskiness and pallor in the face with coldness of the surface; the successive redness, blueness, and blackness perceptible in bad paroxysms of whooping-cough; the extraordinary blueness, aggravated by crying, seen in the "morbus cæruleus" of children affected with congenital malformations of the heart,—all these are physiognomic features of the utmost value.

Sweating is a common feature in certain stages of febrile diseases, whether in children or adults, and the cold sweats of exhaustion also occur in childhood. But in rickety children very profuse sweating of the head and neck, especially when the child goes to sleep, may occur in the most extreme form without any fever whatever.

Distention of the veins of the face and neck may occur to a notable extent in all forms of dyspnœa, and in cases of croup the outstanding veins in the neck often form serious impediments in the performance of tracheotomy. In some chronic affections, and in weakly children, the veins are often unduly visible, and the blueness of prominent nasal veins, and of prominent jugulars, is often very noticeable in rickety children.

---

[1] Jeanin, Des Pigmentations cutanées dans la Phthisie pulmonaire, Paris, 1869.

The appearance of the child lying asleep with eyes half open has since the time of Hippocrates been regarded as of bad omen and indicative of grave brain-disease. Taken roughly, there is of course much truth in this widely current idea; but it is a mistake to attach great weight to this state of the eyes, especially if only an occasional condition, as it undoubtedly occurs not infrequently apart from narcotics, without any implication of the brain, and indeed without any great danger of any kind.

As to special appearances of the face indicating suffering from disease in the head, or in the abdomen or the chest, the writer cannot give directions; for personally he has failed to recognize any really distinctive appearances. No doubt knitting of the eyebrows is common in headaches, and so in the painful paroxysms of brain-affections; and a certain pinched aspect of the face, bordering on or passing into a collapsed appearance, is common in bad abdominal cases; but distinctive features are often absent.[1]

The characteristic appearances of the various febrile rashes and eruptions and of the numerous cutaneous affections of childhood demand such careful and detailed study that it is better to leave them to be dealt with in their special sections.

THE CRY.

The cry of the child has been regarded as capable of affording, along with the expression of the face, distinctive indications of the site of the disease, whether in head, chest, or abdomen. No doubt, however, this has been exaggerated.

It has been pointed out by certain writers that the cry consists of two portions, the expiratory and the inspiratory, and that under certain conditions either one or the other of these portions may be modified. When there is not merely an expiratory cry or moan, but a loud or inspiratory portion ("*reprise*") as well, we may regard the presence of pulmonary mischief as unlikely; but the writer has seen a child crying so vigorously as to make it appear almost unnecessary to strip the clothes off for an examination of the chest, and yet on doing so, after much hesitation, pulmonary consolidation has been found. The cry must, therefore, be interpreted with caution. Crying only, or chiefly, immediately after coughing suggests th idea of pain being set up thereby, as in pneumonia, pleurisy, and some forn of bronchitis. A moaning cry is, of course, a clearer indication of loc suffering or general distress than the lusty cry of mere irritability, sleep ness, or bad temper. Crying with wriggling movements of the pelvis an legs has been regarded as a sign of colicky or intestinal pain. Loud cryin with somewhat similar movements of the legs, seems at times to be due t pain in the kidneys or bladder from gravel. The loud piercing shriek, a if from some sudden dart of pain, has been so frequently noticed in cases o cerebral disease as to have received the name of the "hydrencephalic cry: its absence counts for nothing, and its presence in actual cases of this diseas is not always so typical as to count for much.

---

[1] "Jadelot's lines" are thus given by Dr. Eustace Smith in his "Practical Treatise on Diseases in Children," London, 1884, p. 7:

"The *oculo-zygomatic* line begins at the inner canthus of the eye, passes thence downwards and outwards beneath the lower lid, and is lost on the cheek a little below the projection of the malar bone. This line points to disease or derangement of the brain and nervous system.

"The *nasal* line rises at the upper part of the ala of the nose and passes downwards, curling round the corner of the mouth. This line is a constant feature of abdominal mischief, and is never absent in cases of gastro-intestinal derangement.

"The *labial* line begins at the angle of the mouth and runs outwards, to be lost in the lower part of the face. This is more shallow than the preceding. It is a fairly trustworthy sign of disease in the lungs and air-passages."

Continuous crying or screaming is so often found, by the sequel, to b due to earache, that this should always be thought of in obscure cases; an the result of an examination of the ears, or decided relief from the use o hot or narcotic applications, may clear up the diagnosis; or perhaps th alarming symptoms simulating meningitis may disappear after the discharg of matter from the ears.

In croupy affections the cry may be hoarse.

Mothers and nurses from their experience of healthy infants naturall think of crying as a sign of hunger; but in the presence of serious illnes hunger is less likely to cause crying than thirst, especially in feverish case or where copious discharges from the bowel have drained away much flui from the system.

The absence of crying is often of graver import than its presence. Th sick child, ill and exhausted beyond endurance, may only wrinkle up th lips, as if to cry, without any sound; or in bad pulmonary cases, or even i rickets, the child may not be able to spare the breath required for the cry or in the sopor and coma of brain-disease the child is only too quiet.

The absence of tears, after the age of three or four months, during th crying of the child, is construed as a bad sign. Something of the same kin is often seen in adults: "the dying weep not."

DROPSY.

Dropsical swellings are not very different in children from what the are in adults. General anasarca of renal origin is relatively common at thi age, partly on account of the frequency of scarlatinal dropsy, and partly because parenchymatous nephritis specially affects young subjects. Wheneve a child appears with suddenly-developed anasarca we are bound to think of scarlet fever; we look for signs of desquamation on the fingers and elsewhere, and we inquire for a history of sore throat, red rash, etc. Any mistake in missing the diagnosis of scarlet fever in such cases may be disastrous as regards other children. Once in a while there is a case of genuine renal or scarlatinal dropsy without a trace of albumen in the urine.

In scarlatinal dropsy, and indeed in other forms of acute or subacute nephritis in children, even it may be without dropsy, we must always be on our guard lest the supervention of acute pleuritic or pericardial effusion, or the occurrence of uræmic convulsions, should come on under circumstances which might aggravate the condition or give rise to painful reflections of these being caused by indiscretions.

The dropsy of heart-disease does not differ in the young, in any notable manner, from what we see in adults. Hepatic dropsy and tubercular peritonitis have been already mentioned as giving rise to dropsies limited to the abdomen.

Œdema of the feet or of the eyelids in young children is not uncommon as a result of anæmia with perhaps feeble circulation but without renal or cardiac disease: it may occur in cases of diarrhœa or other chronic illnesses. We see a more peculiar form of the same thing in a swollen state of the hands and feet, the swelling being so tense as not to pit on pressure. A somewhat similar condition, with hardness and swelling more extensively distributed, has been described in newly-born or very young children, under various names ("induration of the cellular tissue," "sclérème," "hide-bound"): it may be complicated by a low temperature and by great debility, and is indeed a most dangerous condition.

As in adults, obstruction to the circulation in the chest may give rise to œdema of the upper part of the body and arms: in children tumors in the mediastinum, giving rise to such symptoms, are usually of glandular nature.

Subcutaneous emphysema, from the rupture of air-vesicles in whooping-cough or other diseases, may seem at the first glance to resemble œdema; but the crackling sound and sensation on testing the parts for pitting, and the resonant percussion, prevent mistakes.

GENERAL PAINS, AND PAINS IN THE LIMBS.

Pains in the head, back, chest, or abdomen, when they can be localized by the child's language or signs, serve, of course, to guide our examination. At times they are present there, but undescribed, and the only indication we get is from the expression of pain in the face, or from the cry, and from the apparent aggravation on moving or pressing certain parts. Elsewhere, persistent crying has already been spoken of as often due to earache.

Sometimes the discomfort, as in adults, is too general to be defined, although extreme enough. In rickety cases the tenderness is in the bones and muscles and is developed on handling the child or disturbing his position. In cerebral meningitis, and more especially in cerebro-spinal meningitis, there is great general hyperæsthesia, with special pains on moving the neck and limbs. In one case the writer made an erroneous diagnosis of rheumatism in a boy with incipient cerebral meningitis, being misled by the pains in the limbs, and also no doubt by the fact that this child had previously suffered from rheumatism. In cerebro-spinal meningitis the resemblance to rheumatism is greater, and cases of this alarming disease are sometimes put down as rheumatic ailments of no great severity, owing to the absen of any swelling in the joints.

Rheumatism in childhood is at times rather difficult of recognition, the articular affection is only slight, and perhaps contemptuously spoken as "growing pains," although such trivial attacks are often associated wi endocarditis leading to permanent mischief in the heart. At times the pai are almost limited to the feet or heels, with some stiffness in the muscle In other cases, of course, acute articular rheumatism may be plain enoug but in children under six or seven years it is not common to have it in glaring form. As in adults, pains more or less distinctly rheumatic m concur with an eruption of purpuric spots.

Another disease sometimes erroneously called rheumatism is acute per ostitis, or "necrosial fever." This affection is often thought of, in t early stages, as typhoid fever, when the pains in the limb are trifling, a is often supposed to be rheumatism when they are more pronounced: t disease frequently advances to suppuration before it is recognized as per ostitis at all. The tibia is the commonest bone affected, but others are al attacked.

Over the tibia the red spots of erythema nodosum may give rise to mu pain with feverishness: they often occur in rheumatic subjects.

The pains in the limbs in the early stage of infantile paralysis often le to a misconception of the nature of the attack, and affections of the join may be suspected, and especially the diagnosis of hip-joint disease is som times made, with, it may be, disastrous results in the subsequent treatmen But joint-affections also occur only too frequently in children, with pa and swelling; scrofulous disease in particular must always be borne mind. Although it is a rare affection, hæmophilia, or the hemorrhag diathesis, frequently gives rise to joint-affections in children, with painf swellings, due probably to effused blood.

Glandular swellings are also sources of pain, especially in the groin an in the neck; in the latter situation the pains arising from them may sim late rheumatic affections of the muscles, or they may give rise to distortion resembling torticollis; from the violent shooting pains, going up to th head, grave cerebral mischief may sometimes be apprehended. The glandula swellings are not always perfectly easily felt, but when enlarged and tend glands are detected the explanation of the pains and feverishness may be a once obtained in otherwise very alarming-looking conditions.

FAMILY HISTORY—HEREDITY.

The family history is of capital importance in the study of sick chil dren, for it is often by the known tendencies to disease in the individua and in the family that we interpret the meaning of existing symptoms.

The best way is to ascertain all the facts known to our informants re garding the ages of the parents and of the brothers and sisters, if they ar alive, their state of health, and their liability, past or present, to any ail ments. If there are deaths, we ascertain, *seriatim*, the age at death and the cause of death; we often require also to get particulars as to the duration of the illness and the leading symptoms, so as to compare these with the name assigned to the disease. In cases of suspected syphilis we may have much light thrown on the nature of the illness by a history of repeated abortions in the early months of pregnancy, then of still-births at the full time, and then (as the intensity of the disease seems to lessen) of live children born with signs of congenital syphilis. After all such information is obtained, we have often to make inquiry as to other relatives, particularly the grandparents and the uncles and aunts on both sides. When we have definite suspicions as to the nature of the illness, as in cases of tubercular disease, rheumatism, cancer, diabetes, etc., we inquire specially as to these, giving a variety of names, so as to help our informants, and asking if any such cases occurred among the relatives named. At other times we gain our point best by asking who was *the nearest relative* affected with consumption, for example: if none are alleged, we then say that most people have had some relatives thus affected, as cousins, for instance; and if any such are admitted, we explore the history of the relatives again on the side under suspicion, and after any discovery we should always ask if there were any others affected, before abandoning the quest. A knowledge of human nature leads us to inquire for such weak points in the mother's family history, questioning, not herself, but rather the father of the child, or, perhaps better still, some of his sisters, and we prefer to do so out of the hearing of the mother and her friends; or, equally, we inquire of the maternal relatives of the child for weak points in the father's family history: having got a clue, we may then, if necessary, push our inquiries among the relatives concerned, always, of course, trying to avoid words, such as cancer or scrofula, which are likely to be offensive, but speaking of "decline with lung-complaints," "swellings in joints," "tumors," etc., as if they were very commonplace ailments. In this way we can often gain information of much value for a due estimate of the case.

Tubercular tendencies are so important and manifold in the diseases of children that we have to make special search for them, including all sorts and forms we can think of, under various popular names. The influence of a mother's phthisis seems more potent than a father's in transmitting such an affection.

In the case of cancer, with which probably other malignant tumors should be grouped for this purpose, we must remember that, although affecting at times even very young children, cancer is notably a disease of advanced life, and that children may inherit the tendency from parents in whom, or in whose brothers and sisters, it may not yet have had time to appear, although it may do so when they become older.

The remarkable phenomenon of atavism must be remembered: large numbers of a family may be swept off by a disease, notably by tubercular disease, although no cases may have occurred in the parents or in their brothers or sisters; the history of the grandparents and of grand-uncles and grand-aunts may come in to clear up the mystery.

Allied to atavism, although distinct from it, is the peculiarity found in the transmission of hæmophilia (or the hereditary hemorrhagic diathesis) by the mother to her children, although not only she but all the female members of her family escape the disease in their own persons. This disease is transmitted always in the female line, but only to male descendants. Peculiarities of this kind in the phenomena of heredity probably account for some of the anomalies of transmission of other diseases, although they may not yet have been worked out as in the case of hæmophilia, which is a favorable disease for the purpose, inasmuch as it is absolutely rare and yet a very striking affection.

In the case of the so-called hereditary ataxia (Friedreich's disease) we have the same nervous affection occurring in various members of a family, although the fact of actual transmission is not made out. It is very doubtful, therefore, if the word "hereditary" should be used, although in view of the facts known as to hæmophilia we can understand that a disease may occur in a family notwithstanding that it is necessarily absent both in the parents and in the grandparents.

The combination of the constitutions of the two parents may determine peculiarities unknown to either of them. The injurious influence of consanguineous marriages may also be explained in some such way, the influences for evil in a family being intensified, instead of diluted, by the marriages of near kin. Further, when both parents, although of different families, are consumptive or rheumatic or neurotic, the danger of transmission is no doubt much greater, if for no other reason than that there is thus a double chance of transmission, or a double portion of the same inheritance.

In rickets the disease has often the *appearance* of heredity, from several children being affected in the same family, and it is notable that the later children in certain families seem especially prone to this affection. The explanation is probably not to be sought in heredity, except in so far as the mother of a large family in poor circumstances is liable to have had her health run down by work, anxiety, and child-bearing; but the children in such a family are of course all apt to be exposed to similar unhealthy surroundings, and with the increase in their number the mother is less likely to be able to take them out in the open air or to attend to them in the special manner in which she could when there were only one or two altogether in the family.

Pseudo-hypertrophic muscular paralysis is notably a family disease: although not traceable in the parents of the affected children, it may show itself, at times, in the uncles as well as in the brothers of the patients.

The tendency to transmission of a disease to children born after the parents have had the affection themselves seems to be more potent than in cases where the children were born before the parents were so affected. In the case of syphilis in a father we can see at a glance that it is only after the parent has had the disease that such can be transmitted: all the earlier children are quite unaffected. We can even understand that in the case of a mother actually affected with advanced consumption during her pregnancy the child thereafter born is more likely to be affected than those who were born before the mother's health had broken down. But, although it is not so intelligible, it would seem to be made out that in the case of gout, of rheumatism, and probably some other affections, the parents, although capable of transmitting these diseases to their offspring before they have had overt manifestations of them in their own persons, are more likely to transmit these diseases, or to transmit them in greater intensity, to those born after they themselves had been affected.

Transmission of disease or diseased tendencies under different forms is a subject of great importance, but as yet not fully worked out. We can easily understand that such disease-manifestations as hip-joint disease, tubercle in the brain, and tabes mesenterica may all be reduced to one common inheritance, and that these occurring in the brothers or sisters of a patient, or in his uncles or aunts, may throw light on cases of mischief in the pleura, pericardium, or lung, or on many other tubercular or scrofulous affections in other members of the same family stock.

Rheumatism, growing pains, chorea, and heart-disease form another group; one child may have all four, but in a rheumatic family one child may have but one of these forms, and a second may have another, or perhaps other two, out of the list, as his portion of the common morbid tendency.

Some would remove chorea from this list and put it among the neurotic group of hereditary ills, classing it rather with hysteria, epilepsy, and insanity. In any case, there is such a neurotic group, and probably a liability to bad or generalized neuralgia, bad headaches, and general excitability should be included as the result of the inheritance of an unstable nervous system, which, however, is quite compatible with great quickness of intellect and general ability; for

> "Great wits are sure to madness near allied,
> And thin partitions do their bounds divide."

These nervous diseases seem to replace one another in the history of the individual at different periods of his life or in different members of his family. It is extremely probable that the inheritance of a bad nervous system predisposes not merely to alcoholism but also to criminal courses of life, and that children of drunkards and of the criminal classes come into the world biassed towards evil courses which may take the place in them of more definitely recognized diseases.

Rheumatism has been already referred to, but it has also other affinities. The rheumatic and gouty inheritance may show itself in the children being liable to psoriasis and eczema; to uric acid gravel, and, it may be, calculus;

or to asthma and to asthmatic bronchitis. Uric acid calculous disease known to be often hereditary without perhaps any connection with oth diseases being ascertained.

Gout is practically unknown in childhood in its articular form; but w may see the little pearly gouty deposits in the ears; and in addition some of the ailments mentioned in the last paragraph we may see granul kidney: in any case, this granular (or so-called gouty) kidney may appe as an hereditary disease in certain families, declaring itself even in earl life.

Saccharine diabetes in children, although rare, can often be traced hereditary: as in the adult, we can sometimes trace relationships betwe diabetes and phthisis pulmonalis or other tubercular disease.

Malformations of all kinds can often be traced as occurring in differe generations of the same family, and even trifling deviations from the norm formation are likewise hereditary. Equally so, as is matter of univers comment, are the family peculiarities of build or feature: indeed, it probably in extension of this inherited transmission to the internal orga and the minute tissues that we are to seek the explanation of a liability many of the inherited diseases with which we are acquainted.

Intermediate between congenital malformations and inherited diseas we may place deaf-mutism and congenital color-blindness, although t exact structural defect may evade our recognition. It would be somethi of an absurdity to speak of the use of concave or convex spectacles bei transmitted in certain families, but the errors in refraction requiring su corrections are undoubtedly often hereditary, the defect in vision dependi on inherited structural peculiarities in the eye.

It would seem, however, as if not only structural peculiarities and t tendencies to, or the beginning of, chronic disease were transmitted fro generation to generation, but that special tendencies to acute disorders a also inherited.

It might be disputed whether a special tendency to catarrh should classed under this heading, or whether it should be referred to structur peculiarities in the mucous membranes; but, in any case, catarrhal tende cies are undoubtedly transmitted; these may lead to wheezing conditions i the chest, or to nasal catarrhs favoring, for example, affections of the tyn panum from this cause, with its attendant deafness, so often found to run i families.

But, further, special families seem specially liable to attacks of the acu specific fevers, and when they do appear there is apt to be a special severit in the disease. We may thus trace a severe type of diphtheria or enter fever, with perhaps grave intestinal hemorrhages, as occurring in differe members or generations of the same family, at such intervals of time or o geographical distance as to preclude the idea of any common infection, an yet with such frequency and severity as to make the idea of special liabilit irresistible.

PECULIARITIES IN THE DISORDERS OF THE VARIOUS SYSTEMS IN CHILDHOOD.

In proceeding now to make remarks on some diagnostic points in the various systems, as an aid to the study of sick children, it is evident that in a chapter like this these remarks must be fragmentary, and any attempt at systematic completeness must be abandoned. In selecting certain subjects for comment, it may appear to many readers that these are not the points specially requiring discussion, and that they have been selected to the exclusion of others more important. The writer must make such a selection as seems to him likely to be most practically useful in view of the scope of the present work and of his own experience of what it has been important for himself to learn or to teach.

In proceeding with the examination of a sick child for the purposes of diagnosis, it is usually well, as in the case of the adult, to follow the various physiological systems, although, as already explained, there must be a great readiness to depart from any fixed method according to the exigencies of the case.

NERVOUS SYSTEM.

Among the disorders of the nervous system we have paralysis in various forms, but some varieties common in the adult are rare at early ages. Thus, hemiplegia from ordinary hemorrhages or degenerations such as occur in advancing years is scarcely known. But hemiplegia does occur and is sometimes suspected to exist when the disorder is really due to something else. Thus, in chorea, really a convulsive disorder, we usually have more or less loss of power, and when the affection is unilateral the loss of power is unilateral also; when by some chance the twitchings are not very plain, or when, as happens rarely but still occasionally, the loss of power *precedes* the twitchings, and the child is brought complaining of a somewhat sudden or of a more gradual loss of power in one arm or in one side, we may by careful examination be able to make a diagnosis of chorea, and so remove much of the anxiety felt at such an occurrence.

One-sided paralysis in children is often dependent on cerebral tumors, usually tubercular; but the presence of staggering and more general weakness often takes away from the precision of the hemiplegia. In children with one-sided paralysis dating from birth, we must always think of the possibility of some hemorrhage or other lesion from injury to the head at parturition. This is apt to be followed by atrophy of the brain on the side affected, and by a spastic state of the paralyzed side: a bilateral lesion may give at times a bilateral hemiplegia, if such a term may be used, with a most remarkable shuffling gait. Paralysis of one arm or of one leg (monoplegia) from infantile paralysis may occasionally suggest the idea of hemiplegia, and in particular, if the two limbs on the same side are implicated in the attack, the idea of a cerebral lesion may be suggested, although the disease is known now to be of spinal origin: all the more likely is such a mistake to arise if convulsions have ushered in the attack. Hemiplegia from thron bosis or perhaps embolism of the cerebral vessels occurs at times in co nection with the specific fevers and other causes, sometimes with unilater convulsions, and occasionally associated with aphasia. Meningitis ar abscess of the brain occasionally give rise to one-sided paralysis, but usual the case is too complicated to be spoken of as hemiplegia. In whoopin cough we may have, although rarely, small hemorrhagic lesions in the brai due probably to the paroxysmal fits of coughing; with these also we ma have aphasia as well as hemiplegia. Hemorrhage on the surface of t brain or into its membranes is more common than marked hemorrhag lesions in the brain-substance: in children with meningeal hemorrhage, w survive the shock, there may be paralysis of one side, and the post-morte examination may show the presence of false membranes.

Paralysis of a limb, or of part of one side, is not very uncommon as sign of cortical lesions in the brain, often associated with convulsions limit to the same part.

The connection of chorea and hemiplegia has already been mentione but after genuine hemiplegia we may have "post-hemiplegic chorea," as is termed (although there is really no relationship to the well-known disea called chorea), with very curious spasmodic movements, of slow evolutio resembling, if not identical with, the affection termed "athetosis." In t paralyzed limbs of hemiplegic children there is often a tremulous or shak state especially noticeable when the arm is used: such cases are often due cerebral tumor.

Paraplegia in children is usually dependent on caries of the vertebr which is relatively common in early life: its features are not specially di ferent from those seen in the adult. Of course spina bifida may give ri to a form of paraplegia special to children. Diphtheritic paralysis is rel tively common in childhood; although usually affecting the palate and t accommodation of the eye more notably, it may assume the paraplegic typ or the whole muscular system of the body may seem implicated. Oth specific fevers are also occasionally followed by paraplegia. Spinal myelit and meningitis may affect children, as well as adults, from obscure cause without impressing any special peculiarity on the case from their age.

Epidemic cerebro-spinal meningitis is not uncommon in children whe the disease is present in a community. The most striking features, in add tion to headache, vomiting, and fever, are the severe generalized pains in th back and limbs, with great suffering on handling the child, the presence o retraction of the head and neck, which is often extreme, the occurrence o herpetic or purpuric eruptions on the body, and the implication of the ey and ear. The pyrexia is more intense and persistent than in the ordinar tubercular cerebral meningitis. Recovery may take place from a conditio which seemed quite hopeless, after the lapse of a few weeks; deafness o some other remnant of the disease may be permanent.

Infantile paralysis, with its pains, feverishness, sudden loss of powe and rapidly-developed atrophy and coldness of the limbs, must be studied in detail elsewhere. The localization of the paralysis, when not absolute and extreme, is different in the upper and lower limbs: in the arm it is usually the upper part which is badly paralyzed, the muscles of the forearm and fingers regaining in time considerable power; in the leg it is especially the muscles below the knee which are weak and atrophied, those of the thigh being often pretty sound. As is well known, the sensation is not affected in infantile paralysis, and the sphincters almost never. Pain in the early stage of this affection often leads to erroneous ideas suggesting hip-joint disease and various other painful disorders; very often the true diagnosis is not suspected till the paralysis is detected when the child is supposed to have recovered from the acute disease.

A form of paralysis limited to children, or at least always beginning in early life, is the pseudo-hypertrophic muscular paralysis described by Duchenne. It tends to occur in certain families, although really a rare disease. The child begins to fall easily, and his companions often amuse themselves by knocking him over, as the process of rising is difficult and in a sense comical. The abdomen stands out, from the presence of a saddle-back curvature in the spine, and the child's manner of lifting the feet suggests a resemblance to the walking of a turkey. The limbs, instead of being wasted, appear as if hypertrophied, in the earlier stages at least, and the calves of the legs are especially prominent: the hypertrophy, however, is spurious, and the limbs are really weak.

Aphasia has been found again and again in children under circumstances pointing to a lesion of the brain in the usual situation, but it is far from being so common as in adults. At times, however, it has also been seen with left instead of right hemiplegia. Occasionally there is aphasia of a temporary character after an attack of enteric fever.

Affections of the speech and other symptoms closely resembling those found in bulbar paralysis usually prove to be due, in children, to tumors involving the floor of the fourth ventricle, as the regular progressive labio-glosso-laryngeal paralysis does not occur in early life.

Paralysis of the cranial nerves is common in childhood. The portio dura of the seventh pair is often involved in ear-disease at this age. In young children this nerve may be affected from acute suppurative inflammation in the middle ear, without destruction of the nerve, as proved by the subsequent recovery. The other causes of peripheral facial paralysis are also operative in childhood, but do not call for notice here: facial paralysis of central origin occasionally dates from an obscure affection in early infancy pointing to cerebral disorder; in this last case the paralysis, although of old date, does not prevent the muscles from responding to the faradic current perfectly.

Paralytic affections of the ocular muscles, with squinting, immobility of the eyeball, lateral deviation, and nystagmus, are very common in childhood in connection with cerebral tumors: these affections must be studied

and worked out in detail, just as in adults, so far as the child's conditi
and intelligence render this possible. In childhood the occurrence of squi
ing may readily be brought about by any acute illness, so as to occur at
particular time, although from errors in the refraction of the eye its appe
ance sooner or later might be inevitable: in such cases, of course, the squi
is not paralytic.

Affections of the optic nerve and retina are likewise of frequent occu
rence, and the diagnostic points in connection with the ophthalmosco
examination are very valuable; but a mere passing allusion is all that
here required, as the subject requires full consideration elsewhere.

Marked intolerance of light, with spasm of the eyelids and lachrym
tion, always suggests the idea of keratitis; and we may have photopho
from this cause without the lachrymation. In various brain-affections, a
specially in meningitis, the child often shuns the light, without any lo
affection of the eyes, the headache being apparently intensified by any brig
light. A similar objection to the light may occur in headaches from oth
causes, although seldom to the same extent.

The state of the pupil has often to be carefully examined in childre
During healthy sleep the eyeball is drawn upward and inward, but if t
lid be raised the pupil is found contracted: if the child awakes during t
examination, the pupil dilates with awakening, but contracts immediate
from its exposure to the light. Immobility of the pupil on exposure
light may be taken as an index of blindness if the pupil is of normal siz
in testing critically, the eyes should be exposed separately to light and shad
If the pupil happens to be either much dilated or much contracted, or und
the influence of atropine, this test is not of much use; when response
light is obtained it shows a certain sensitiveness of the optic nerve.

Enlargement of both pupils is common in cerebral meningitis with effi
sion into the ventricles, but, as in the adult, some of the most grave cerebr
lesions produce contraction. Inequality of the pupils is also common
meningitis, but it often varies much as to its degree or even its presenc
and also as to the side on which the dilatation occurs from time to tim
Enlargement of one pupil is common in paralysis of the third nerve, usuall
with other signs of this nerve's implication. Inequality from paralysis
the sympathetic shows itself by contraction on the affected side, or rather
a want of dilatation on shading: it may occur in spinal caries involving t
cervical region, or from other implications of the sympathetic in the neck.

Oscillation of the pupil under the stimulus of light, so that it contract
and dilates while the light is held steadily before the eye, is not uncommo
in children with meningitis.

Rhythmical oscillation of the pupil has been noticed in the deep stert
rous breathing of coma, and in the deep breaths of Cheyne-Stokes breathin
from cerebral lesion; the pupil dilates with inspiration and contracts wit
expiration; in Cheyne-Stokes breathing the pupil contracts during the perio
of apnœa.

Enlargement of the pupil from atropine applied locally usually, of course, affects only one side, but during its internal administration, if pushed, both pupils are enlarged and somewhat imperfect in their response to light. In opium narcosis contraction of the pupil is a valuable guide. The pupils in children are usually somewhat larger than in adults, and the extent and readiness of their response to light more notable.

Tubercular meningitis is one of the most alarming diseases of childhood, and in its early stage one of the most difficult for diagnosis. In the section on digestive disorders reference will be made to the difficulty often experienced in estimating the significance of persistent vomiting. In the remarks on respiratory disorders a short reference will be made to the confusion of this disease with pneumonia. Other diseases, and especially otitis, may also be confused with it,—for which the reader must refer to special sections of this work. But in this place some allusion may be made to the frequent difficulty experienced in deciding whether a case is one of meningitis or of enteric fever. In both we have fever; in both, oppression or excitement or, it may be, coma; and in both we may have a congested state of the lungs. The points which aid us are (1) that in enteric fever sufficiently severe to cause cerebral symptoms the temperature is usually very feverish, whereas in tubercular meningitis, after it produces marked cerebral symptoms, the temperature is usually moderate. (2) In tubercular meningitis the child has *usually* been falling off in condition before the acute symptoms come on. (3) The state of the abdomen and bowels may guide us, not merely as to looseness, although this is so extremely uncommon in meningitis as to count for much, but more particularly as to tumidity of the abdomen; this is rarely absent entirely in enteric fever, while in meningitis the abdomen is seldom full, often flat, and sometimes retracted. (4) The family history, and (5) the mode of onset, may also help us.

Another condition sometimes confused with meningitis is "hydrencephaloid disease" due to exhausting illnesses, and especially to diarrhœa. In both diseases the child may lie in the same apathetic condition, with little or no fever. The history of diarrhœa with the vomiting may often guide us; for, as already said, this is a rare complication in meningitis. The collapsed fontanel in young children may also often guide us, for it is in them that mistakes are most likely to occur. Occasionally we try by the effect of stimulants, or even of opium in small doses, to obtain confirmation of our diagnosis, when we think we are dealing with the less serious disease.

Convulsive diseases are of special importance in childhood, for they occur not merely as complications of grave disorders of the brain or from uræmia, as in the adult, but also as manifestations of general disorder and disturbance. Thus, in the acute fevers, or in pneumonia, we may have convulsions ushering in the illness or occurring during its progress; as Dr. West puts it, "in a large proportion of cases convulsions in the infant answer to delirium in the adult,"—a most suggestive view, taken in connection with the demonstration of motor centres in the cerebral convolutions. But in early life, errors in diet or disorders in the digestion which in adults might be called trivial may give rise not only to diarrhœa or vomiting but to violent convulsions. No doubt some special susceptibility may exist in the nervous system to favor such an occurrence in some children or families rather than in others; and in connection with rickets this predisposition undoubtedly exists in many, so that trivial disturbances, not always easy to trace, may reveal themselves by convulsive attacks. In connection with violent spasm of the glottis,—itself a convulsion,—whether in whooping-cough or in laryngismus stridulus, we often see general convulsions supervening. In cases of prolonged diarrhœa or other forms of exhausting disease, we may have convulsions apparently in the same way as from loss of blood. After scarlet fever which may have been so slight as to pass unrecognized or at least to be little regarded, the renal complication may also have been little if at all thought of, till sudden uræmic convulsions may startle all concerned: those physicians who have been once surprised thus are usually very careful to see that nothing is wanting in the care of scarlatina convalescents.

Convulsive twitching movements of the face and limbs, with erratic behavior of the voluntary muscles when called into action, are characteristic of chorea. This is essentially a disease of childhood; it may, however, appear in those who have attained puberty in both sexes, although very uncommon in young men. It is less rare in girls of this age, but usually then occurs as a relapse; it is well known, also, that it may complicate pregnancy, usually as a recurrence. Not a few diseases, however, termed chorea are scarcely entitled to be called so: the post-hemiplegic chorea already referred to (which is not limited to children) clearly points to grave mischief in the brain, and some other of the chronic forms of chorea, or of very localized variety of chorea, no doubt point in the same direction. Occasionally a tremulous jerky state of the arms may simulate chorea pretty closely, although really constituting an early symptom of cerebral tumor; and in certain cases of congenital mental defect the excited jerky movements of the body and limbs may suggest the idea of chorea to the on-looker. Whenever, indeed, chorea departs from its known characteristics as regards age, duration, localization, and concurrent symptoms, we must always suspect that something worse may be actually present. Some varieties of the so-called electric choreas are probably momentary epileptic attacks.

The diagnosis of epilepsy in children is often raised in connection with the occurrence of convulsions, a subject of great importance, which must be fully discussed elsewhere. The early discrimination is often impossible. The wisest course is usually, in all cases of doubt, to let our treatment be guided by the graver view, and to let our prognosis, as expressed, lean always to the more hopeful side. The course of the case alone can decide.

A remarkable form of convulsion limited to children is that known as eclampsia nutans. These "nodding convulsions" usually consist in the rapid bobbing of the head up and down or back and forward. Occasionally they are more extensive, with a bending or bowing of the whole body, so as to merit the name of "salaam convulsions." The disease is probably closely allied to epilepsy; and, like epileptiform seizures of the more ordinary kind, these nodding fits are probably at times due to the presence of tubercles in the brain.

The curious spasms of the fingers and toes, or of the wrists and feet, known as "tetany," may be regarded as almost special to children, although they occur in others also, and especially in nursing women. Occasionally a graver and more continuous form, resembling tetanus more closely, may occur in childhood. Slighter forms, again, of these "carpo-pedal" spasms are often detected as manifestations of partial convulsions, or as the precursors or remnants of general eclampsia. In connection with wasting diarrhœa a chronic spasm of these parts is often associated with a swollen state of the backs of the hands and feet, due apparently to anæmia: in such cases the nervous affection may pass off as the general state improves, without any generalization of the spasms.

Hysteria is by no means excluded by early age from our diagnosis: it may occur even in young boys. It would be useless to give illustrations of the various forms it may assume in children, one of the most striking being the paralytic. Closely allied to hysteria in various ways, although never to be regarded as synonymous with it, is simulation,—pretended deafness, dumbness, twitchings, paralysis, etc. Even the remarkable phenomena of hystero-epilepsy may occur in children; the writer has seen typical attacks of this in boys; and in one boy it was associated with the most distinct "crucifixion attitude" as described and figured by Charcot.

Of mental disorders, idiocy and imbecility are the most important in childhood. These defects cover a multitude of special ailments,—inability to speak, to walk, etc. Frequently the mother brings her child to have the frænum linguæ cut (as she considers the inability to speak depends on the child's being "tongue-tied"), without any notion, so far as we can gather, that there is any mental defect at all.

Violent maniacal fits after epileptic attacks, or replacing them, it may be, are likewise well known in children. A certain passionate violence in children sometimes goes to such a length as to suggest hysterical mania or some other instability of the mental faculties: in some cases similar attacks are connected with the uric acid diathesis.

In this connection, but much short of any serious mental aberration, may be classed the terrible "night terrors" of young children, arising from the vividness of their imagination, coming on during night, probably in connection with dreams. Somnambulism also in various forms and degrees occurs in children, or excessive talking in bed when asleep, or it may be with the eyes open; in this condition the child may be able to answer, in a kind of way, various questions directed to him. These conditions of sleep-walking and sleep-talking are often dependent on, or at least aggravated by, undue application to studies or continuous anxiety in connection with them.

Headaches in children are discussed in a special chapter.

PULMONARY AND CARDIAC SYSTEM.

One of the most striking symptoms in disorders of the respiratory system in childhood consists in the appearance of rapid or labored breathing, with excited action of the alæ of the nose, so that when we see this, with heat of skin, we can scarcely go wrong in alleging a respiratory disease or complication. Another very striking feature of respiratory distress in children at the breast consists in their giving over sucking or in their readily abandoning the attempt, although perhaps eager to try; they have not breath enough to suck from the breast, and may even be unable to suck from a bottle, although this is a less taxing effort in such conditions. When this inability is reported, we always think of pneumonia or severe bronchitis.

The violent efforts at inspiration occurring in croup, or other obstructive diseases in the upper passages, have already been referred to in connection with the appearance of the chest. But the presence of stridor in the respiration, with a curious hoarse or squeaking sound, and the hoarse yet clanging sound in the cough, with at times a similar hoarseness in the voice, constitute points of equal importance in the diagnosis. Some of these croupy attacks, although alarming to look at, are practically devoid of danger, the affection being only a catarrhal laryngitis aggravated by spasm: in cases with deposits in the larynx or trachea, on the other hand, the danger is always great; the alarming dyspnœa is in such children more constant,—never quite relaxing even for a moment,—although in them also the element of spasm is clearly present, aggravating the permanent obstruction. The throat should always be examined for diphtheritic patches; but there is often laryngeal diphtheria without any affection of the fauces.

Occasionally retro-pharyngeal abscess gives rise to symptoms somewhat similar to croup, and so the case may be misunderstood. Spasm of the glottis from nervous causes or from foreign bodies in the windpipe may also do so.

Sometimes very rapidly increasing pleural effusions—especially in scarlatinal nephritis—may induce the most awful efforts at respiration, resembling the paroxysms of asthma rather than croup. The sniffling noises in the nose with the respiration, from congenital syphilis, constitute a well-known sign of much importance.

The absence of sputum in children with disease of the respiratory organs is habitual: in chronic pulmonary phthisis with excavation (usually in children over five) we may, however, have the well-known nummular and globular sputa. Even cough is often absent or obscured in many cases. A peculiar squeaky cough is heard sometimes in bed, pleuritic accumulations threatening suffocation. In empyema children sometimes spit up the pus from the pleura with a favorable result. The aspect of children as regards flushing and lividity has been mentioned already. The decubitus is similar to that in adults, and has similar variations, or is even more varied, from the restlessness of youth; in bad pleural effusions the child lies on the affected side.

The rhythm of the breathing is sometimes very irregular in children. Irregular or sighing respiration is frequent in cerebral affections, especially in meningitis. This is usually characterized by a few slow or shallow breaths, almost imperceptible, followed by a deep inspiration. At times the implication of the breathing is the terminal phase of a case of cerebral tumor, the breathing stopping while the pulse is good; it may even be possible to re-establish the breathing for a time in such cases by artificial means. A certain slowness of the respiration is very common in cerebral cases. Occasionally this altered rhythm becomes "regular in its irregularity," the ascending and descending series of respirations, with a period of apnœa, described as "Cheyne-Stokes respiration," being perfectly marked in cases with gross cerebral lesions: in the opinion of the writer there is no difference in kind, but only in degree, between the perfect Cheyne-Stokes rhythm and the irregular or sighing or cerebral respiration so frequent in meningitis. But this same irregular respiration may occur in cases not primarily of a cerebral nature, and it is frequent in grave cases of enteric fever with cerebral symptoms: the writer has seen the most perfect Cheyne-Stokes respiration in an infant overwhelmed with the poison of scarlet fever.

The irregular breathing of opium narcosis, perhaps from an overdose of medicine, must likewise be mentioned: it resembles the cerebral breathing just referred to, but is more characterized by intermissions in the breath than by irregularity or by any definitely altered rhythm.

In chorea we often see a very marked irregularity in the breathing both when the child is lying quietly and when it is trying to speak or swallow, the management of the breathing, as regards time, being so far out of control as to prove troublesome in these actions.

In rickets we have often a very great increase in the rate of respiration, so that it may run up to fifty or sixty in the minute, and this not only in connection with catarrhs and slight pneumonic attacks, but apparently as the normal condition of the rickety child's respiration.

In auscultation and percussion we have the same general facts as in adults. Chinking percussion and the "bruit skodique" are relatively common in childhood during advancing and receding pneumonias and pleurisies, and the student must beware of making a diagnosis of cavity from the "cracked-pot sound," in the case of an infant, unless supported by other strong evidence.

Phthisical disease of the lung is much commoner in early life than it was formerly supposed to be: we must not expect, however, the same great liability of the very apex of the lung to be involved as in adults: this and the implication of both sides in the consolidation help to make the diagnosis more difficult, and we have to rely much on the general aspect, the course of the case, and the family history.

"Bronchial phthisis" is often suspected in the case of children with a suspicious appearance and history of phthisis when auscultation gives little sign of pulmonary softening. We may have tubular breathing especially between the scapulæ; dulness on percussion there and at the upper part of the sternum; and perhaps loud fits of coughing, with almost a crowing inspiration, resembling pertussis. Occasionally in such cases cheesy fetid masses are expectorated.

Bronchitis is seldom difficult of recognition, from the presence of wheezing, snoring, or moist râles, or of all kinds mixed up together. The very high pitched wheezing sounds suggest, of course, the finest tubules as implicated.

Pneumonia, however, is often very difficult of recognition. In the lobar form this arises from the physical signs frequently being late in appearing, so that, although the disease may be suspected and careful watch kept on the chest, day after day may pass without physical signs, and thus the violent fever, delirium, or other forms of nervous excitement may lead to the suspicion of cerebral inflammation,—particularly if the child passes by and by into a kind of comatose state. The physician, now thrown off his guard, may have given over the exploration of the chest at the very time physical signs could be made out: and when hope is almost given up, in view of meningitis, we may see the child recovering, and perhaps a troublesome cough coming on for the first time. The clue to the case is often found in the very violence of the fever, and of the symptoms generally, at the onset; for with the ordinary meningitis pronounced cerebral symptoms usually coincide with comparatively moderate fever.

Cerebral excitement from pneumonia has been supposed to be specially common when the disease affects the upper lobe: in such cases the pneumonia is of the lobar or croupous form. It is of special importance to recall this situation of the disease, as experience in the adult leads us to search for pneumonia rather at the base. In children the localization of the disease in the upper lobe has not quite as much gravity, in indicating a tubercular origin, as in the adult.

In broncho-pneumonia (which may also simulate cerebral affections) the lobules are involved in the catarrhal process, and so the physical signs vary much in distinctness. If extensive, we may have the dulness, tubular breathing, etc., as plain as in the other form; but if the condensed patches are small and scattered, the physical evidence of their presence may be obscure, and the signs are often fluctuating, one day pretty clear, another scarcely recognizable; one day we may think the right lower back is the site of the disease, the next we may think the dulness and alteration in the breathing are in the left; one day the whole side may seem implicated, another only the base. The auscultatory signs vary much. Often we have tubular breathing more or less marked; sometimes there is rather feebleness of the breath-sounds. If either of these changes coincides with distinctly appreciable relative dulness in the back (however slight), fine moist râles, rapid or labored breathing, excited action of the nostrils, and high temperature, we may put the case down as a pneumonia in some form: having done so, we do not readily change our opinion although the physical signs may seem to become less amidst the persistent fever.

Judging from the signs just enumerated, we may think a broncho-pneumonia impending, or already begun, when the sequel shows that whooping-cough is the real disease; but the local conditions in the lung are probably closely allied to the other condition if much fever exists. Even in the course of a moderate case of whooping-cough the signs referred to may be all present, and may almost completely disappear, for a time, after a fit of coughing, with or without vomiting.

In childhood collapse of the lung plays an important part in the changes brought about in bronchitis and catarrhal pneumonia; but patches of collapse, sometimes of large extent, may occur without much concurrent inflammation, especially in whooping-cough: the signs are dulness on percussion, feeble respiration, partial immobility of the affected side, and by and by there may be a falling-in of the ribs, either permanently or only for a time.

With regard to special auscultatory signs in childhood, the name of "puerile breathing" will recall the fact that a very full and somewhat harsh inspiration is natural to children. The occurrence of tubular breathing in pleuritic effusions, especially at the back, instead of the feeble or suppressed respiration more often looked for, seems to be relatively more frequent in children than in adults, so that we are apt sometimes to make a diagnosis of consolidation of the lung when there is really a pretty large effusion in the pleural cavity.

In pleurisy we may frequently miss in the child the initial friction-sound: indeed, the diagnosis at this age has usually to be made on the ground of pain in the side with restriction in the breathing without any audible friction; in a day or so we may have our diagnosis confirmed by the presence of dulness on percussion, at the base behind, with feeble respiration and diminished vocal resonance. With the subsidence of the effusion we may have the friction audible for the first time.

In cardiac diagnosis we have the same phenomena as in adults: affections of the heart in children are very much commoner than was formerly supposed. We must remember the occasional occurrence of congenital malformations, with signs of stenosis of the pulmonary artery and other indications of defective development: there may or may not be concurrent cyanosis. We may practically exclude aneurismal disease from our diagnosis of cardiac disease, although dilatations of this kind have been seen at this age.

With regard to pericarditis, we may, of course, have it in acute rheumatism; in cases of chorea also, with or without distinctly rheumatic symptoms, pericarditis may supervene, always a most grave complication in this disease. In young subjects the tubercular form of pericarditis is relativel commoner than in adults. With the extension of pleuritic inflammatio so as to give rise to pleuro-pericardial friction or genuine pericarditis, w are often in doubt as to whether there may not be a tubercular basis fo the extensively distributed mischief. The course of the case alone ca decide. The pericarditis of Bright's disease must also be remembered.

A very special variety of pericarditis may be said to be limited to youn subjects, essentially of pyæmic origin, but developed in connection wit "acute phlegmonous periostitis:" this usually involves the tibia, but othe long bones may also suffer. In such cases pericarditis seems to mark th constitutional affection: it may persist for a long time, or it may be rapidl fatal. With the pericarditis we have often endocarditis also, and the disease in the valves gives rise to further dangers and complications, such a pyæmic emboli in the kidneys, etc.

In children, perhaps even more than in adults, rheumatic pericarditi may suddenly become highly dangerous from the excessive effusion, but th signs are not peculiar at this age.

DIGESTIVE SYSTEM.

The disorders in the digestive system are full of peculiarities in children and especially in infants, but just on this account they may be dealt wit slightly in this chapter, for in connection with weaning, artificial food, diarrhœa, etc., the reader will find all the matters of special importance enlarge on elsewhere. The undigested milk with curdy motions; the aspect of th fæces when, as sometimes, they are green when passed, or sometimes onl become green after exposure to the air; the influence of feeding in determining a motion, so that, as the nurses say, the milk seems to "run through' the child at once; the dreadful smell of the motions at times, and the controlling influence in this respect of boiling the milk; the tenacity of th curd as vomited by the infant,—the importance of these and of many othe such matters has to be learned by the student of pediatrics, but only a detailed discussion of the subject could be of any use, and this must be sough in other chapters.

The significance of vomiting, and especially of persistent vomiting, i the child, has even a wider range—wide as that is—than pertains to diseas of the digestive system itself. Vomiting is extremely common at the beginning of the acute fevers, and if carefully inquired for it will be foun that it is very usually present at the onset of scarlatina. Even with pneumonia it is very common, and with various other serious febrile illnesses It is, however, as the index of meningitis or other cerebral affections tha it is most anxiously considered by physicians when called to a case of persistent or very frequent vomiting. Again and again we try to explain i away as due to some digestive derangement, to the use of purgatives or t the want of them, to errors in diet or management, and the like, but ar forced to admit that these explanations are untenable and that we are dealing with the vomiting of incipient brain-disease. At other times we may have the greatest anxiety as to the significance of such vomiting, till the whole disturbance subsides without further mischief. From this it will be gathered that the writer knows of no special points by which cerebral vomiting can be discriminated from that of other origin. We aim at discovering if the vomiting had any obvious cause in the diet of the child, or if the tongue and state of the bowels point to disorder there; we try to make out if the vomiting was preceded by a feeling of nausea for some time before it occurred; for it is when the vomiting seems most "causeless," in these respects, that we suspect a cerebral cause. We further attach great importance to the concurrence of severe headache with the vomiting, and even to the concurrence of headache with any special turn of vomiting. The state of the temperature may also guide; for if suddenly rising very high we rather think of some impending fever, the elevation being, as a rule, very moderate in tubercular meningitis. The state of the bowels may guide, for if there is concurrent diarrhœa the chances of meningitis are very small, since this disease is usually attended with constipation, and, further, the looseness points to digestive disorders likely of themselves to cause vomiting. If constipation is present, this in meningitis can usually be overcome after a little trouble by physic; but if intractable, both constipation and vomiting may be due to intestinal obstruction of some kind; the state of the abdomen may guide, for if obstruction exists some distention is usually present, but in meningitis there is no distention, and indeed in the course of the case we may even have retraction. Very often we can only wait, holding possibilities in view and trying to steer a course as regards treatment which will be as free as possible from objection whatever the eventualities may be.

Intestinal obstruction has just been referred to as a cause of vomiting. While all forms may occur in childhood, we must remember that intussusception is relatively common in infants and children, and the presence of intestinal discharges of blood, the discovery of a tumor in the abdomen, or an examination of the rectum by the finger, may clear up cases which were doubtful till such assistance was obtained.

Another cause of intestinal obstruction in childhood which is apt to give rise to mistakes and confusion, from the rarity of this accident in adults, is the presence of tubercular peritonitis. Of course it is well enough known that in this condition there may be troublesome constipation; but at times we have, apparently from the agglutination of adjacent coils of intestine, a distinctly mechanical obstruction produced, revealing itself not only by general abdominal distention, but also by violent peristalsis of the coils of bowel above the obstruction, this being visible through the abdominal wall, as in many cases of stricture of the intestine.

An examination of the mouth reveals at times disorders so common in childhood as almost to be called peculiar,—viz., stomatitis in its various forms. We may have little blisters with clear fluid on the tongue and mucous membrane of the mouth, or the spots may be rather like litt superficial ulcers; in either case the salivation may be extreme, and there often great fetor of the breath; the tongue also is often foul and the whol digestive system deranged. A further form of stomatitis is the gangrenou (noma, cancrum oris), in which the edges of the mouth on one side becom black; the disease may also involve the gums, the teeth falling out, an great destruction of the parts often results. This destructive disease usuall follows measles or some general disease, or, at any rate, occurs in connectio with some great constitutional depression. A similar gangrenous diseas may attack the vulva in little girls.

Another form of stomatitis is the parasitic, formerly and often even no spoken of as aphthous: its popular name is "thrush," and its French nam "*muguet*." In this we see white patches on the tongue, on the inside of th cheeks, or on the throat. It is specially prone to occur in infants reare artificially, and assumes its greatest intensity in such when they are reduce to the last stage of wasting. Under the microscope the thallus and spore of the Oïdium albicans may be recognized. At times it presents, when o the fauces, a certain resemblance to diphtheria.

The examination of the throat has greater importance in children because we cannot always be guided by them to the seat of their pain. tonsillitis may at once explain the existence of a violent febrile disturbance or with a suspicious scarlet rash the appearance of the throat may at onc enable us to declare scarlatina; in other cases the presence of the whit patches of diphtheria may explain an otherwise unintelligible illness.

The discrimination of the various forms of sore throat is far from easy often, indeed, it is impossible. Redness with patches of exudation (so-calle ulcers) on the tonsil or on both tonsils, coincident with a high temperatur and a uniform scarlet rash, we must always regard as scarlatina; but whe the rash is measly rather than of uniform scarlet color, when the throat i a little sore and red, but not very red, and quite destitute of patches, an when the rash is very bright and abundant and the temperature only slightl elevated, we get into great difficulties. Sometimes the so-called Germa measles (rötheln, epidemic roseola) may be the cause of the symptoms. A other times, with slight rashes and no sore throat we may be in the presenc of a trifling erythema, or perhaps of a rash due to some special surgica dressing or to some internal remedies which are being used.

Tonsillitis with patches may occur without any connection with scarlatina, but it may also precede, but only for a day, the appearance of the scarlet rash. We may, however, miss the rash if very evanescent, or if search had not been made in time for it. Probably also scarlet fever may affect the child and its throat without any rash appearing at all; and also a sore throat may appear as a local manifestation of the poison (particularly in those already protected by an attack) without the whole system being contaminated by the contagion.

Similar difficulties beset the diagnosis of diphtheria: when well marked, nothing is more easily recognized; thus, we may have the white membranous exudation on the uvula, palate, and tonsils, with regurgitation of fluids through the nose, moderately high fever, and albuminous urine. But in a case with one or two insignificant white spots, like those of follicular tonsillitis, we may subsequently find, either in the same case or in another member of the family, that the trivial-looking illness was the fatal diphtheria. All such cases should be treated with care, and all should be labelled as more or less infectious, although it is not necessary to declare the existence of diphtheria openly till the symptoms or the sequel make it certain.

Itching at the nose and at the anus, and grinding of the teeth in sleep, have been regarded, with justice, as evidences of gastro-intestinal irritation: the first, indeed, has acquired a reputation as diagnostic of intestinal worms, especially when combined with pallor and wasting notwithstanding a good appetite. It is certain that picking the nose is very common in a multitude of cases where no worms appear. The writer, indeed, would counsel the beginner to refuse to make the diagnosis of worms till some are seen. Itching and scratching at the anus, if quite pronounced, are very suggestive of "seat-worms" (oxyurides vermiculares), but even then delay should be had till, after a purgative or an enema, the little worms are actually seen.

The "round worms" passed by children, or sometimes vomited by them, seem often to be expelled rather because of the child's illness from some other cause than to be themselves the cause of the acute attack. Sometimes they lodge in enormous numbers in the intestines of unhealthy children.

Tape-worms occasionally infest even quite young children: here, too, segments of the worm must be seen, to warrant a diagnosis.

Towards the beginning of this paper there are warning words against teething being regarded as a cause of disease; but these remarks do not warrant any neglect in ascertaining the actual state of the dentition, a subject to be fully discussed in another chapter.

In connection with the physical examination of the child, some remarks have been already made in the present chapter on peritoneal effusions and on glandular disease in the abdomen. Details as to these, and the varied disorders of the digestive organs, must be studied in the chapters devoted to them.

GENITO-URINARY SYSTEM.

Disorders in the genito-urinary system present fewer points calling for notice than in the case of the other physiological systems.

The occurrence of renal affections after scarlatina is in this connection one of the most important considerations, and general dropsy or albuminuria in the young should always make us think of this, although, apart from any fever, parenchymatous nephritis is specially prone to affect young subjects. The other forms of Bright's disease likewise occur in children,—contracted kidney occasionally, and amyloid kidney frequently.

In grave cases it is always well to examine the urine for albumen and sugar, and by the microscope. To do this, it will often be necessary have recourse to the catheter, if a sample cannot otherwise be obtained.

Gravel in the urine accounts probably for many painful attacks wi screaming, but it is only when we can recognize that the pain is with mict rition, or when we see the uric-acid crystals soon after the water is passe that we may be able to make the diagnosis. Occasionally, no doubt, the are renal colics quite unrecognizable in our young patients, although tl urine may guide the treatment if charged with uric acid or if mixed wi blood.

When the stone is in the bladder, painful micturition, with bloo especially at the end of the act, or the occasional stoppage of the strea or the presence of pus and mucus in the urine, may help the diagnosis; b this can be made certain only by sounding the bladder. Vesical calcul in children is almost always limited to males.

Pyelitis occurs in childhood; it may arise from a calculus, but probab is caused more often by scrofulous deposits in the pelvis of the kidney. Tl diagnosis is to be made as in adults.

Hydronephrosis occurs in children, and may indeed be congenital. Tl presence of a tumor and its variations in size, with great alterations in tl quantities of urine passed, may guide the examination.

Cancer or sarcoma of the kidney often attains to an enormous size i children, with great swelling of the superficial veins and the most extre wasting.

Diabetes, both in the saccharine and in the insipid form, is found i childhood: the saccharine variety is at times clearly traceable as an hered tary affection. The diagnosis is made as in adults, but the prognosis i childhood of diabetes mellitus is the worst possible.

Polyuria from granular and amyloid kidney must be remembered i making the diagnosis of diabetes insipidus.

Urinary sediments in childhood, apart from pus, blood, casts, and ep thelium, usually consist of urates or uric acid. Both deposits are often much paler than in adults, and white urates, sometimes with hedgeh crystals, are frequently responsible for the milky urine so often described b mothers and nurses: occasionally, of course, the milkiness may be due pus. Uric-acid gravel is comparatively common in childhood. Oxalat are often seen likewise in the sediments. Occasionally cystine is found i the urine of children, sometimes with, sometimes without, the concurren of calculus; cystinuria, although really rare, may be found in several mem bers of the same family. Cholesterin in the urine is very rare: the writ has seen it in a case with old inflammatory collections in the kidney.

Wetting the bed at night (enuresis, nycturia) may be regarded as esse tially an affection of childhood: it will be discussed elsewhere, but it i mentioned here more especially because the passing of water in bed may b the only available sign of an epileptic fit occurring during the night.

Disorders of the sexual organs need not detain us. The precociou development of them in childhood has been already mentioned. The discussion of malformations of these parts will be dealt with, no doubt in great detail, elsewhere. The irritation of a phimosis or its influence in determining masturbation or enuresis is often a matter for inquiry or for surgical treatment. In girls the occurrence of vulvitis and of purulent discharges from the genital passages may at times raise very difficult and disagreeable questions; these also must be discussed in special chapters.

In thus passing rapidly in review some of the symptoms which assume special importance in childhood or which occur under peculiar forms at early ages, it may seem as if the object of the writer had been to accentuate such peculiarities. Such accentuation is no doubt, in a sense, important and also desirable; but it is still more important that the diseases of childhood should be studied in the widest possible manner, comparing the symptoms of the same diseases as they occur in early life, in adults, or in the aged. The study of the diseases of children, from their variety and multiplicity, is not so apt as some other specialities to contract the mental view of those devoted to it; but strenuous efforts must be made to resist any injurious educational influence which may tend to arise even from the restrictions of age. The symptoms of disease must be viewed as they occur at all ages: the value and significance of these have been discussed by the writer at considerable length in his "Clinical Manual," to which he ventures to refer the reader, especially as under its various chapters bibliographical references to standard works and special memoirs will be found: it is only in such full and elaborate treatises as these that the satisfactory discussion of symptoms is at all possible.

THE

INFLUENCE OF RACE AND NATIONALITY

UPON DISEASE.

BY J. WELLINGTON BYERS, M.D.

[illegible] designation it will be appropriate to enumerate and discus [illegible] characteristics, associated with the phenomena of disease [illegible] be dependent upon or attributable to differences of rac [illegible] That the various communities of mankind, situated a [illegible] dissimilar circumstances of life, should manifest diver [illegible] proclivities, as well as in their physiological tendencies [illegible] worthy of acceptance. While it is true that each race, as [illegible] susceptibility to the morbific influence of all ordinary dis [illegible] can lay claim to complete immunity from any, still w [illegible] that there are many marked variations and contrast [illegible] and in the manifestations of morbid phenomena [illegible] remarked, à *propos*, "Man is not born, does not live, does no [illegible] in the same manner, on all points of the earth. Birth [illegible] death all change with climate and soil, all are modified b [illegible]." In passing to a consideration of the causes or condi [illegible] these differentiations in disease, we must not conjectur [illegible] determining factors are the same as those normal or physiologica [illegible] subsist between the various races. Deductions based upo [illegible] such as color of skin, character of hair, size, shape, an [illegible] dimensions of the skull and face, which in themselves, as ca [illegible] appreciated, could not possibly modify or control the expressio [illegible] are not to be interrogated here. It seems best, both from pas [illegible] and from careful estimations, to attribute variation in susceptibility to certain fine differences in the structure and activity of the tissue that are concerned in the manifestations of morbid processes,—particular that belong to the domain of the histologist. As to exactly how these disparities between people came to originate and are now perpetuated, without fully comprehending their precise nature, we cannot ascribe a better sourc than that of *climate* acting through long periods of time. We know that in this we have a powerful element for good and for bad. Again, we know that a race indigenous to a certain country acquires, through many generations, *characteristics*, the formation of which can be distinctly traced to climatic and telluric influences. We know, further, how habits of life definitely determine the susceptibility and non-susceptibility to certain diseases, and that change of environment always induces more or less change in the physique, mental traits, and morals of any man, or group, or even nation, of men. While no attempt will be made to theorize, yet it does seem probable that an explanation of many of the dissimilarities which are incidental to the morbid phenomena of the different races might be traced to these contrasts of environment, such as soil, climate, geographical position, food, and social condition, all of which are known to be influential or qualified to alter the type and *personnel* of disease.

Among these predisposing conditions that are found to modify disease, none seems more prominent and worthy of our consideration than that of climate. Of course this of itself is totally incapable of generating *specific disease*; yet that it does institute constitutional peculiarities and tendencies, through molecular modifications of the tissues, which gradually become permanent and capable of hereditary transmission, would seem to be amply warranted by past experience and observations. Man, like the animals and plants, bears the stamp of geographical areas, and as the varied regions have their natural order of fauna and flora, so it is with the different races: neither can transgress its fixed limits without danger, deterioration, and ultimate destruction. By reference to the latitudinal position of the different races it will be seen that each one has its own special limits of health defined by rigorous laws of climate. The Icelander perishes with phthisis if transported to Copenhagen, the tropics are fatal to Europeans, and the Dutch have been totally unable to people Sumatra and Java. For the white race, we find them enjoying the most perfect health and the highest type of physical and intellectual development in the temperate zones, above 45° in the Eastern and above 40° in the Western Hemisphere. Whenever they migrate many degrees below these lines a series of profound physiological changes set in, which continue in the ratio of the length of residence, together with the conjoined effects of heat and other influences incident to hot climates and habits of life.

On the other hand, if we look at the intertropical races approaching the latitudes of the white race, we see a list of deleterious influences begin that gradually grow worse as they come nearer. In some climates everything tends to hinder. No Asiatic race has ever shown that it possessed the power of resistance or the power of progress. Neither the Mamelukes, who were a Caucasian race, nor the Turks, who are Mongolians, unless they married native women, which the Mamelukes never did, could continue their race in Egypt, all their offspring perishing in the first or second year (Volney).

Dr. Kenneth McKinnon says of the children in Bengal that, "Even when there is no tangible disease, nutrition and oxygenation do not appe to go on favorably; the skin is pale, the muscles wanting in substance ar tone; the joyous spirits of children are wanting, the body is inert and tl mind listless." How utterly unfavorable the circumstances are here doubly emphasized by Dr. Twining, who states that, after much search an careful inquiry, he was unable to find anywhere a sample of the third ge eration from unmixed European stock. The Hindoos are of Caucasia origin, but exposed during countless generations to the same succession c depressing influences of climate, so that a temperament is begotten whic differs widely from that of Europeans, as is seen in their incapacity withstand disease, and in their low nervous and muscular power.

While it is true that man can and does overcome many of the barrie of climate, yet these violations of his natural habitat and régime are alwa sooner or later followed by reactions that bring about disaster to his phys cal well-being. The Khirghis pasture their flocks on the Pamir platea perhaps the highest steppe in the world, and they dwell in the tropical par of Southern Egypt, as well as at the ill-famed Massowah, on the Red Se We find the Chinese at Kiakhta, on the Siberian boundary, where th mean temperature is below freezing and the thermometer falls in winter t 40° Réaumur, and we find them in the island of Singapore, which almo touches the equator. Turkish races, such as the Yakuts, are settled on th Lena, where Kennan found them gossiping in the open air at a tempera ture of 32° Réaumur, clad only in a shirt and a fur coat. These are suffi cient to show that man is capable of forcibly adapting himself to almo any circumstance of life; but he does so at great cost to his physical an intellectual well-being, for in every instance where the departure from hi original habitat has been distant and at short intervals of time he ha suffered proportionally.

There are certain other facts associated with man's geographical distri bution over the earth, among which must be mentioned that in approachin the equator it has been found that the mortality is increased, and conse quently the average duration of life is lessened.

Again, further observation goes to show that the proportional numbe of individuals who attain a given age differs in different climates, and th warmer the climate, other things being equal, the shorter the average life early puberty being always associated with premature decay. In Europ this is illustrated, being for Italy one death in twenty-eight, and for England one in forty-six. From estimates made a few years since (Dr. C. F Campbell, *Science*) upon the adult males belonging to the society of Odd-Fellows, in the United States, it was found that the death-rate of thos of the Southern or tropical States was just twice as great as that of thos in the temperate ones of the North, it being for the former, as an annual average, 1.42 per 100 death-rate and .946 week of sickness, and for the latter, as an annual average, .70 per 100 death-rate and .266 week of sickness. In making a comparative use of these statements to govern observations in regard to sickness and deaths among children, it must be added that, while high temperature is nearly always inimical to them, still the causes of death in children afford very many contrasts to those of adults, and consequently our conclusions should be guarded. Under this special heading we have the results of the compilations of Dr. Eklund, of Stockholm, which gives the annual death-rate of children under one year in various parts of the world. For Europe, the average he states as being twenty-five per cent. of all deaths, and for the largest cities of the United States he says, "The infants dying under one year average fifty per cent. of the whole number born;" though in contrast to this assertion I shall have to add that the average death-rate of such children for thirty-one registered cities, as given by the Tenth Census, was only 267.5 per 1000.

Signor Bodie has recently published some figures that corroborate Eklund's data very substantially as regards Europe. He, however, states that only twenty per cent. of the children die in the first year, that ten per cent. die in the first month, and that full thirty-three per cent. of the remainder die before they reach the fifth year. Following the data of Eklund, the lowest death-rates are found in Belgium, Denmark, Sweden, England, and Switzerland, where they vary from 14.3 to 26.2 per 100, the highest rates, from 31 to 48 per 100, being found in Austria, Germany, and Russia. For Berlin it is given at 58.1, for Paris 30.8, and for St. Petersburg, where the death-rates are known to exceed frequently the births by as many as 100 per month, the rate is 32.5 per 100. The variations in these figures are not sufficiently marked and general to warrant the conclusion that the factor of climate is as important in governing the diseases of children as in adults. However, when we come to consider the factor of *class or social condition*, we shall find a decidedly prevalent and potent influence at work in this kind of individuals. M. Körösi, of Buda-Pesth, has found the following to represent his experience in the matter of susceptibility to infectious and contagious diseases among children. Cholera, small-pox, measles, and typhoid fever are more prevalent among the poor, while diphtheria, croup, pertussis, and scarlet fever are found among the well-to-do. Zymotic diseases, as a whole, are sixty per cent. more frequent among those living in basements than in higher domiciles. But the increased mortality in underground tenements applied only to certain diseases, especially measles and pertussis, while diphtheria and scarlet fever were ten per cent. less than in people living above-ground. In regard to other statistics bearing upon the subject, those of the city of Dublin show that *class* has a decided and important influence upon the longevity and health of town-dwellers. For the first class of society, including the members of the professions and independent people, the death-rate was found to be 22.5 per 1000, for the middle or second class 25.4, for the third class, shopkeepers, artisans, and trades-people, 26.1, and for the fourth class, composed of those in service, jails, and workhouses, 37.2 per 1000. In London the death-rate has been found to be, among the rich, from 12½ to 25 per 1000, and for the poor, fro 25 to 35 per 1000. The well-to-do in England have an average life fifty-five years, while the artisan class live only a little over half of th —namely, twenty-nine and a half years.

From the foregoing statements of climate, and its undoubted influen through temperature, food, etc., of geographical distribution and its rel tions to birth, maturity, and decline, and of class, with the differenc that it is seen to produce in sickness and mortality, I think that may properly infer that they constitute some of the principal sourc through which we may explain the diversities in type and susceptibility the different diseases among different people. If, as will be seen, the neg and the yellow race differ widely from the European in their suscept bility to the morbific influences of certain diseases, then we must conce that this is because of the differences in the methods of their living, the location, diet and habits, together with the other peculiarities that exist the structure and functions of their tissues, to which we can now on briefly allude. Reasoning from analogy of those diseases that we know exist by acquired constitutional peculiarities, such as ichthyosis, where is the structure and mode of growth of the epidermic cells, hæmophili where it is the structure, presumably, of the blood-vessels, and Daltonis where it is the finer details of the retina, we are led to believe that it is t same with the races, and that in each it is the peculiar structure and activit of the tissues which are mostly concerned in each disease, that are at faul Of course our present knowledge does not furnish us with sufficient data offer a description of these subtile changes; yet that this is a proper explan tion appears to be warranted by such knowledge as we do possess, bein particularly applicable to those classes of disease due to infection,—tho obviously attributable to morbid animal poisons and bacteria. The limi of this chapter preclude any further notice under this section, and we sha now enumerate a few of the principal diseases in which race and nationalit are seen to affect their order and phenomena, enabling the reader to see th scope and purpose of these distinctions.

Whooping-Cough.—The history of this disease begins in the middl of the sixteenth century, when Baillou gives the first description, though must be said that a disease presenting such well-defined characteristics mu have existed long before. The native habitat of it is probably very narrow and does not correspond to its present limits. In Europe the disease is tole ably uniform in its diffusion, the Scandinavian countries showing it quit extensively. In Sweden, from 1749 to 1764, more than 43,000 childre died of it (Rosenstein, "Kinderkrankheiten," 1785), and from 1862 t 1881, according to official returns, there died 86,000. It is said to b equally fatal in Russia, Denmark, and Norway. In Prussia, from 187 to 1880, nearly 85,000 children succumbed to pertussis. In England an Wales, from 1848 to 1855, there were 72,000 deaths, and from 1858 to 1867 120,000. It is scarcely less fatal in France, Belgium, Germany, Holland

Scotland, and Ireland. In the latter, according to Wylde's account (*Edin. Med. and Surg. Jour.*, 1845), it has the character of an endemic malady, with a mortality that ranks fifth on the list of the causes of death, it being in 1841 the cause of 37,300 deaths. Only four examples of it have been noted in Iceland during the present century, and Finsen in a practice of ten years never saw a case there. There have been only three epidemics in the Faröe Islands. According to Tobler, it is epidemic in Palestine. In Australia it is comparatively a new disease, and has occurred only since 1830, but it was epidemic in the years 1843 and 1855. It first appeared in New Zealand in 1847. In Africa it has occurred only in a few regions and some of the adjacent islands, such as Mauritius, Madagascar, and Cape Colony, also in the interior of South Africa, Western Soudan, Algiers, and Egypt. In Egypt, Pruner has seen it in children of every color. According to Heymann and Waitz ("Diseases of Children in Hot Climates," 1851), the disease in the East Indies is found as much among the children of the Malay and Japanese population as among theEuropeans. Milroy, Mackay, and others speak in general terms of its occurrence among the native children of India: there it occurs from the lowest altitude up to 6500 and 8000 feet. It is rarely found in Central America, though often seen in Brazil, Chili, and Peru. In the Northern and Western States of this country it is frequently as common as in Europe, and occurs in all seasons of the year. According to experience, it attacks all circles and classes of population, though it is more fatal in the poor than in the rich. The disease is more fatal in males than in females, in the country than in cities, and assumes its most unfavorable type in mountainous sections. In regard to the influence of race, it is more than twice as fatal for blacks as it is for whites, the proportion being, according to the Tenth Census, for whites 14.3 and for blacks 33.0 for each 1000 deaths from all causes.

Cerebro-Spinal Meningitis.—The general history of this disease until quite recently seems to have been largely occupied by Europe. The countries that it has been oftenest and most extensively seen in are France, mostly in the southern, western, and northern parts, Italy, particularly in the provinces of Sicily, and Sweden as far north as 63°. It has been prevalent, though rarely, in Ireland, Russia, Denmark (previous to 1848), Hungary, Austria, and Greece. It is absolutely rare in the Iberian Peninsula, Roumania, and Turkey. It is not common in England, the Netherlands, and Switzerland, except in very slight and occasional forms. Scotland, Belgium, North and Central Italy, and Iceland seem, as yet, to have well-nigh complete immunity. The only references to it in the Orient are from Persia, Syria, and Asia Minor, and some few cases that have been met with in India. So far as published reports are calculated to give information, the West Indies, Mexico, and Central America are free from it. With regard to Africa, Algiers is the only place where we hear of it, and, according to Pruner, the Arabs suffered equally with the whites. There seem to be some discrepancies in the accounts of this disease as it pertains to the white and colored races. In several epidemics that have visited the United States, the negro race has suffered with unusual severity. Of 85 patients treated by Ames, of Montgomery, Alabama, 23 were white and 63 black. In the epidemic at New Orleans in 1850 the negroes suffered most, and it was chiefly confined to them. The same was the case in Memphis, Tennessee, in 1862–63, when it broke out among the colored troops. There were strong evidences of the unusual susceptibility of the colored race in the epidemics in Mississippi, 1862–63 (Hughes, "Trans. Miss. Med. Convention, 1878"), in Mobile, Alabama, 1864–65, and in Maryland in 1864. According to Williams, of Montgomery, in 1848, out of 84 patients 10 were under 10 years, and 23 between 10 and 20, the disease being particularly severe in the negro. In the disease at Philadelphia in 1867, Githens says that it was "particularly severe in the negroes." At the present, and for several years past, this country has been the chief seat of the disease. In distribution it has extended from Canada to the Gulf, and from ocean to ocean. The first outbreak presenting an epidemic character occurred in 1814, and swept over the New England States, lasting until 1816. A remarkable feature of this occurrence was its being limited nearly entirely to children, the various epidemics throughout the world heretofore showing marked differences in this respect. There can be no doubt, judging from the data of past experience, that this disease is more severe in the dark races than in the whites, the ratio of susceptibility and severity being exactly proportional to the depth of color, the blackest suffering most.

Trismus Neonatorum.—One of the most notable points connected with the geographical distribution of this disease, as compared with tetanus in the adult, is the much greater prevalence of the former in the higher latitudes, and even in the extreme polar regions, of both hemispheres. This is particularly true of large and populous towns, where most of the deaths from trismus among new-born infants are among the poorer classes and in the ill-kept foundling or lying-in hospitals. In Cayenne this disease is terribly fatal. In British Guiana, according to Hancock, it kills, upon an average, more than half the children born. In Cuba it is very disastrous, there being 369 deaths reported in the official documents in two years. In Jamaica twenty-five per cent. of the negro children die of it each year; and it is said to be equally destructive in Barbadoes, Grenada, and St. Thomas. Bourel-Roncière estimates the deaths from trismus in Rio Janeiro at one-fourth of all the infants born in one year. In the city of Buenos Ayres, with a population of 200,000 and a yearly increase of one thousand, the deaths from trismus in 1875 were 445, though they have steadily declined ever since, and in 1880 were only 108 for the first six months of the year. Mantegazza witnessed a frightful epidemic in Montevideo in 1852. The deaths in New Orleans during the same year were 249 out of a total mortality of 6617, or 37.6 per 1000. The mortality in Charleston, South Carolina, in 1856 was 40 per 1000. In the tropical regions of the Eastern Hemisphere the disease is equally severe, epidemics having visited India, Africa, and the Malay Archipelago. There seems to be little information of recent date concerning this disease in Southern Europe. According to Wylde, in 1854 it became quite common in Ireland. It has also been mentioned by Paterna as occurring in Italy in 1835. It became epidemic in Iceland a good many years since, and was attended with great loss of life: during the endemic which visited the Westmanna Islands, off the southern coast, the mortality reached as high as sixty-four per cent. Trismus is said to be more common among Jewish and Mohammedan children than among those of other races. We are indebted to Baldwin, of Alabama, and Dowell for a description of this malady in the negro child. Experience shows that in negroes the disease is always attended by a high rate of mortality, very few getting well when once attacked. In the Dublin Rotunda Hospital the death-rate from 1757 to 1882 was sixteen per cent., and a large proportion of these were said to be caused by trismus. Viewing this disease in any of its causative relations, whether filth, urine on the cord, pressure upon the brain, or what not, one thing is always prominent, and that is neglect or inattention to the ordinary care of the infant. I have never seen a case in a well-kept, clean, and sanitary place. On the other hand, in my experience, every colored child born in dirty surroundings that takes it certainly dies.

Scarlet Fever.—The area of diffusion of scarlet fever is much less than that of small-pox or measles. That the continents of Asia and Africa, which are the chief seats of the latter two diseases, have never been severely visited by scarlet fever, is a remarkable fact. It occurs as an epidemic much less frequently than measles, and in some places its visits are ten or twenty years between. Again, there are great fluctuations in its types, some severe, others mild. Withering, one of the earliest and best authorities upon scarlet fever, states, from his Birmingham experience of 1778, that the disease raged severely in many elevated, dry, and airy places, while the dwellers in low, damp, and ill-ventilated parts of the town suffered to only a slight degree. Graves says ("System of Medicine," 1843) of the epidemic of 1839 in Ireland, "The nature of the disease was not in the least affected by the situation of the dwelling, it being equally bad in Dublin as in the hills." The origin and first habitat of scarlet fever are unknown, its history being linked with that of measles. It appeared in England and Scotland in 1661 (Sibbald, "Scotia Illustrated," 1684), at Berlin in 1716, at Florence in 1717, and in Denmark in 1740. When and where it first appeared in Asia and Africa cannot now be determined. At present it is most met with in Europe,—in the countries of Germany, France, England, Russia, and the Netherlands. It goes as far north as Iceland, and in the south extends to Brazil and Chili. Greenland has escaped, and it occurs only at rare intervals in Newfoundland.

Pruner states that, so far as he knows, the colored races are exempt from scarlet fever, though Moulin believes that the negroes of Senegambia have it rarely. It seems from the account of the disease given by Frick, of Balti-

[illegible] in Amer. Jour. Med. Sci., 1855, that the whites and negroes at one time suffered in equal proportions, the figures given being for the epidemic of [illegible] in Baltimore as follows: for whites, 13.8, and for negroes, [illegible] per 10,000 of population. According to Drake, the South is less frequently visited than the North. In South America, following the accounts of [illegible], Sigaud, and others, the disease is frequently of the same type among the colored as among the whites. Mantegazza (*Edin. Med. Jour.*, [illegible]) states that the creoles suffer more than the whites; and that the red-skinned natives of North America (in Canada) are at least not exempt follows from the remark of Stratton (*Lancet*, 1870), "In epidemic scarlatina it appeared to me that the Indians were less susceptible of an attack than the whites."

The United States Tenth Census shows the difference of mortality in the races as follows: for whites, 20.9, for negroes, 3.9,—a very marked contrast. As to nationality, the German has a death-rate of 30.1, and the Irish [illegible], though the mortality of the latter, in children under five years, is very much higher. The census shows that this disease is far more prevalent in the northern portion of the United States, especially in New York, Pennsylvania, the Ohio Valley, Wisconsin, Minnesota, Kansas, and Nevada, than in the South. This corroborates past experiences, though the disease is much rarer now in the South than in former times.

Measles.—The area of distribution of this disease extends nearly [illegible] the entire earth. According to Lange, it had not occurred in Greenland previous to 1864. Vinson says that New Caledonia has, up to the present time, also escaped. The susceptibility to the virus of it, as is seen from its geographical distribution, is uniformly shared by the whole of mankind, of whatever race or nationality. If among the colored people it runs in its worst forms, and leads to disastrous results exceptionally often, the reason does not lie in their physiological peculiarities, but in their unfavorable conditions of life. It appears to be quite independent of climatic influences, though the disease occurs mostly during the colder seasons of spring, autumn, and winter. It seems to run as mild a course in tropical and subtropical as in temperate climates, though in some sections, as the Himalayas, and in Honduras and Madagascar, the disease takes on peculiarly malignant forms. Fuchs says of parts of England, France, and Holland that the disease is most malignant especially in the presence of malaria. Squire gives an account (*Medical Times and Gazette*, 1877) of an epidemic in the Fejee Islands which destroyed twenty thousand people, or nearly one-fourth of the entire population. Among uncivilized peoples it is known that the disease often assumes its worst forms. In 1749 an epidemic destroyed thirty thousand Amazon Indians, of Brazil. In 1849, one-half of an entire tribe of American Indians perished. It was equally fatal in 1846 among the inhabitants of Hudson Bay, and in 1852 among the Hottentots. In Paris during the siege (1871), out of 215 of the Garde Mobile who took measles, 86, or 40 per cent., died. In the epidemic which prevailed among the Confederate forces during the civil war (1864) there were 38,000 cases, and 1900 died, the mortality being in some hospitals as high as twenty per cent. Masterman says of the epidemic of the National Army of Paraguay, at the beginning of the war with Brazil, that "it swept away, in three months, nearly one-fifth of the entire army." Hirsch considers that malignancy of type is due more to mistakes in dieting and treatment than to other influences, and he accounts for the severity of the disease upon these grounds. Fuchs says, "In the north, and in the elevated districts, the inflammatory forms occur, while in the south, and in the flat districts, it is more usually the asthenic and putrid forms that occur." Among the Chinese measles is quite common, though with the peculiarity of not protecting them against the kind that afflicts the white race. Measles has increased in Paris since 1865 from 31 to 46 per 100,000 in 1887. For the United States the average age of those dying from measles is seven years. The deaths are greater in the rural districts (12.3) than in the cities (7.4). Among the whites the ratio is 9.1 per 1000, for negroes 17.7, for Germans 8.5, and for Irish only 5.3.

In regard to the peculiarities connected with eruptive fevers in the dark races, of course the feature of color is a variable one. In the pure negro the eruption of measles appears as yellowish spots slightly elevated and giving a sensation of roughness; in the mulatto, as dusky brown, ill-defined spots; and in the lighter shades, as more distinct, reddish-brown spots, approaching the characteristics of the disease in the white race. The high mortality of this disease among the negro race is doubtless due to want of attention and absence of proper diet, although the subtile influence of race certainly has some contributing power. Among them in the South, as a rule, the disease is considered harmless, and no precaution is taken for protection or to prevent its spreading. Among the Indians this disease occupies a prominent position on the list of death-causes, and it is often followed by serious sequelæ, which lead to fatal results.

Diphtheria.—This is one of the standard diseases of modern times. Whether some races are especially predisposed to take diphtheria, while others enjoy a pronounced immunity from it, cannot be decided for certain with the scanty and conflicting information that we possess relating to epidemics in localities where the population is a mixed one. The statement of Odriozla, that the negro is protected in Peru from diphtheria as well as yellow fever, is contradicted by Tschudi, who says very emphatically that it is the children of the negroes in that country who suffer from malignant sore throat, an opinion that has also been supported by the published experiences of Goldsmith, of Oakland, Missouri, and of Smart, of the Bermudas. The inference drawn from the exemption of the Chinese during the epidemic in Victoria, that the Mongolian race has immunity, is just as little warranted in fact, for the disease, according to Dudgeon, is widely diffused through Northern China, and has not spared the Japanese. According to the reports of the English, Southern China has not, as yet, been visited by the disease.

In the epidemic which visited Pekin in 1866 there died from diphtheria no fewer than twenty-five thousand Chinese. Japan was visited in 1877, and Yokohama suffered severely. In other parts of Asia, such as Smyrna, it became epidemic in 1865 and spread throughout Asia Minor, though up to 1868 the disease had not been severe in Syria. Persia had a disastrous visitation in 1874–78. In India there have been several epidemics, the first occurring in 1800. In Africa there are but few accounts of the disease, and up to the present it seems to have occurred only in Senegambia and on the West Coast sporadically, though in South Africa there was a general and severe epidemic in 1866. New Caledonia has never been visited. On the Eastern coast the disease has been indigenous since 1837. Pruner found it in Egypt, and it occurs in Algeria and Tunis. It has been epidemic in most South American countries and the West Indies. Climate and season do not seem to influence the disease,—the same features appearing in Sweden as in Spain. The inference is that the exemption of those countries which have thus far escaped it is due rather to a want of disease-producing cause than to non-susceptibility on the part of the race or nation. Since 1856 the disease has become generally diffused throughout North America and Europe. From statements made in the United States Tenth Census, it appears that diphtheria is especially prevalent among the German population of the Northern States; and upon the whole the disease seems to be making progress. In England during the year 1880 the deaths from diphtheria were 532 per 100,000 deaths from all causes. W. Roger Williams, from an examination of ten million hospital cases of various diseases in England, concludes that the disease is more common in females than in males,—a result that finds corroboration in the government statistics of this country. In regard to my personal experience among the colored race of the Southern States, I have found that they are less disposed to contract the disease than the whites, and out of one hundred deaths from this cause I have not found more than one-fifth to occur among the blacks. From statistics obtained by the government, it appears that diphtheria is more fatal in rural than in urban districts, the order of frequency in the different races being, for each 1000 deaths from all causes, as follows: whites, 52.63; Indians, 37.36; blacks, 23.27. From these figures it will be seen that diphtheria as it occurs in this country is more than twice as fatal among whites as among colored, notwithstanding the fact that diphtheria is a disease of the poorer classes and mostly confined to the country, or at least most fatal there.

Cholera Infantum.—We are indebted to Benjamin Rush for the first account of cholera infantum in this country. The disease certainly occurs all over the globe, and it is proportionally worse in those localities where impurities of water and soil exist, together with extreme heat and a crowded population. As regards the influence of race on the incidence of cholera infantum, it must be mentioned that in 1855 Frick, of Baltimore, regarded the mortality as being twice as great in the white as in the colored race (*Amer. Jour. Med. Sci.*, 1855). Upon reference to the last census we find that the proportion of deaths for each 1000 from all causes is as follows: whites, 99.2; colored, 71.2; Irish parentage, 68.0; German parentage, 90.2; though as regards these figures it must be added that the term employed by the census enumerators also embraced dysentery, cholera morbus, and enteritis. The total number of deaths from this cause has largely increased during the past two decades, and the number dying under five years constituted sixty-six per cent. The death-rate in cities is higher than in the country, being for the former 96.1 and for the latter 86.7 per 1000. A study of the geographical distribution of this disease reveals the fact that the regions showing the largest proportion of deaths are the Valley of the Mississippi, Texas, Kansas, part of Missouri, Georgia, and South Carolina. In Massachusetts and Michigan the death-rate from this disease amounts to 15 per cent. of the annual mortality, and in Boston it is 22.18. In the city of New York, from 1805 to 1837, the deaths from cholera infantum were nearly 2 per 1000 of population, and in recent years it has increased considerably. In Philadelphia the mortality in 1872 was reckoned at 2.6 per 1000 of population, while from 1819 to 1860 it amounted to one-fourth and one-third of the deaths from all causes (*Trans. Penna. Med. Assoc.*, 1873). It has been higher than this in Baltimore, Louisville, Natchez, St. Louis (1841–43), and at Memphis, which has been termed the graveyard of children (Grant, *Amer. Jour. Med. Sci.*, 1853).

There is lack of information concerning this disease in Mexico and Central and South America. At St. Pierre, in Martinique, we learn from Rufz (*Arch. de Méd. Nav.*, 1869) that it is the chief cause of death in those under one year. At Barbadoes, Jackson says, "it is by no means as common as might have been expected from the tropical situation." In Africa the only mention made of the disease is at Port Said. For Asiatic countries there are no accessible reports or data. Richardson says (*Edin. Med. Jour.*, 1869) that the mortality is very high in Australia.

In Europe there is unanimity in the opinion of its being one of the leading causes of death in children, if not the first. This is especially true of the large cities. There appears to be little difference in the death-rates of the cities of the Eastern and those of the Western Hemisphere. In Berlin from 1877 to 1882 the death-rate had a mean of 2.6 per 1000 of population. In Hamburg from 1874 to 1884 it was 1.9, and in Stuttgart from 1873 to 1878 it was 2.4, per 1000 of population. In Birmingham, England, the mortality for each 1000 population was, from 1873 to 1875, 2.04. In St. Petersburg, where the mortality of infants is extremely high, the death-rate for 1887 was 3.08 to each 1000 of population. In Cairo for the same period it reached 12.08 per 1000 of population, which is doubtless the highest of any city in the world.

With regard to this disease in the colored people of this country, my personal experience shows the mortality to be very high. Very few of the cases get well, if at all protracted. The absence of proper food, nursing, and skill, together with the inherent impotence that the darker races have always shown when attacked with diarrhœal diseases, makes it peculiarly fatal.

Small-pox.—Experience shows that the colored races, and especially the negro race, are, *cæteris paribus*, in greater risk from this disease than whites. "The human family," says Pruner, "that are most susceptible to this poison are negroes. Not only in their native lands, but in other parts of the world as well, they are the first to succumb to the epidemic influence, and also the last. It is no unusual thing to see negroes attacked by this disease as soon as they arrive in Egypt (where they certainly change their way of living as well as climate), and that, too, at times when the disease does not exist among the other inhabitants." Similar statements, equally emphatic, as to the increased intensity of the disease in negroes, are made by Daniell for the West Coast of Africa, for Martinique by Rufz (according to his observations in the epidemic of 1836–37 and 1848–50), for Curaçao, and for Cayenne. For Peru, Bajon says the same. During the epidemic of Baltimore in 1850 the deaths among 10,000 of population were 8.1 for the whites and 14.5 for the negroes. In 1838 an epidemic of small-pox among the Mandan Indians swept away all but 133 persons of the entire tribe of 1800 souls.

Hirsch considers India and the countries of Central Africa as the native foci of this disease. Martin says that he has never seen a case of small-pox in India in a European child, although there was not a year in which it did not occur. Among the yellow races of the East, the Chinese, Japanese, and Coreans, this forms the chief scourge to which they are subjected, it being a common thing to be daily jostled in the streets by those in every stage of eruption. In the countries of South America small-pox frequently plays sad havoc with the native population of Indians and half-breeds. The official report for the city of Rio Janeiro for 1887 shows a mortality from small-pox of 22.5 per 100 deaths from all causes. The southern countries of Europe—Italy, Spain, and Portugal—furnish the largest annual contingent to the total mortality from small-pox, Rome showing for 1887 a rate of 35 per 1000, and Lisbon 65 per 1000. Small-pox is often severe in the higher and colder sections of Siberia and Russia. My own experience in an epidemic at Atlanta, Georgia, in 1882, leads me to believe that negroes are far more susceptible to the pathogenic factors of this disease than the whites, since out of one hundred cases the death-rate for the blacks was ten per cent., the whites escaping without a single death.

Syphilis.—This disease has extended its influence over the entire habitable globe. Like typhoid fever, it relentlessly dogs the steps of man, and no race, people, or nation enjoys complete exemption from its withering curse. Reference to the past history of syphilis shows that it has pursued very different careers among the various races. The first outbreaks of this disease among a people are generally considered the worst, and these are, commonly speaking, proportionally severe as the people approaches a state of nature. Certain sections, such as Iceland, Greenland, Newfoundland, South Africa, and Madagascar and the adjoining islands, have so far been signally free from its visitations. For Iceland, Finsen, after a practice of nine years, says he saw only five cases, and these were in strangers. It has been known to be twice imported into Iceland, once in 1756 and again in 1824, the latter epidemic infecting the natives to the extent of only twenty-two cases.

Lange is reported as saying for Greenland, "It is a remarkable fact that there is absolutely no syphilis here. About this fact there is no doubt; and the strangeness of it will be less when we remember that the same is true of Iceland. The circumstance is explicable simply and solely on the ground that Greenlanders and Icelanders have an immunity from syphilis, for there is no lack of opportunities for infection, since vessels frequently visit here with the disease on board, and prostitution is far from uncommon." The Icelanders are of the same Scandinavian stock that pays a heavy tribute to syphilis in Sweden, Norway, and Denmark, while the Greenlanders are of the same blood as the Esquimaux that inhabit the west coast of North America, and they from time to time have had terrible visitations from the disease. Dr. Moncorvo, of Rio Janeiro, stated during the International Medical Congress of 1887 that syphilis furnished full 65 per cent. of all causes of infantile disease in Brazil.

Darwin and Borius assert that the negroes of Madagascar are exempt, while the Hovas, of the Malay race, are frequently and seriously affected. There seems to be some ground for the popular notion that syphilis contracted from persons of Mongolian blood by Europeans is particularly noxious, as all examples of such prove very intractable. The yellow races of the Orient are great sufferers from this disease, the countries of Japan and China being particularly infected. The Indians of North America have shown during recent years a steady increase of venereal diseases, and the hereditary form has well-nigh pervaded a majority of them. With regard to syphilis in Africa, Dr. Livingstone says in his Travels, "A certain loathsome disease which decimates the North American Indian and threatens extirpation to the South Sea Islanders dies out in the interior of Africa without the aid of medicine; and the Bangwatse, who brought it from the West Coast, lost it when they came into their own country, southwest of Kolobeng. It seems incapable of permanence in any form in persons of pure African blood anywhere in the interior of the country. In persons of mixed blood it is otherwise, and the virulence of the secondary symptoms seemed to be, in all the cases that came to my care, in exact proportion to the greater or lesser amount of European blood in the patient. Among the Corannas and Griquas of mixed blood it produces the same ravages as in Europeans; among half-blood Portuguese it is equally frightful in its inroads upon the system; but in the pure negro of the central part it is quite incapable of permanence." Fritsch, in commenting upon this opinion of Livingstone, says "that syphilis is very rare, and it occurs in Bechuana Land only in scattered cases, mostly imported from Cape Colony, though there are materials with which to controvert the assertion that this disease does not hold with pure Ethiopian blood." That this disease does attack the negro in a mitigated and less virulent form than it does the white and other races appears probable from past experiences in this country. The last census shows the disparity; and from my own personal observation the disease is far less formidable in the negro, and is readily cured by appropriate treatment. It certainly, upon the whole, pursues a milder course, there is less damage to the system, and there are fewer lesions of any kind.

Scrofula.—Europe is the classic ground for scrofula. Few or none of its countries are free from the inroads of this disease. In Italy, Sormani says, it has so contaminated the populace that 3.5 recruits out of each 1000 are exempt on account of it. In France affairs are worse, the exemptions from military duty being three times as many as for Italy, the proportion reaching as high as 15 to 20 per 1000 in some sections. Belgium, Holland, Switzerland, Germany, and Austria are equally infected. Scrofula is generally diffused throughout the Scandinavian countries, and Ireland pays a heavy tribute. For England the number of scrofulous persons in each 1000 has been estimated by Phillips to be 24.5, in some parts as low as 11, and in others as high as 72.

Cooper says that it is common for people who leave England for the Indies to acquire it upon their return home. Pritchard alleges the same for those who leave the Southern States and Brazil and go to reside in Paris. Scrofula was so prevalent in Lisbon in 1842 that Rozas asserts that the children of the orphanage of that city had it to the extent of 35 per cent. In Asia it is one of the commonest diseases met with, it being well-nigh universal among the Japanese and the Chinese. The harems of Egypt are said to be infected, while in Tunis and Algiers it occupies among the negroes the first place as a disease. Black affirms that scrofula is frequent among the Kaffirs and Hottentots of Cape Colony, though Livingstone says that it is rare in Africa, being quite unknown between latitudes 15° and 25° south in Central Africa. Among the negroes of the Southern States this disease is seen very often, being, according to the last census, more than twice as common as among whites. The Indians are fast becoming the most scrofulous people on the earth. Sixby writes of the Shetland Islands, "Scrofula is connected with every family." In Australia and the Hawaiian Islands this disease has existed only during recent years. In Greenland and Iceland, according to Lange, it is exceptionally rare. Gordon, writing of India, in the *Medical Times and Gazette*, 1855, says, "Scrofulous affections were the most frequent ailments of children of both sexes, and the mortality was exceedingly high."

In bringing this article to a close, I must here say that the list of diseases presented by no means exhausts the list of those manifesting racial characteristics; for some of the most important ones, such as malaria, typhoid fever, yellow fever, cholera, dengue, etc., have been purposely omitted. I must acknowledge my very great indebtedness to Prof. Hirsch's "Handbook of the Distribution of Disease" for many of my citations.

OUTLINES OF PRACTICAL BACTERIOLOGY.

BY EDWARD O. SHAKESPEARE, M.D., PH.D., ETC.

IN this article it is proposed to outline only practical methods which have been found, in the writer's own experience, to be useful in examination of the relations of bacteria to disease. The limited space makes it impossible, and the requirements of such an article perhaps also undesirable, to enter into historical or theoretical considerations.

This field of investigation was opened for the medical scientist by the classical researches of Pasteur in the settlement of the question of spontaneous generation and in his subsequent studies of the processes of fermentation. With the investigations of that distinguished French savant concerning the etiology of chicken-cholera, began our first positive knowledge of the definite relations of bacteria to disease in the animal kingdom. The next great step in this direction was gained when absolute proof was furnished that a certain bacillus is the specific cause of splenic fever (charbon, milzbrand) in sheep and other herbivora. Through his experiments concerning this latter disease, Prof. Robert Koch, of Berlin, first became widely known to the scientific world. Since these investigations, progress in knowledge of the etiological relations of bacteria to disease has been rapid and constant. Perhaps the greatest strides made are due to improvement of old and invention of new methods of research more than to any other cause; and to three men chiefly are we indebted for the greatest advances in this respect,—namely, Pasteur and Chauveau, of France, and Koch, of Germany. To the latter, however, we owe the greatest debt in this direction for the means which he has devised and perfected of isolating, with rapidity, ease, and certainty, different species of bacteria, and of following their cycles of development in pure cultures under varying circumstances. It is to the description, more or less in detail, of methods of examination, isolation, and cultivation of bacteria that most of this chapter will be devoted.

Although the knowledge of the characters and peculiarities of numerous species of bacteria which has been acquired in recent years has been considerable, it is not yet sufficiently comprehensive to render a satisfactory classification of these organisms possible. No classification thus far proposed is free from serious faults and objections. Perhaps that offered by W. Zopf is the most preferable in the main, because it is based upon the whole cycle of development of the species with which it deals, and by following it systematically in investigations of new as well as of known species we shall probably sooner be in possession of that full information necessary upon which an exact and entirely satisfactory classification must be based.

The classification of Cohn has been perhaps most generally followed. We introduce it here in order that the reader may comprehend the terms so frequently used.

TRIBE 1. GLÆOGENES.

Cells free or united in glairy families by an intercellular substance.

A. Cells free or united by 2 or 4:	
Cells spherical	Chroococcus, Näg.
Cells cylindrical	Synechococcus, Näg.
B. Cells united in glairy families, amorphous in state of repose:	
a. Cellular membrane, confounded with the intercellular substance:	
1. Cells without phycochrome, very small:	
Cells spherical	Micrococcus, Hallier.
Cells cylindrical	Bacterium, Duj.
2. Cells with phycochrome larger:	
Cells spherical	Aphanocapta, Näg.
Cells cylindrical	Aphanœtice, Näg.
b. Intercellular substance formed of several membranes enclosed one within the other:	
Cells spherical	Glœocapsa, Kg.
Cells cylindrical	Glœoethiece, Näg.
C. Cells united in glairy families of definite form:	
a. Families of a single layer of cells disposed in plates:	
1. Cells in fours forming a plane surface	Merismopedia, Meyen.
2. Cells without regular arrangement, forming a curved surface:	
Cells spherical, families with reticulated rupture	Clathrocystis, Henfr.
Cells cylindrical, cuneiform, families divided by constriction	Collosphærium, Näg.
b. Families with several layers of cells united in spherical corpuscles:	
1. Number of cells determined:	
Cells spherical, colorless, arranged in fours	Sarcina, Goods.
Cells cylindrical, cuneiform, with phycochrome, without regular arrangement	Gomphosphæria, Kg.
2. Number of cells very great and indeterminate:	
Cells colorless, very small	Ascococcus, Billr.
Cells colored by phycochrome and larger	Polycistis, Kg. Coccochloris, Spr. Polycoccus, Kg.

TRIBE 2. NEMATOGENES.

Cells disposed in filaments.

A. Filaments not branched:	
Filaments free or interlaced:	
1. Filaments cylindrical, colorless, articulations not very distinct:	
Filaments very slender, short	Bacillus, Cohn.
Filaments very fine, long	Leptothrix, Kg.
Filaments larger, long	Beggiatoa, Trev.

2. Filaments cylindrical, with phycochrome, the articles well defined, without cellular reproduction	Hypheothrix, Kg. Oscillaria, Bosc.
3. Filaments cylindrical, articulated, with conidia:	
Filaments colorless	Crenothrix, Cohn.
Filaments with phycochrome	Chamæsiphon.
4. Filaments spiral, without phycochrome:	
Filaments short, light, sinuous	Vibrio, Ehr.
Filaments short, spiral, rigid	Spirillum, Ehr.
Filaments long, spiral, flexible	Spirochæte, Ehr.
With phycochrome:	
Filaments long, spiral, flexible	Spirulina, Link.
5. Filaments in chaplet:	
Filaments without phycochrome	Streptococcus, Billr.
Filaments with phycochrome	Anabæna, Borg. Spermosira, Kg.
6. Filaments flagelliform, slender	Mastigothrix, etc.
b. Filaments united into glairy families, by intercellular substance:	
1. Filaments cylindrical, colorless	Myconostoc, Cohn.
2. Filaments cylindrical, with phycochrome	Chthonoblastus. Limnoclide, Kg.
3. Filaments in chaplet	Nostoc, etc.
4. Filaments flagelliform, slender	Rioularia, etc.
B. Filaments with false ramification:	
1. Filaments cylindrical, colorless	Cladothrix, Cohn. Streptothrix, Cohn.
2. Filaments cylindrical, with phycochrome	Calothrix, Ag. Scytonema, Ag.
3. Filaments in chaplets	Merizomyria, Kg.
4. Filaments flagelliform, slender towards the extremity	Schizosiphon, Kg. Geocyclus, Kg.

According to Zopf, "from the latest investigations concerning algæ and bacteria there can be no doubt that between certain representatives of both these groups there is a perfect morphological homology. This homology might easily lead to the classification of the bacteria as forms of algæ which contain no chlorophyl, as Cohn, Kirchner, and Van Tieghem have already done on the basis of a very limited knowledge of morphological relations. Although such a classification might not appear to be undesirable, yet it must frequently be misleading; for, as the latest investigations show, our knowledge of the development of algæ is still not perfect, and on that account the present classification will probably, through the close study of its various representative forms from this new point of view, suffer here and there more or less important modifications."

The difficulty of even using a proper nomenclature in the descriptions of bacteria is still frequently experienced on account of variation in forms during the cycle of development of many individual species. Doubt has, consequently, not infrequently arisen as to what technical term should be employed for the conveyance of an exact idea of the object under view, and some uncertainty and misconception of the exact meaning of writers discussing or reporting bacteriological observations have sometimes been unavoidable. It should be premised that in the subsequent pages the tern "bacteria" in a general sense may apply to any or all of the forms includec in the foregoing or following classifications. In this sense it is intended t(be synonymous with the general French term "microbe," exclusive of fung or moulds.

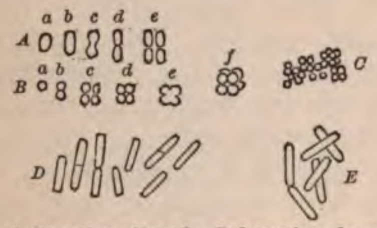
Rabenhorst presented the following classification, which has been adoptec by Flügge:

SCHIZOMYCETES.

Cells round or ovoid.	Isolated, or in chains, or united in amorphous gelatinous families						Micrococcus.
	Forming gelatinous families of definite form.	Colonies solid, filled with cells.	In large numbers, in irregular colonies				Ascococcus.
			In small but definite numbers, in regular groups				Sarcina.
		Colonies with simple layer of cells at the periphery					Clathrocystis
Cells cylindrical.	Short, isolated, or in small groups loosely united, or in irregular gelatinous families						Bacterium.
	Long, cylindrical, forming filaments.	Filaments isolated, interlaced, or in bundles.	Without ramifications.	Straight filaments.	Short, distinctly jointed.		Bacillus.
					Long, indistinctly jointed.	Very thin	Leptothrix.
						Thicker	Beggiatoa.
				Wavy, or in spirals.	Short, rigid		Spirillum (Vibrio).
					Long, flexile		Spirochætæ.
			Pseudo-ramifications				Streptothrix. Clathrothrix
		Cells included in roundish gelatinous masses					Myconostoc.

In the system proposed by Zopf the imperfectly-known bacteria are not included. Those concerning which we are in possession of more or less exact knowledge are placed in the following four great groups:

Fig. 1.



A, formation of tetrads; *B*, formation of sarcina; *C*, formation of tetrads and sarcina in irregular masses; *D*, *E*, irregular groups of short rods. (After Hüppe.)

1. Coccaceæ. They include only cocci and chains of cocci.
   Genus: Leuconostoc.
2. Bacteriaceæ. They include four forms of development:—Cocci, bacteria (short rods), bacilli (long rods), and leptothrix forms (filaments). The latter contain no subdivisions from one end to the other, and no typical appearance of spirilli.
   Genera: Bacterium, Clostridium.
3. Leptothricheæ. They include cocci, short rods, filaments (which show division), and spiral forms.
   Genera: Leptothrix, beggiatoa, crenothrix, phragmidiothrix.
4. Cladothricheæ. They include cocci, short rods, filaments, and spiral filaments. The filaments present pseudo-branches.
   Genus: Cladothrix.

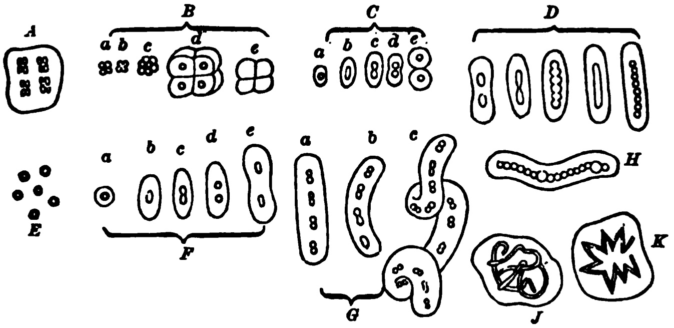
This classification has been followed by Crookshank, and we shall, with some slight modification of order, introduce here for convenient use a short abstract of the elaboration of this system which the latter author has published.

GROUP 1. COCCACEÆ.

Genus I. Micrococcus.—Division in one direction and the cocci after division remain isolated or aggregated in irregular clumps or masses. (Photo. No. 1.)

M. pyogenes aureus (staphylococcus pyogenes of Rosenbach), round; yellow growth. Pathogenic.

FIG. 2.



Capsule formation of tetrads, *A*; of sarcina, *B*; of single and double micrococci, *C*; of streptococci, *D*; multiplication and capsule formation of micrococci (leuconostoc), *E* to *H*; spiral forms within gelatinous capsule or zoogloea of myconostoc, *J*, *K*. (After Hüppe.)

M. pyocyaneus: ellipsoidal; produce pyocyanin, green-blue or blue pus. Pathogenic.

M. choleræ gallinarum (or bacterium of fowl-cholera,—they should be classed among bacteria): mono- and diplococci, the latter in fig. 8 form; 2. to 3. μ in diam. Pathogenic.

M. prodigiosus: round; in masses, rose- or blood-red; single, without color; 0.5 to 1. μ in diam.

M. septicæmiæ in rabbits: ellipsoidal (should properly be classed as bacteria); 0.8 to 1. μ long. Pathogenic.

M. pyæmiæ of rabbits: mono- and diplococci; 0.25 μ in diam. Pathogenic.

M. of progressive suppuration in rabbits: 0.15 μ in diam. Pathogenic.

M. auranticus: oval; single or diplo; orange growth; 1.5 μ long.

M. chlorinus: green growth.

M. violaceus: violet-blue color; ellipsoid.

M. luteus: yellow growth; ellipsoid.

M. fulvus: rusty-red growth; 1.5 μ in diam.; frequently diplo.

M. hæmatodes: red growth; human sweat of axilla; and in other localities.

Genus II. Streptococcus.—Division in one direction only; individual cocci remain united together to form chains or chaplets. (Fig. 4, *A*.)

Streptococcus pyogenes (chain cocci in pus; c. of pyæmia): cocci single and in chains. Pathogenic.

Str. coc. erysipelatis: 0.4 to 0.3 μ in diam.; round. Pathogenic.

Str. coc. diphtheriticus (Oertel): oval; 0.35 to 1.1 μ; mono, diplo, and strepto. Pathogenic (?).

Str. coc. of progressive necrosis in mice (Koch): 0.5 μ. Pathogenic.

Str. coc. bombycis in *flacherie*: oval; mono, diplo, and strepto; 0.5 μ. Pathogenic.

Str. coc. vaccinæ: mono, diplo, and strepto; 0.5 μ. Pathogenic (?).

Str. coc. perniciosus (Parrot disease). Pathogenic.

Str. coc. ureæ (m. ureæ or bacterium ureæ): mono- or streptococci or short rods; may have capsules.

Genus III. Merismopedia.—Cocci; division in two directions only. (Fig. 1, *A*, and Fig. 2, *A*.)

Gonococcus: mono, diplo, and tetra; 0.83 μ in diam. Pathogenic (?).

M. tetragenus: tetrads held together in hyaline envelope or capsule (often associated with phthisis).

Genus IV. Sarcina.—Cocci; division in three directions or less. (Fig. 1, *B*, *C*, and Fig. 2, *B*.)

Sarcina ventriculi: 4. μ in diam.; united in cubes of 8 or multiples of 4; cells greenish or yellowish red.

Sarcina intestinalis (Zopf): very regular in form; never in such large packets as in the stomach.

Sarcina lutea: mono, diplo, tetra, and cube-groups, etc.; individuals of a tetrad may themselves be divided; canary-yellow growth.

Sarcina urinæ (Welcker): 1.2 μ in diam.; cubes or multiples thereof. (Sometimes in the bladder.)

Sarcina littoralis (in sea-water): 1.2 to 2. μ in diam.; cells contain one to four sulphur granules.

Sarcina Reitenbachii (on putrefying water-plants): 1.5 to 2.5 μ in diam.; colorless cell-wall lined with rose-red layer of plasma.

Sarcina hyalina (in marshes): 2.5 μ in diam.; nearly colorless groups of 4 to 24 cocci.

GROUP 2. BACTERIACEÆ.

Genus I. Bacterium.—Short rods, with blunt, rounded or pointed ends; individuals may be indistinguishable from cocci; single, diplo, or united to form threads (strepto); endogenous spores unknown. (Fig. 1, *D*, and Fig. 4, *B*, *C*.)

Bacterium ureæ (m. ureæ): oval; 1.25 to 2. μ long; single or in chains and short rods; tendency to form capsules and zooglœa masses. Produce *ammoniacal fermentation*, converting urea into urea carbonate.

Bact. aceti: cocci, bacteria, bacilli, filaments, and zooglœa masses; converts the alcohol in wine and in other fruit juices into vinegar.

Bact. Pasteurianum (Hansen): morphology similar to bact. aceti, but cells contain a starch-like material made blue by iodine; occurs in beer wort.

Bact. Zopfii (Kurth): cocci, bacilli, and wavy filaments, which again break up into cocci (in intestine of fowls).

Bact. merismopedioides (Zopf): filaments, 1. to 1.5 μ thick, subdivide into long and short rods, finally into cocci; the latter divide in one and two directions forming tetrads grouped in 64 or more; these again form rods and filaments (in putrid water).

Bact. pneumoniæ crouposæ; cocci round and oval, and rods; single or in pairs; tendency to form capsules when naturally growing in animal tissues, but not in artificial cultures. Pathogenic (?).

Bact. Pflügeri phosphorescens (Ludwig); round cocci and threads.

Bact. isuthinum (Zopf): short and long rods breaking up into cocci. Colonies intense violet.

Bact. synxanthum (bact. of yellow milk): oval, motile, 0.7 to 1. μ long, much resembles bact. termo; lemon-yellow growth.

Bact. of diphtheria in man and pigeons: cocci or short, thick rods. Pathogenic (?).

Bact. Panhistophyton (nosema bombycis, corpuscules du ver à soie): oval cocci; 2. to 3. μ long and 2. μ wide; single or diplo or short rods, 2.5 μ thick and twice as long (cause of pébrine). Pathogenic.

Genus II. Leuconostoc.—Cocci and short rods; spore formation present in cocci; thick, tough, gelatinous capsules.

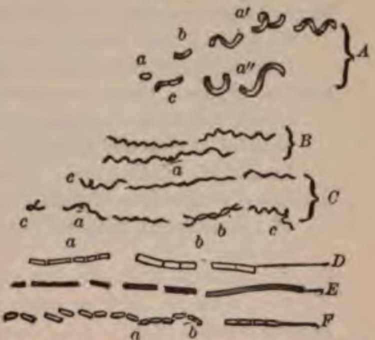
Leuconostoc mesenterioides (Cienkowski) (gomme de sucrerie, froschlaichpilz, frog-spawn fungus): cells single, in chains or zooglœa; cocci elongate into rods; latter segment and form endogenous spores; exceedingly rapid development,—49 hectolitres of molasses containing 10 per cent. of sugar was converted within twelve hours into a gelatinous mass.

Genus III. Bacillus.—Cocci and rods, or rods only; may form straight or twisted threads; endogenous spore formation. (Fig. 3, *D*, *E*, *F*.)

Bacillus subtilis (hay bacillus): cylindrical rods, and threads, rounded ends with flagella attached, motile; 0.6 μ thick and about 6. μ long; rapid multiplication in sufficient nourishment, by transverse fission; slower multiplication by endogenous spore formation in impoverished media. If the multiplication be rapid the filament may include cocci and short rods; aërobic; whitish or yellowish-white growth.

Bacillus anthracis (cause of splenic fever of sheep, etc., charbon, milzbrand): rods 5. to 20. μ long and 1. to 1.25 μ thick; long chains of cocci and rods; the ends are square, have no flagella, are non-motile; multiply by fission and endogenous spore formation; aërobic. Pathogenic.

FIG. 3.



A, spirillum undula; *B*, recurrens spirochæte; *C*, spirochæte of the mouth; *D*, anthrax-bacilli,—*a*, of mouse, *b*, of rat; *E*, bacillus of malignant œdema; *F*, bacillus subtilis. (After Koch.)

Bacillus tumescens (Zopf): cocci, short and long rods; endogenous spore formation; forms gelatinoid masses.

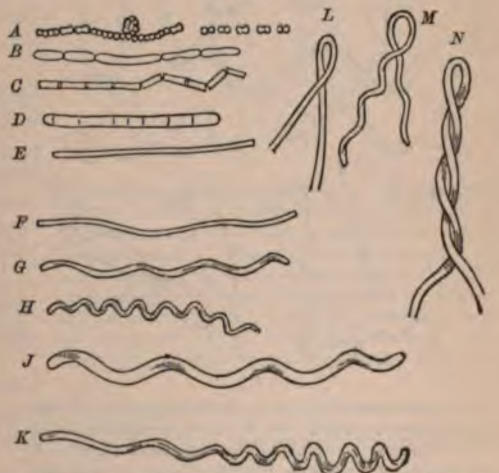
Bacillus megaterium (De Barry): large róds, 2.5 μ thick and 4 to 6 times as long; usually slightly curved; multiply by fission and endogenous spores; may form irregular chains; motile.

Bacillus Fitzianus (Zopf): cocci, short and long rods and filaments; causes fermentation and *produces ethyl alcohol*; accompanies hay bacillus.

Bacillus tuberculosis (Koch): very fine rods, 2. to 4., occasionally 8. μ long; straight or slightly curved, often beaded, rounded at ends; single, in pairs or bundles; sometimes associated with granules stained in some manner; non-motile; spore formation within animal body. Cause of tuberculosis. (Photo. No. 3.)

Bacillus lepræ (Hausen): slender rods 4. to 6. μ long, less than 1. μ thick, sometimes pointed at ends; some motile, others not; may contain spores and present beaded appearance; resembles bacillus tuberculosis. Probably the cause of leprosy.

FIG. 4.



A, streptococci; *B*, *C*, strepto-bacteria; *D*, jointed thread; *E*, unjointed thread; *F*, wavy thread; *G*, *H*, *J*, *K*, spirilli more or less stiff; *L*, *M*, *N*, spirilli more or less flexile and looped. (After Hüppe.)

Bacillus mallei (bacillus of glanders): fine rods 2. to 6. μ long. Cause of glanders.

Bacillus cyanogenus (bacillus of blue milk): rods single or in pairs, 2.5 to 3.5 μ long; multiply by fission; they may form chains; may be surrounded by capsule.

Bacillus acidi lactici: short and long rods 3. to 4. μ long, also filaments; endogenous spores. Causes acidity of milk if not above 45.5° C. (not identical with bact. acidi lactici).

Bacillus œdematis maligni (Koch) (vibrion septique of Pasteur): rods 3. to 3.5 μ long and 1. to 1.1 μ thick; usually in pairs; rounded ends; may form filaments, straight or slightly curved; motile; anaërobic; spore formation. Pathogenic. (Fig. 3, *E.*)

Bacillus septicæmiæ of mice (Koch): very small and slender rods, 0.8 to 1. μ long and 0.1 to 0.2 μ thick; single, in pairs or in chains of 3 or 4 links; no filaments; often collected together in masses; spore formation.

Bacillus typhosus (bacil. of typhoid fever, Eberth, Gaffky): rods, 0.2 μ thick and length 3 to 4 times as great; filaments upwards of 40. μ in length; or short rods, rounded at ends and sometimes constricted in the middle. Motile; doubtful spore formation. Pathogenic. (Photo. No. 2.)

Bacillus diphtheriticus of man: rods 2. to 5. μ or more long, twice as thick as bacillus of tuberculosis; may be linked in pairs or chains. Found also in calves and pigeons.

Bacillus malariæ (Klebs): rods 2. to 7. μ long; may develop spiral filaments; spores may form at centre, ends, or sides; aërobic. (Probably not the cause of malaria.)

Bacillus dysodes (Zopf): cocci, long and short rods; spore formation; causes a destructive fermentation of bread; may accidentally accompany yeast.

Bacillus Hausenii (Rasmussen): rods 2.8. to 6. μ long, 0.6 to 0.8 μ thick; spore formation; growth chrome-yellow; volatile aromatic gas of fruit-like odor given off.

Bacillus erythrosporus (Cohn): rods, with spore formation, and filaments; motile.

Bacillus ruber (Frank): minute rods, single, in pairs and in fours; growth brick-red.

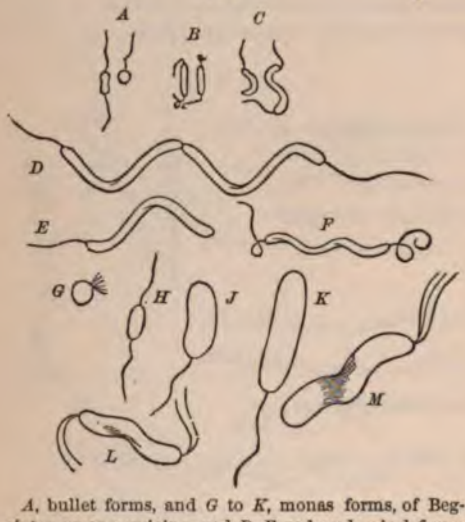
Genus IV. Clostridium.—Same as bacillus, but spore formation takes place in characteristically enlarged rods.

Clostridium butyricum (bacillus amylobacter, bacillus butyricus, bacillus of butyric acid fermentation—Prazmowski): rods less than 1. μ thick, 3. to 10. μ long, motile, anaërobic; develops into long unjointed filaments; both rods and filaments may be slightly curved; spore formation by swelling of the rod or filament at the point of its location; changes lactic acid of milk into butyric acid and causes ripening of cheese; also active in formation of sauer-kraut; cells sometimes yield iodine reaction for starch.

Clostridium polymyxa (Prazmowski): filaments, rods of variable length, cocci, and spores; cause of certain fermentations; some yield weak iodine reaction for starch.

Clostridium of symptomatic anthrax (blackleg, quarter evil, rauschbrand, charbon symptomatique—Arloing): rods, with rounded ends, motile, usually containing an end spore. In cultures develop into chains of rods and cocci.

FIG. 5.



A, bullet forms, and *G* to *K*, monas forms, of Beggiatoa roseo-persicina, and *D, E*, rod and spiral forms of cladothrix, crenothrix, and beggiatoa (Zopf); *B*, bacterium; *C*, spirillum undula (Koch); *F*, spirillum volutans (Cohn); *L, M*, ophido monas forms (Warming): with flagella.

Genus V. Vibrio.—Filaments, screw form, in long or short turns; spore formation. (Fig. 4, *F, G.*)

Vibrio rugula (Müller): rods and filaments 0.5 to 2.5 μ thick, 6. to 16. μ long; motile, with an end flagellum, mostly axial rotation and progressive motion; rods simply curved or slightly spiral; filaments more or less spiral; one end may enlarge and a round spore form therein.

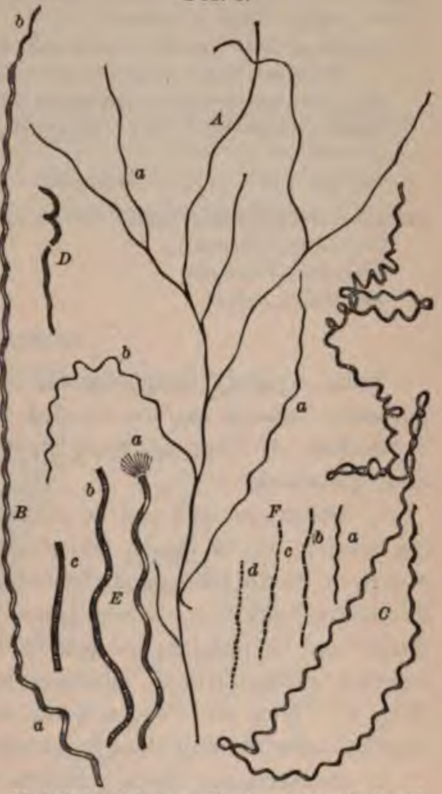
Genus VI. *Spirillum.*—Filaments more or less spiral, rods curved or partly spiral; spore formation doubtful or unknown. (Fig. 3, *A*, *B*, *C*; Fig. 4, *G* to *N*; Fig. 5, *C* to *F*; Fig. 7, *H*, *J*. Photo. Nos. 11, 12, 13.)

Spirillum choleræ Asiaticæ (comma bacillus of Koch): curved rods, spirilli, and filaments; the rods about half as long as the average tubercle-bacillus, their thickness about $\frac{1}{5}$ to $\frac{1}{3}$ their length; single or attached end to end in pairs so as to form shape of the letter S; or they may be attached in chains of three or more; multiply by lengthening and transverse fission. In old cultures there are often spherical bodies of variable size, isolated or connected with the rods, and they are regarded by most observers as involution forms; by Ferran and Hüppe they are looked upon as vegetative forms endowed with a greater power of resistance,—the former names them oogonia, the latter arthrospores. Nearly all observers admit absence of spore formation; exceedingly motile with more or less rounded ends. Pathogenic. (Fig. 7, *H*, *J*. Photo. No. 13.)

Spirillum plicatile (marsh spirochæte—Ehrenberg): thin spiral filaments with close coils, many μ long; besides the shorter regular curves, they have also coils of greater length and depth; blunt ends, rapid movement. In cultures the filaments divide into short or long rods, or ultimately cocci, observed in drop cultures and in stained preparations.

Spirillum Obermeieri (spirochæte Ob.; spr. of relapsing fever—Obermeieri): exceedingly thin filaments resembling spir. plicatile; length 16. to 40. μ; motile; occurs in blood of relapsing fever during the access. (Fig. 3, *B*.)

FIG. 6.



Cladothrix dichotoma (Zopf). *A*, branched, with longer (*a*) and with shorter (*b*) screw-like curves; *B*, spirillum, one of whose ends (*a*) shows greater curves than the other (*b*); *C*, longer spirochæte-like branch, with loops; *D*, a portion of branch, with close and with loose coils; *E*, spirilli,—*a*, unjointed, *b*, appearance of joints, longer, *c*, shorter; *F*, spirochæte form, at *a* unjointed, at *b* to *d* schematic joints, at *c* into short rods, at *d* into cocci. (Hüppe.)

GROUP 3. LEPTOTHRICHEÆ.

Genus I. *Leptothrix.*—Threads articulated or unarticulated; successive subdivision of cells not continuous; cells sulphurless. (Fig. 4, *E*.)

Leptothrix baccalis (Robin): long, thin threads, often united in thick bundles or felted together; are composed of long rods, short rods, and cocci.

Leptothrix gigantea (Miller): long rods, short rods, and cocci, which vary in thickness often in the same thread.

Genus II. *Crenothrix.*—Threads articulated; cells sulphurless; habitat water.

Crenothrix kuhniana (Rabenhorst): cocci, rods, and filaments, which latter are colorless and may be club-shaped, and the ends are articulated and ensheathed; the sheath bursts, the cells are set free and develop into new threads.

Genus III. Phragmidiothrix.—Threads jointless; successive subdivision of cells is continuous; cells sulphurless; habitat water.

Phragmidiothrix multiseptata.

Genus IV. Beggiatoæ.—Threads unarticulated; cells with sulphur granules; habitat water. (Fig. 7, *A* to *E*.)

Beggiatoa alba (Vauch): cocci, rods, spirals, and threads; the protoplasm contains numerous highly-refractive granules, consisting of sulphur.

Beggiatoa roseo-persicina (Bacterium rubescens): cocci, rods, spirals, and threads.

Beggiatoa mirabilis (Cohn): the threads are filled with sulphur granules.

GROUP 4. CLADOTHRICHEÆ.

Genus I.—Cladothricheæ (Cohn): threads resembling those of leptothrix. (Fig. 6.)

Cladothrix dichotoma.

Cladothrix Foersterii.

Spherotillus natans.

MORPHOLOGY.

From a purely morphological stand-point, and simply for descriptive purposes, bacteria may be divided as follows: 1, Coccus; 2, Bacterium; 3, Bacillus; 4, Filament (straight, wavy, or spiral—long rod, vibrio, spirillum, spirochæte).

1. **Cocci** are spherical or ovoid forms: isolated (*monococci*); in pairs (*diplococci*); or in chains (*streptococci*); four together in the form of a square or rosette (*tetragoni*); in cubes or oblong packets of eight, sixteen, thirty-two, sixty-four, or multiples of either (*sarcinæ*). (Figs. 1, 2.) These forms may be isolated, grouped in irregular clumps, or in a mass, held together by a gelatinoid substance in which they are embedded (*zooglœa*). (Fig. 2.) They are frequently the subject of active Brownian movement, but they have no individual progressive motion. (Photo. No. 1.)

2. **Bacteria** are short cylinders of an average thickness equal to one-half their length, with square, blunt, rounded, or more or less spindle-shaped extremities, and various species may be provided with one or more extremely fine terminal motile cilia (*flagella*) which serve for the purpose of locomotion. (Fig. 5.) The bacteria may be single or double, united together end to end, or they may form chains with three or more links; and the terms "mono," "diplo," and "strepto" may be applied to them, as in the case of cocci. They may be motile or non-motile. (Fig. 1, *A*, *D*; Fig. 4, *B*.)

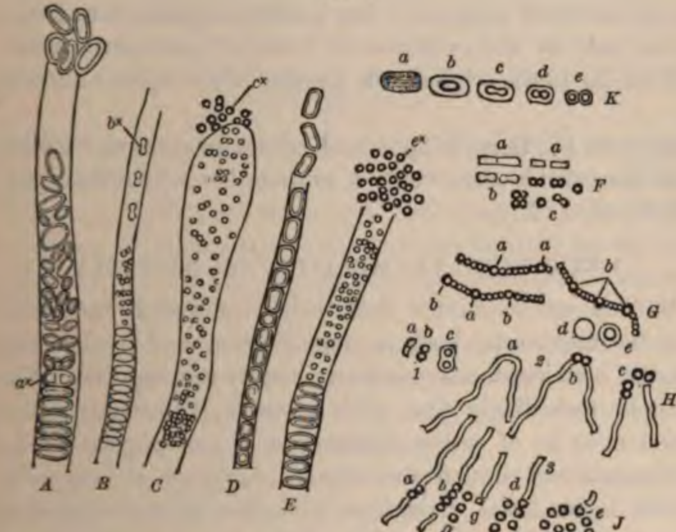
3. **Bacilli** are long cylinders of variable length, but longer than the bacteria. Their extremities may be square, rounded, or pointed, and when motile they also may be furnished with one or more cilia. They may be joined together end to end, forming chains or jointed filaments, and these filaments may be straight or wavy, motile or non-motile. (Fig. 1, *E*; Fig. 3, *D*, *E*, *F*; Fig. 5, *B*.)

4. **Spirilli** fully developed consist of a somewhat stiff filament, more or less regularly coiled in the form of a spiral, and more or less indistinctly jointed or not; the extremities may be provided with cilia, and progressive motion is chiefly secured by an axial rotation. The smallest developmental

form of the spirillum is usually a short, slightly curved, more or less pointed bacillus, and these latter forms may be united in pairs, end to end, representing more or less perfectly the outline of the letter S; united end to end in greater numbers they form a longer or shorter, more or less spiral, wavy line. (Fig. 3, *A, B, C;* Fig. 4, *G* to *N;* Fig. 5, *C* to *F;* Fig. 7, *H, J;* Photo. Nos. 11 to 13.)

The *cocci increase* in number by elongation of the diameter in one direction until it is about double the former length, then constrict in the middle, and, by continuation of the process of transverse fission, form diplococci, streptococci; when fission occurs in two directions, tetragoni; and when fission is in three directions, sarcinæ,—the final result of the process of fission resulting in the formation of entirely separate and distinct forms more or less identical with those from which they sprang.

FIG. 7.



A, B, C, D, E, beggiatoa tubes, showing macro- and microgonidia; *F,* bacterium Zopfii, —formation of arthrospores, *c; G,* streptococci, showing larger cells, *a* and *b,* regarded by Van Tieghem as cystic, by Du Bary and Hüppe as arthrospores; *H, J,* spirilli of Asiatic cholera, also showing arthrospores (Hüppe, or gonidia of Ferran); *K,* process of arthrospore formation in bacterium. (After Hüppe.)

The process of *multiplication* of the *bacterium* is also by transverse fission. The bacterium first elongates, then becomes constricted in the middle, and finally forms in this manner a diplobacterium or a streptobacterium. The member may remain thus attached, or the process of fission may continue, to end in separation.

The process of *multiplication* of *bacilli* is in some species twofold; in others it is simple. The simple process of multiplication is also by transverse fission. The rod may elongate and divide into two or more by the process of fission, which may or may not extend to complete separation of the joints and a new formation of isolated bacilli similar to those whence they sprang. The other process of multiplication consists in the endogenous formation of one or more spores in the interior of the rod, usually after it has more or less elongated and slightly increased in thickness, particularly at the point where the spore is to be formed. The location of the spore may be at the end or near the middle of the rod. The spore consists of a more or less oval, glistening, highly refracting, resistant body (Fig. 7, *K*, *b*), which usually when fully formed occupies the whole diameter of the cylinder, and indeed at this point the diameter of the latter may be somewhat increased. After the spore is formed, the protoplasm of the cylinder retrogrades, the delicate limiting membrane disappears, and the spore is set free to undergo its cycle of development. Development from the spore may take place from one of its more or less pointed extremities or from the side of the spore, and by the extrusion of a bud of protoplasm which grows into a rod similar to the original, the remains of the spore ultimately disappearing.

According to Du Bary, Hüppe, and others, micrococci, bacteria, bacilli, and spirilli have still another mode of propagation,—by arthrospores. (See Fig. 7, *F*, *G*, *H*, *J*, *K*.)

METHODS OF EXAMINATION OF BACTERIA.

The bacteria are so minute that their examination taxes the utmost powers of the microscope both in magnification and in definition; consequently, the best instruments in every respect are required. The microscope must be exceedingly firm, with a steady, stiff stage; the sub-stage illumination must be of such a character as to pass a great flood of light upward, through and around the object. Although it is possible to see many of the larger forms of bacteria with the old style dry objective as weak as a quarter, if it have excellent defining and resolving powers, yet such a lens is at best an extreme tax on the eye, and, moreover, there are many of the minute forms which are practically invisible under such a lens. The immersion objectives are essential for bacteriological investigations, and those of a homogeneous oil-immersion system are greatly to be preferred. The focal length should be from a twelfth to an eighteenth of an inch, with a good working distance and high angular aperture. Among the best of the cheaper lenses of such a character are the Leitz twelfth oil-immersion and Reichert fifteenth oil-immersion. Among the best of the high-priced objectives, those of Powell and Leland oil-immersion sixteenth and of Zeiss or Spencer oil-immersion twelfth or eighteenth may be preferred. The very best lens which can be obtained at the present time for bacteriological work is the new Zeiss system oil-immersion apochromatic. It is quite expensive, but in resolving and defining power is a wonderful advance over the best oil-immersion lenses heretofore made. It may be stated that there are also a few water-immersion lenses of a short focus and high angular aperture which can be used with more or less satisfaction.

The best sub-stage illuminating apparatus above referred to is that devised by Abbe, which is somewhat expensive. But there are numerous cheaper modifications of this apparatus which work satisfactorily. Perhaps the simplest form, constructed after the design of the writer, is that which consists essentially of an upper hemispherical lens a half-inch in diameter and a lower biconvex of two inches focus and a diameter of an inch, suitably mounted in a cylindrical holder which slides up and down below the stage of the microscope. Such an apparatus can be made in this country for the sum of six dollars, and gives satisfaction in its use.

It should be stated in connection with the sub-stage illuminating apparatus that in the examination of bacteria it is desired to obtain the greatest flood of light possible, quite contrary to the use of the sub-stage condenser for ordinary histological purposes.

METHODS OF PREPARATION OF BACTERIA FOR MICROSCOPIC EXAMINATION.

With the best lenses bacteria can be examined with some satisfaction in their native condition,—that is to say, without staining. They may be examined fresh in fluids or in thin films dried upon the under surface of a thin cover-glass. For such an examination the light of the sub-stage condenser must be toned down by diaphragms much in the same manner as for ordinary histological work.

Examinations of unstained bacteria are, however, more or less unsatisfactory, and, as a rule, are exceedingly trying to the eyes. Moreover, examination of the smallest forms taxes greatly the resolving and defining powers of the very best lenses at present known.

In later years it is regarded as essential to color bacteria artificially in preparing them for close study. They may be examined in fluids, or the material containing the bacteria may be spread in a thin film upon the surface of a thin cover-glass. The latter method is the one at present most generally in use. Furthermore, bacteria may be examined directly in the tissues which contain them. Here also they may be observed in the natural state—that is, free from artificial coloring—or they may be subjected to a process of staining. In this case it is usually necessary to make thin sections of fresh tissues by means of the freezing microtome, or sections of tissues which have been previously hardened. For examination of bacteria in sections of hardened tissue, the best hardening agent is absolute alcohol; for bacteria in tissues without staining, it is necessary to make use of the clearing effect of some of the strong acids or alkalies, such as glacial acetic acid, pure or slightly dilute, or a strong solution of caustic potash or soda.

In examination of a film upon a cover-glass, it is usually necessary to employ some means of fixing the film firmly thereon. This is conveniently done in the case of albuminous fluids by employing heat. The cover-glass should be held between the thumb and finger by the edges, and quickly passed through the flame of the spirit-lamp, or Bunsen burner turned very low, care being taken to have the film side uppermost. The usual procedure is to pass the cover-glass quickly through such a flame three times successively in as many seconds. The sensibility of the fingers will prevent any danger of overheating. It is found that by employing heat in such a manner the bacteria are not injured for staining purposes and are but little, if at all, altered in form.

METHODS OF STAINING.

It has been known for a number of years that certain solutions of carmine, particularly the ammoniacal, possess the property not only of staining various histological elements of tissues, but also of tingeing, more or less, certain kinds of micrococci; and it was later learned that hæmatoxylin possesses a similar capability. In the systematic study of methods of staining tissues for histological examination, it was found that numerous aniline dyes possess the power of staining intensely various forms of bacteria.

To Weigert, Ehrlich, and Koch we are indebted for the first systematic employment of the aniline dyes for the special tingeing of bacteria. This special use of the latter coloring-agents is based upon two fundamental principles: the relative affinity of the various histological elements for these dyes, and the persistency with which they retain them. It has been found that for most of the aniline colors the selective affinity of bacteria is greater than that of the other histological elements, and that the persistency with which the bacteria resist decoloration after staining is also greater.

Aniline dyes may be divided into two principal groups,—the acid and the basic; that is, those which are simple acids or acid salts, and those which are simple bases or alkaline salts. The acid aniline colors may be subdivided further into four classes:

1. Fluorescine: *e.g.*, fluorescin, eosin.
2. Nitro-substances: *e.g.*, martius yellow, picric acid, aurantia.
3. Sulphur acids: *e.g.*, tropæolin.
4. Primary acid colors: *e.g.*, rosalic acid, alizarin, purpurin.

Of the basic aniline colors, the following are most generally used: fuchsin (hydrochlorate of rosanilin); methyl-violet (hydrochlorate of trimethyl-rosanilin); gentian-violet; methyl-blue; vesuvin. Less frequently; methyl-green, cyanin, safranin, magdala, dahlia. Of these, especially the violets (methyl-violet, gentian-violet, iodine-violet, dahlia) possess a species of double coloring power which is sometimes made use of. Methyl-violet, for example, colors an amyloid substance, not violet,—like the bacteria and the nuclei,—but red; methyl-green colors the nuclei and bacteria green and the amyloid substance violet.

The intensity of the coloring and the persistence with which it resists decolorization vary sometimes to a considerable extent, according to the nature of the color and to the species of bacteria. It has been found that alcohol, glycerin, and acetic and other acids possess in high degree the faculty of discharging aniline colors from all the elements, including the bacteria, which have absorbed them.

Theoretically the brown anilines (vesuvin, Bismarck-brown, aniline-brown) possess in the highest degree the power of selective affinity and at the same time resist the action of decolorizing agents. They are also for the ordinary photographic plates most useful for photographic purposes. Next after the browns, in selective affinity and persistence with which the color is retained, come, in regular order, fuchsin, methyl-violet, gentian-violet, methyl-blue. It must be remarked, however, with respect to this order, that certain dyes have been found by experience to be preferable for staining certain species of bacteria.

EMPLOYMENT OF ANILINE DYES FOR THE DEMONSTRATION OF BACTERIA.

The basic aniline colors are most commonly used, and are conveniently prepared in the following manner:

1. *Concentrated Watery Solutions.*—These are used either in full strength or in solutions diluted with distilled water. The concentrated solution is made by boiling the required quantity in distilled water; after settling, the supernatant fluid is drawn off and kept for use. It is found necessary to freshly filter these watery solutions.

2. *Concentrated Alcoholic Solutions.*—Commonly about twenty to twenty-five parts of the color-material to one hundred parts of alcohol are sufficient for saturation. These concentrated alcoholic solutions are not commonly used in full strength, but generally after dilution with a certain quantity of water. Five or six drops of the concentrated alcoholic solution may be added to a small watch-glass of distilled water to make a proper fluid for immediate use.

3. Vesuvin, Bismarck-brown, aniline-brown, are usually not employed in alcoholic solutions, for when water is added precipitation occurs. If a concentrated solution is desired for use, it is commonly made in equal parts of glycerin and water.

4. *Alkaline Solutions.*—(*a.*) Weak alkaline solution of Koch: 1 c.cm. of concentrated alcoholic solution of methyl-blue; 200 c.cm. of distilled water; 2 c.cm. of a ten-per-cent. solution of caustic potash.

(*b.*) Strong concentrated solution of Loeffler: 33 c.cm. of concentrated alcoholic solution of methyl-blue; 100 c.cm. of a weak solution of caustic potash (1 part to 10,000 parts of water).

5. *Aniline Water.*—5 c.cm. of pure aniline oil are shaken for a moment or two with 100 c.cm. of distilled water and allowed to stand for five minutes or so; the emulsion is then filtrated through filter-paper previously moistened with distilled water. The filtrate should be clear; if not so, it should be again filtrated. It is best to prepare this aniline oil-water freshly, for it becomes darkened by the action of light and a fine deposit occurs. This precipitation may, however, be impeded by the addition of

five to ten per cent. of strong alcohol. The aniline water thus prepared is used as a solvent for the various aniline colors, most commonly fuchsin, methyl-violet, and gentian-violet; and it is strongly recommended that when the most satisfactory results are desired these aniline-water color-mixtures should be made freshly.

6. Instead of aniline, toluidin may serve as a menstruum; so also turpentine, carbolic acid in five per cent., and ammonia in one-half per cent., or even borax solutions.

For double staining, carmine or hæmatoxylin may be used for coloring the histological elements of tissues,—the first for bacteria stained blue or violet, the latter for those stained red; and of the aniline colors, especially eosin, in dilute alcoholic solution, may be used in preparations where the bacteria are stained blue or violet. Instead of the ordinary carmine solution, picro-carmine may be used for staining preparations of bacteria tinged blue: it colors the nuclei an intense red, the fibrilli of the connective tissues a light red, and the protoplasm of the cellular body more or less yellow,—so that really a threefold coloration is the result.

One of the best hæmatoxylin solutions for this purpose is constituted as follows:

Hæmatoxylin	2 parts;
Alcohol	100 parts;
Distilled water	100 parts;
Glycerin	100 parts;
Alum	2 parts.

This hæmatoxylin solution stains cocci and several forms of bacilli, and at the same time also to some extent their zooglœa masses. When picric acid enters into the staining employed, in order to preserve the yellow tone of this dye it is necessary after staining to treat the preparations with alcohol containing picric acid, and to use dammar varnish as a permanent mounting-medium.

COMMON METHOD OF USING STAINING SOLUTIONS FOR DEMONSTRATION OF BACTERIA.

Cover-Glass Preparations.—(*a*). The fresh fluid containing the bacteria to be examined is spread out over the surface of a perfectly clean cover-glass and allowed to evaporate partially. After the drying has progressed somewhat, but while the film is still moist, a drop of the dilute color-solution selected is placed upon the film, and the cover-glass is inverted upon an ordinary object-slide. The bacteria, surrounded by the staining fluids, may be directly observed under the high power of the microscope, a certain amount of the coloring matter having been quickly absorbed by the bacteria. This method allows the motile bacteria to be observed while in motion, and in certain cases is advantageous to employ.

(*b*.) The dried film fixed upon the surface of the cover-glass by the agency of gentle heat after the manner already described is best stained by placing a drop or two of the color-fluid upon the film and inverting the cover-glass upon an object-slide. After the film has thus been subjected for a few moments to the action of the color-solution, the latter is drawn off by capillary attraction in the following manner: a drop of sterilized distilled water is placed at one edge of the cover-glass, and at the opposite edge a small piece of bibulous paper is placed in contact with the color-fluid, which is thus drawn off, and at the same time replaced by the clear distilled water at the side. The same method may be made use of for removal of the coloring fluid in the case of the bacteria examined before complete desiccation, as above described, and is especially useful where films of non-albuminous liquids cannot be fixed upon the cover-glass by the agency of heat. Occasionally it may be found beneficial to use, in place of simple distilled water, that which has been rendered slightly acidulated by the addition of acetic acid (one drop to a half-ounce or more, according to circumstances).

After the coloring matter has been thus washed out, the object may be at once examined under the microscope, or the film may be prepared for permanent mounting in the following manner. The cover-glass may be slid sideways to the edge of the object-glass and gently withdrawn. The water is then allowed to drain off the surface by resting the cover-glass in a more or less vertical position upon a small piece of bibulous paper. In a few moments the film becomes dry. Or the dehydration may be expedited by plunging it for an instant in strong alcohol, which latter is also to be drained off in a similar manner; but frequently the use of alcohol for this purpose is objectionable because of its decolorizing power. In many instances the bacteria lose, even during treatment for such a short time by alcohol, a considerable amount of color.

After the film has become quite dry it may now be permanently mounted; and the best mounting-medium for the permanent preservation of bacteriological specimens is perhaps Canada balsam, preferably dissolved in xylol. The tendency of all objects, including bacteria, stained with the aniline dyes, is to become decolorized in the course of time, and after a few months to a few years the coloring which originally may have been quite intense may be found quite faint, or to have entirely vanished, and this tendency is increased by the existence in the washing fluids or in the permanent mounting-medium of substances which naturally have the power of decolorizing. For this reason, in the processes used in preparation of bacteria, it is always well to avoid, unless absolutely necessary, the use of acids and of most of the volatile and essential oils.

(c.) The staining of bacteria in tissues is accomplished with somewhat greater difficulty than in cover-glass films. It should go without saying that the sections should be exceedingly thin and regular in thickness. They should be either fresh or from tissues hardened (and not too long preserved) in absolute alcohol, and they should not be cut long before they are stained. It is found that long soaking, in strong alcohol, of lumps of tissues containing bacteria sometimes seriously interferes with or entirely prevents a subsequent satisfactory staining of the bacteria; and when thin sections containing the latter are kept even for twenty-four or forty-eight hours in strong alcohol before staining, much the same difficulty is met with. The sections should, therefore, immediately after cutting, be subjected to the staining solution. And it may be said in a general way that the color-solutions to be used should be of greater intensity than those required for the treatment of cover-glass preparations; furthermore, the sections should be subjected to the action of the coloring fluid for a longer time, varying from a few moments to as many hours, according to the nature of the bacteria to be stained. After sufficient staining, the section is removed from the coloring fluid and for a short time subjected to the action of from 70 to 90 per cent. alcohol. This decolorizes all the elements, but the bacteria to a lesser degree than the others,—the object being to secure such a state of decolorization that the bacteria, and perhaps the nuclei also to a lesser extent, alone shall retain the color. To accomplish this desirable decolorization, alcohol slightly acidulated with acetic or lactic acid is sometimes used, and even distilled water acidulated in a similar manner is often employed; but in all cases where acids are employed greater care is necessary to prevent discharge of the color from the bacteria.

Although, as a rule, the various species of bacteria possess a singular avidity for the absorption of the aniline colors, yet there are some, and even many of the most important from a pathological stand-point, which form exceptions to this rule. For instance, the bacillus typhosus (Photo. No. 2 of typhoid fever) is one of a number of varieties of bacteria which are frequently quite difficult to stain well. In sections of tissues these bacteria are frequently difficult to find on this account. For their demonstration, Gaffky has recommended that the coloring solution employed be a concentrated, watery solution of methylin-blue freshly prepared. The sections should be immersed in this strong solution from twenty to twenty-four hours, and should then be washed in simple distilled water *free of any acid;* they are to be now dehydrated *for a few instants* in absolute alcohol, cleared in turpentine or cedar oil, and then mounted in balsam.

Loeffler has suggested the following solution as especially useful for the staining of the typhoid bacillus, the bacillus of glanders, of septicæmia, and other bacteria which are more or less difficult to stain: a saturated alcoholic solution of methylin-blue or other color-material, 33 c.cm.; a solution of caustic potassa (1 part to 10,000), 100 c.cm. After the section has been subjected to this fluid for a certain time, from a few minutes to half an hour, they are washed rapidly in distilled water slightly acidulated with acetic acid (about one drop to the half-ounce). This strong staining fluid is especially useful for demonstration of the bacillus of glanders.

(*d.*) There are certain bacteria already known which require special methods of staining for their demonstration. Among these are the bacillus of tuberculosis, of leprosy, of syphilis, of glanders, etc.

Bacillus tuberculosis.—This bacillus does not stain by the ordinary methods. In fact, there are some of the aniline colors for which it has no selective avidity whatever. For instance, it cannot be stained brown. On the other hand, the tenacity with which it retains certain colors which it absorbs with avidity is so phenomenal as to remove it entirely from the class of bacteria which become readily decolorized through the action of acids, especially the mineral acids. These peculiarities have been utilized by Koch, Ehrlich, and others for devising a color-method of differentiation which makes it possible and easy to distinguish this micro-organism from others known, and to furnish also a ready means of clinical diagnosis of great practical value.

For the demonstration of the tubercle-bacillus the following methods are readily applicable and at the same time reliable. A color-solution is made in the following manner:

A saturated solution of aniline oil in water	90 parts;
A saturated alcoholic solution of methyl-violet (or of fuchsin)	11 parts;
Absolute alcohol	10 parts.

Although the mixture containing this proportion of alcohol may keep for eight to ten days, or even longer, it should be filtered previous to each application; and in cases of great importance for diagnostic purposes, it is better to prepare the fluid freshly. The method of staining is as follows:

Have six watch-glasses in a row, and in imagination let us number them from left to right: No. 1, a small watch-glass, is nearly full of the aniline-water coloring mixture above mentioned; in No. 2, which should be a large watch-glass, is a considerable quantity of dilute nitric acid (1 to 3); in No. 3, also a large watch-glass, is a considerable quantity of 60 per cent. alcohol; No. 4, a large watch-glass, contains a considerable quantity of 90 per cent. alcohol; No. 5, a small watch-glass, contains a dilute watery solution of Bismarck-brown (or vesuvin) if methyl-violet has been used in No. 1, or of methyl-blue if fuchsin has been used; No. 6, a large watch-glass, contains distilled water.

The matter to be examined, if it be sputum or other semi-fluid or fluid material, should be spread in as thin a film as possible over the surface of a previously-cleaned cover-glass. Care should be taken to use for this purpose the thick purulent matter, avoiding the saliva, in the case of sputum. The film is dried and fixed upon the cover-glass in the usual manner, by heat. It is then inverted upon the surface of the coloring fluid in watch-glass No. 1. The latter is placed over the flame of a spirit-lamp or Bunsen burner, and heat carefully applied until steam-bubbles begin to rise in the fluid. The watch-glass is then set aside and allowed to cool for a few moments (from two to five). The cover-glass with the film intensely stained is now taken up between the points of fine forceps and for a second or two is dipped and moved to and fro in the acid fluid contained in watch-glass No. 2. It is then at once immersed and moved backward and forward in the alcohol of watch-glass No. 3, until the most of the color has disappeared. It is then removed and washed in the strong alcohol of watch-glass No. 4, until to the naked eye, by transmitted light, the film seems to be completely decolorized and has become decidedly gray. Then the cover-glass is floated film downward upon the surface of the contrast watery coloring fluid in watch-glass No. 5, where it is allowed to remain for a minute. It is removed from this and washed in the contents of No. 6. When the superfluous fluid has been thus washed off, the cover-glass is then stood up on edge upon a small piece of bibulous paper to drain and dry, or dehydration is secured more rapidly by immersion for a few instants in strong alcohol, after which it is likewise stood upon edge and completely dried. It is now mounted in Canada balsam for permanent preservation. Care should be taken that the film be completely dried before the balsam is applied; otherwise, if there is any moisture still present, the film will remain cloudy and furnish an indistinct, unsatisfactory view for the microscope.

After washing the film in watch-glass No. 6, the cover-glass may be at once inverted upon an ordinary object-glass and examined under the microscope immediately.

By skill and practice in the use of this rapid method there is no difficulty in preparing the tubercle-bacilli and demonstrating them under the microscope in less than six minutes. In fact, less time is required for such an examination than is necessary for an ordinary microscopic examination of urine, such as the clinician has long been familiar with.

If heat be not applied in the staining process, it is necessary that the film should be subjected to the action of the coloring fluid for twelve to twenty-four hours, and in the opinion of some, where absolute exactness is desirable, not only in finding tubercle-bacilli in the matter examined, but also in estimating their number, this long, tedious process is essential; but in the experience of the writer, and in that of many others in the habit of making such examinations, the shorter method, by the intervention of heat, is quite as reliable.

It is often desirable to search for the existence of tubercle-bacilli *in situ naturæ* in animal tissues. In this case it is necessary to harden a piece of tissue in absolute alcohol and cut from it exceedingly thin sections. The latter are treated in a manner very similar to that above described, except that the use of heat is discarded and the tissues allowed instead to remain in the staining fluid for twenty-four hours or more. The decolorization by the agency of nitric acid and alcohol is more difficult than in the case of films. The sections should be flirted in the dilute nitric acid for a little longer time and afterwards subjected still longer to the action of the alcohol. Indeed, it may become difficult, or sometimes even impossible, to render the section absolutely colorless to the naked eye; but when the color becomes quite faint, the sections may be treated by the contrast-color in watch-glass No. 5 for a few moments, then quickly washed in distilled water and subsequently dehydrated rapidly in absolute alcohol. When this is completed, the absolute alcohol is displaced by a drop or two of oil of turpentine or oil of cedar, and the sections are then permanently mounted in Canada balsam.

There are other methods which have been suggested for the differential demonstration of the tubercle-bacilli, which have been proved to be more or less satisfactory; but, as this is not the proper place to introduce complete details for the practice of bacteriological researches, they will not be mentioned here. (For Bacillus tuberculosis, see Photo. No. 3.)

Bacillus Lepræ.—The bacillus of leprosy behaves in a manner quite similar to the bacillus of tuberculosis, with respect to the method of staining the latter, above described. The bacillus lepræ differs, however, from the bacillus tuberculosis in this respect,—viz., in the fact that the former is much more quickly acted upon by the coloring fluid; and, besides, it is capable of staining by common methods, already mentioned, applicable to most of the bacteria. It is capable also of being stained brown.

A method of differentiating between the bacillus lepræ and the bacillus tuberculosis is based upon the readiness with which the former absorbs the special coloring matter. If the cover-glass film containing the lepra-bacilli is allowed to remain from six to seven minutes in a cold dilute alcohol solution or fuchsin (five to six drops of a concentrated alcoholic solution in a watch-glass full of distilled water), and is subsequently decolored for fifteen seconds in acidulated alcohol (one part of nitric acid to ten parts of alcohol), then washed in acidulated distilled water, and afterwards stained in a dilute watery solution of methylin-blue, then washed and examined at once in water, the lepra-bacillus, after such treatment, appears as a red rod upon a blue ground; whilst the tubercle-bacillus treated in a similar manner has not yet absorbed any of the coloring matter. Other methods of differentiation have been proposed which are more or less satisfactory and of varying readiness of application, which it is not necessary to mention.

Bacillus of Syphilis.—Lustgarten employs the following method for the differential demonstration of this bacillus. The sections are first stained from twelve to twenty-four hours at the ordinary room temperature in the following solution:

Concentrated alcoholic solution of gentian violet . . . 11 parts;
Saturated solution of aniline oil in water . . . . . 100 parts.

They are then kept in the same solution for two hours at a temperature of 60° C. The sections are now washed for a few minutes in absolute alcohol, then for ten seconds in a one-and-a-half-per-cent. solution of permanganate of potash, after which they are immersed an instant in a concentrated solution of chemically pure sulphurous acid. If the section is not entirely decolorized, this double decolorization is repeated three or four times, after which the sections are dehydrated in alcohol, cleared in essence of girofle, and mounted in balsam.

The bacilli of leprosy can be colored by the same procedure, and those also of tuberculosis; while all the other bacilli remain uncolored. The bacilli of syphilis are readily distinguished from those of tuberculosis because the former are not colored by the method already described for the demonstration of the tubercle-bacillus.

Alvarez and Tavel, in 1885, published an account of the discovery of a bacillus in the smegma and desquamation of the moist parts of the genital regions, which to coloring agents behaves in a similar manner to that of Lustgarten. According to these authors, their bacilli are stained by fuchsin, and the color resists the action of hydrochloric acid like the bacilli of tuberculosis, but they are finally decolored by alcohol. Their coloration is less resistant than that of the tubercle-bacilli. Cornil and others have confirmed the existence of bacilli in the smegma closely resembling the bacillus of syphilis described by Lustgarten, but Cornil declares that it does not absolutely correspond to the Lustgarten bacillus. The latter does not stain by simple methods of coloring, nor by the method of Ehrlich for the differentiation of the tubercle-bacillus; while, on the contrary, the bacillus of Alvarez does color by this latter process, as Cornil has confirmed.

Bacillus of Glanders.—This bacillus is also one of those usually difficult to stain satisfactorily. The method proposed by Loeffler is perhaps the best. For cover-glass preparations, a strong alkaline aqueous solution of methyl-blue, already described, is used, the film being subjected to its action for several minutes to half an hour. If the cover-glass has been inverted on a glass slide, the staining fluid is removed by capillary attraction in the manner already mentioned, but, instead of pure distilled water, that slightly acidulated with acetic acid is used, and this is immediately in turn drawn off by capillarity and washed out several times by means of pure distilled water. If the bacilli are contained in the tissues, the sections must be made and immersed in the staining fluid from twenty to forty minutes. They are then washed in distilled water slightly acidulated with acetic acid, rapidly dehydrated in strong alcohol, and mounted in dammar, after clearing with cedar oil or oil of turpentine.

Gram's method of staining is sometimes useful for differential demonstrations of certain bacteria. It is thus employed: the cover-glass preparations of bacteria or the sections containing the latter are stained for ten minutes to a half-hour or longer in a strong solution of gentian-violet in saturated aniline water. They are then removed, without washing, to a solution of iodine in potassium iodide, in which they are allowed to remain until they become dark brown or nearly black. They are now decolored in alcohol until they become quite gray. For those bacteria which can be stained by this method, it will be found that they remain intensely colored, whilst the ground is unstained. If it be desirable, the latter may be tinged with a contrast stain, a watery or a weak alkaline solution being employed for this purpose.

Methods of Staining Spores.—The spores of bacteria are exceedingly resistant to the action of coloring fluids. They appear to be enveloped in a thin, dense, more or less impenetrable membrane, which greatly opposes absorption of the coloring matter, and in order to overcome this impediment and secure the staining of these objects it is necessary to resort to special methods. One commonly used, and which has been found to be quite satisfactory, is as follows:

It has been learned by experiment that high heat or the more or less prolonged action of strong mineral acids or of strong alkalies destroys this resistance of the spore-envelope. Many kinds of spores can be stained if the cover-glass preparation, instead of being drawn three times through the flame, is drawn through it ten or more times; so likewise if the cover-glass preparation be exposed, in the sterilizing oven, for a half-hour to an hour, to a temperature of 150° to 180° C. Furthermore, many species can be stained, if the cover-glass preparation be, as usual, fixed by heat and then immersed for twelve to twenty-four hours in a strong alkaline solution of the color-material; or, better still, if it be floated for ten to twenty minutes upon the surface of a heated strong aniline watery solution of the color selected. A contrast-stain for the protoplasm of the bacteria should in this case be subsequently used. The double staining in these cases is more certain if before using the second color solution the first is washed in distilled water and then subjected for a few seconds to the action of absolute alcohol. If instead of alcohol a diluted mineral acid is used, the boundary between the spore and the adjacent protoplasm is more sharply defined. If the bacteria containing spores are in sections of tissues or are associated with cellular bodies, the histological elements may be stained with still a third color, such as hæmatoxylin, carmine, or picro-carmine.

METHODS OF ISOLATION OF SPECIES OF BACTERIA AND MODES OF CULTURE.

Customarily, it is only in the tissues of internal organs that it is possible to encounter pure natural cultures of the specific pathogenic micro-organisms. Even in such cases it is not uncommon to find the specific microbe associated with one or more different species extraneous to the disease. It is not seldom that secondary affections are grafted upon, or become the sequel of, the primary disease. Even when this is not the case, saprophytic bacteria may in various ways find entrance into the tissues. Different species of bacteria are so frequently associated together in substances that afford them nourishment that some method of separation of different species is essential for an intelligent study of their peculiarities of form, development, and physiological or pathological characters.

Without going into the history of the development of these methods, it is sufficient to remark that until Koch in 1881 proposed a method of culture upon a solid medium having for its solidifying substance animal gelatin, the only methods of obtaining pure cultures of individual species in practical use had been that of fractional culture proposed by Klebs, and that of dilution proposed by Lister, both making use of fluids for this purpose, the object aimed at in each of these two methods being the separation and equal distribution throughout the fluid used of single bacteria to such a degree that a single drop of the fluid should contain, as a rule, no more than a single micro-organism. When this degree of separation was believed to be attained,—usually from calculation, oftentimes based upon actual observation under the microscope,—a large series of test-tubes or small flasks, containing the fluid culture medium previously prepared, were inoculated each with a single drop from the diluted fluid suspending the isolated micro-organisms, and were then set aside for further observation. After a few days it was found that a certain number of the tubes or flasks, thus inoculated, had become more or less cloudy or shown in other ways that a growth had occurred. If the separation of the bacteria had been made with sufficient care, the non-occurrence of growth in a small number of the tubes furnished the proof that the drop with which they had been inoculated contained not even one germ capable of development.

If in a fluid containing two or more species of bacteria—one, however, predominating—the majority of the tubes would be found to contain growths of this latter species, and a certain, although perhaps limited, number of such tubes contain absolutely pure cultures.

This method of procedure required such extreme care, consumed such an enormous amount of time, and produced results which were so frequently uncertain, and therefore unsatisfactory, that, until better methods were devised, progress in the knowledge of the specific nature of bacteria advanced with slow steps, the enormous labor involved deterring most of the best observers from undertaking it.

After pure cultures of species had been obtained in this troublesome manner, Klebs made use of another method for the purpose of directly studying under the microscope the development of a colony from a single microbe. For this purpose he proceeded in the following manner, the minute details of which will be omitted here. Instead of a fluid culture medium, he used one which at the ordinary temperature is solid but transparent. To the ordinary fluid culture medium (such as meat broth) he added a sufficient amount of gelatin to render the whole solid when cool. In preparation for the examination, this solid culture medium was rendered fluid by gentle heat. It was then inoculated with such a quantity of a pure fluid culture as that each individual germ should be sufficiently separated from the others. This was secured by the employment of his method of dilution. A drop from this fluid gelatin-medium containing the individual germs evenly dispersed was now placed in a capillary cell, the top of which consisted of glass so thin that a high microscopic objective could easily work through it. When the drop had solidified, the cell was placed under the microscope and a germ brought distinctly into the field. This germ now became the object of observation, and was carefully watched, being kept in view for hours at a time without interruption. In this manner Klebs succeeded in following the process of multiplication of a single germ, and the development of a colony of similar individuals therefrom.

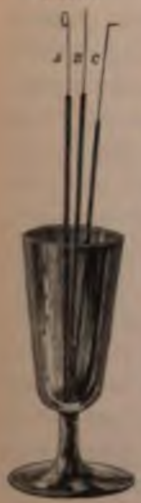
This method of cultivating colonies from single individuals in a solid medium became the basis of the celebrated method proposed by Koch for isolating single species from mixtures thereof, following them in pure cultures through their cycles of development and studying their vital and other characters.

Previous to the introduction of this new method of Koch, however, Brefeld had already made use of solid culture media for obtaining pure cultures of bacteria. His method essentially consisted in pouring melted gelatin containing nutritive substances over a flat level surface. After it had solidified, the point of a needle was dipped in the fluid containing the bacteria and numerous separate scratches with the point were made through the surface of the gelatin. The layer was then set aside protected from the dust. After a short time it was found that bacteria began to grow along the course of the scratches, and it was observed that in those later made the development occurred usually at interrupted points and consisted of colonies of individuals which had sprung from a single germ which had been rubbed off and left behind at that point. In this way it was possible with some rapidity and exactness to obtain pure cultures of different species. The method of Koch, however, has been found by experience to be of far more general, wider, and certain application, and it is now most frequently employed. It will be described later in some detail.

INSTRUMENTS AND APPARATUS REQUIRED FOR BACTERIOLOGICAL INVESTIGATIONS.

Instruments.—All instruments and all vessels containing culture media for such investigations must, as a fundamental principle, be thoroughly sterile; that is to say, absolutely free when in use from the accidental contact of living germs.

FIG. 8.



Besides the instruments employed in ordinary histological examinations, the following are deemed essential for bacteriological researches:

Plain glass rods. Glass rods into one end of which a fine stiff platinum wire is fused (Fig. 8): several of these should be at hand, one or two with the wire habitually straight (*B*); as many with the end turned at right angles to the length (*C*); as many more with the end made into a small loop (*A*). Numbers of test-tubes of ordinary size, three-quarters of an inch in diameter; fewer an inch in diameter. Small shallow glass cups two inches in diameter, with plain flat bottoms and a plain cap. Square or round wire cases of stiff galvanized wire, for holding small collections of test-tubes in a vertical position (Fig. 9). Flat-bottomed, long-

FIG. 9.

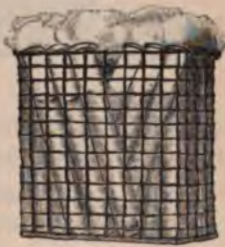


Fig. 10.

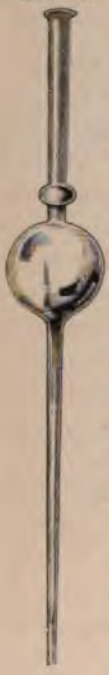
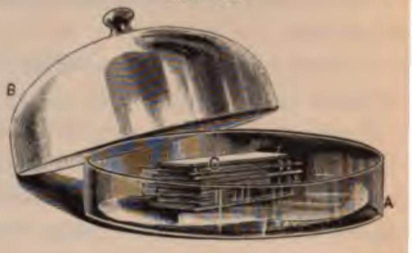
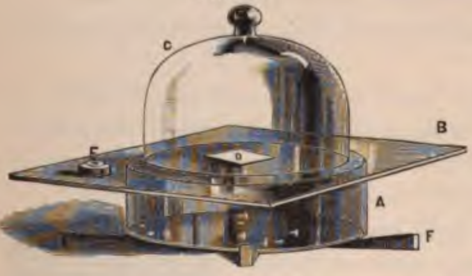


Fig. 11.



necked, thin, globular flasks, well annealed, of capacity from an ounce to a half-gallon. Long glass pipettes (Fig. 10) with a globular expansion in their course, of a capacity of an ounce, and the lower end sufficiently long to reach from the top of the neck of a flask to the bottom. Shallow glass dishes (Fig. 11) with flat bottoms and vertical sides, two inches high and eight or nine inches in diameter (A); shallow bell-glasses (B) to fit within these; glass benches to be placed within them, upon which oblong square plates (C) three inches broad and five inches long are to rest,—the whole to serve as *moist chambers.* When the moist chamber is in use, it should be sterilized and a layer of bibulous paper moistened with corrosive sublimate solution should cover the bottom of the shallow glass dish. Two or three thin copper cases of the proper dimensions for holding within them a dozen or so of the oblong glass plates above mentioned. These copper cases should be fitted with a suitable, closely-fitting, metallic cap having a convenient handle. A *cooling apparatus* of the following construction (Fig. 12): a glass or metallic cylinder (A) three or four inches high and six or seven inches in diameter filled with water to the brim; upon the top of this rests a square plate of rather thick glass twelve inches square (B), with the surfaces ground; upon the top of this rests a bell-glass (C) eight or ten inches in diameter, with a ground edge; under the bottom of the cylinder containing water are necessary three long thin wedges (F) at equal distance around the circumference; a small circular spirit-level (E) rests upon one corner of the glass plate; when in use a block of ice is placed in the water, the surface of the latter being in contact with the bottom of the glass plate, free from bubbles, and the surface of the ground-glass plate is perfectly levelled by means of the wedges. This apparatus is to be used in the preparation of the *gelatin plates.*

Fig. 12.



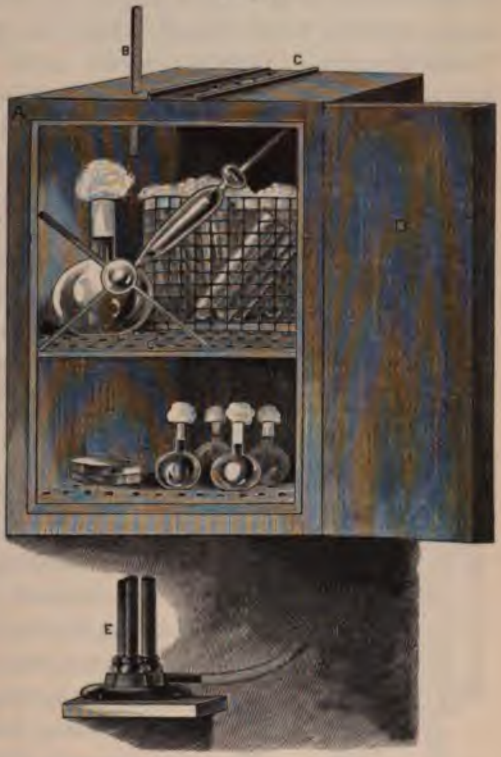
Quantities of bibulous paper and quantities of washed absorbent cotton, as well as vessels containing chemical germicides, should be at hand, such as carbolic acid in five-per-cent. solution, bichloride of mercury 1 part to 1000 parts of water.

STERILIZING APPARATUS.

Sterilization is best accomplished by dry heat or by steam.

Dry Heat Sterilizer (Fig. 13).—In order to apply dry heat for sterilizing, an oven of sufficient inner capacity, capable of standing at least 150° C., is required. One of the best ovens of this kind is constructed in the following manner. An oblong square box of strong thin sheets of galvanized iron is constructed with a double wall (A), in order to allow the passage of a flame, and the heated air therefrom, around the whole exterior of the inner case. The inner dimensions of the box should be, conveniently, eighteen to twenty-four inches high, a foot wide, and a foot deep. Supporting cleats should be fastened on the two sides at convenient heights for the purpose of supporting a perforated or wire shelf (G) in a horizontal position about the middle of the box, in order to divide it into two compartments. Outside of this inner case is built another at a distance therefrom of an inch in every direction. A hole is made in the top through both cases, and a short tube cemented therein which places the interior in communication with the external air. In this tube is inserted a cork carrying a Centigrade thermometer (B) with a scale indicating 200 degrees C., in such a manner that the bulb shall penetrate an inch or two below the top of the inner case. Besides, a row of half-inch holes an inch apart should be cut through the top of the outer case. A band of metal of somewhat greater length and breadth should be punched by a like number of similar holes at equal distance. This band should be fastened in cleats (C) in such a manner that it can slide backward and forward over the holes in the outer case in order to close the openings in the latter or to open them at will. In the centre of the bottom of the outer case a hole some two inches in diameter should be cut, and under this a triple Bunsen burner (E) is placed, connected with the gas-pipe, in order to supply the necessary heat. The top of the burners should be two inches below

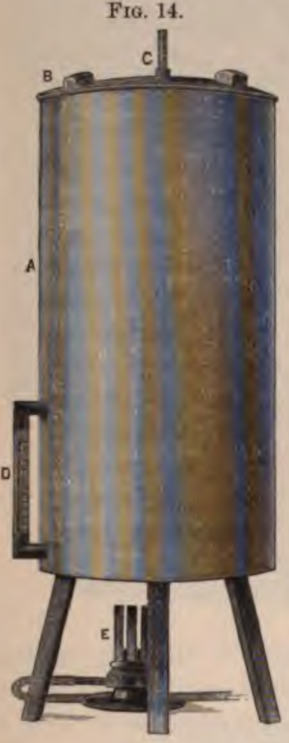
FIG. 13.



the level of the bottom of the outer case. A hollow door with an air cushion between the two sheets is hinged at the front (D) in such a manner that the inner box can be well closed from the external air.

Test-tubes previous to sterilization should be washed perfectly clean with distilled water. Their mouths should be stopped with plugs of washed cotton extending at least an inch into the tube. The desired number of test-tubes thus prepared are placed in one of the wire cases already mentioned, and the whole placed within the chamber of the sterilizing oven. Flasks intended for holding culture media are to be cleaned and stopped with cotton in an analogous manner. Pipettes are also washed and placed within the oven, as also the copper cases containing the glass plates, and any other apparatus which is desired to be thoroughly sterilized and which will not be injured by high heat. The Bunsen burner is now lighted and the temperature of the oven raised to 150° C. and kept at that height for at least three-quarters of an hour. At such a prolonged temperature all living organisms die, and even the white plugs of cotton become more or less singed. The flame is then turned off, and the oven allowed to gradually cool with the door kept closed.

FIG. 14.



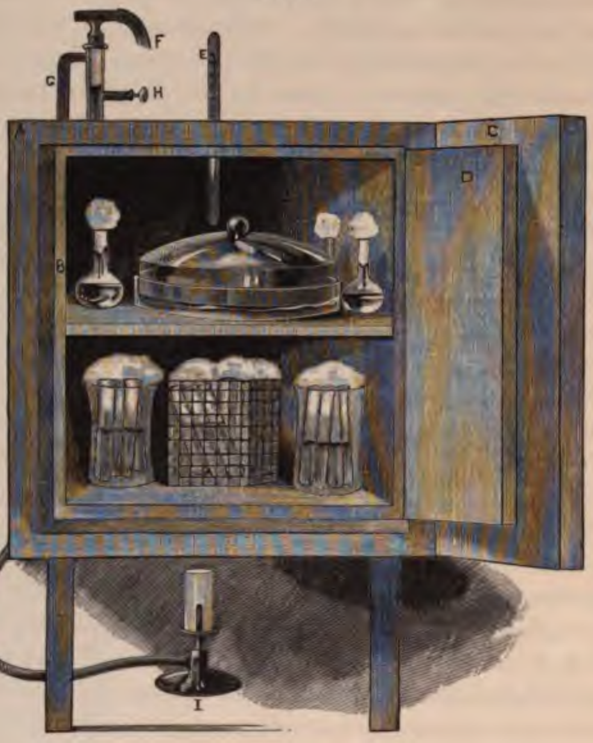
Steam Sterilizer (Fig. 14).—A steam boiler of the following dimensions and construction should be made. A tin or other metallic cylinder (A) fourteen to sixteen inches in diameter, and twenty-four to thirty inches high, with a tightly-fitting top (B), in the centre of which is a perforation for a cork carrying a Centigrade thermometer (C) registering 100° C., the bulb of which should extend two or three inches below the top, which latter should also be furnished with two convenient handles and should be covered with thick felt or thick asbestos-paper. Attached to the side of the cylinder near the bottom should be a water-gauge (D), for the purpose of constantly indicating the height of the water. Six or eight inches above the bottom of the cylinder should be fastened on the inside three or four small arms for the support of a removable metallic grate closely fitting the diameter of the cylinder, and best made of stiff wire. The cylinder is completely covered with an external envelope of heavy felt or thick asbestos-paper, to impede radiation. Of course the bottom itself is uncovered. This apparatus is furnished with heat by a triple Bunsen burner (E) attached to the gas-pipe. Previous to use, it should be seen that there are at least three to four inches of water in the cylinder; and this water should be raised to the boiling-point and kept there. Vessels, such as flasks, test-tubes, etc., containing culture media, both fluids and solids of various kinds, are placed within the cylinder for sterilization and subjected to the action of steam at the boiling-point for at least an hour.

Sterilization by Interrupted Lower Temperatures.—For the purpose of sterilizing blood serum and some other culture media which coagulate and become more or less opaque at temperatures below the boiling-point, it is necessary to use another apparatus, which consists of a cylinder with a double wall for the purpose of providing a layer of water about an inch thick all around the cylinder. From the bottom of the inner cylinder is a hollow tube an inch in diameter in communication with the water-cavity at the bottom; the cap of the cylinder is also double, so as to carry a layer of water an inch thick, and at one edge projects outward and slightly downward a hollow tube four inches long and an inch in diameter, closed at the outer end, the inner end being in communication with the cavity of the cap and likewise filled with water. The whole of this apparatus, except the bottom, is covered with a thick layer of felt or asbestos-paper, and the lid is perforated in the centre in such a manner that a cork and Centigrade thermometer reach downward into the water contained in the tube extending up from the bottom of the inner cylinder. The temperature is to be raised to 57° C. and kept at that point when the apparatus is in use. Blood serum to be sterilized, contained in test-tubes or flasks, should be placed within the inner cylinder containing air and kept at a temperature of 57° C. for half an hour. The vessels containing it are then removed and placed in the culture-oven, where they are kept at a temperature of 37° C. until the next day. Again they are placed in this sterilizing apparatus and subjected for half an hour to 57° C. They are again removed and placed in the culture-oven for another day; and so again this operation of sterilization is repeated in the same manner at least six or eight times. Afterwards these vessels are kept for several days in the culture-oven at a temperature of 37° C., by which time, if sterilization has not been perfect, this will become apparent by a development of bacteria in the serum. In the great majority of cases, however, it will be found that the fluid remains perfectly sterile.

Blood serum is used for culture-purposes either fluid or solid. In order to solidify the serum, an apparatus should be provided in which the tubes can be kept in a nearly horizontal position, with the top end of the tube raised about an inch. For this purpose the following apparatus has been devised. It consists of a square metallic box with double walls so constructed that its inner cavity is surrounded by heated water. The bottom of the inner box is so inclined that the higher edge is an inch above the height of the lower edge. The box is provided with a glass lid, and the whole is covered, except the bottom, with thick felt or asbestos-paper. A thermometer is attached to this box in such a manner that the bulb enters the water surrounding the box and registers the temperature. The temperature should be gradually raised to 62° C. and kept for a short time at this height until coagulation takes place, and the tubes should be watched carefully and removed as soon as this occurs. In this manner blood serum can be solidified without much loss of transparency of the medium.

Culture-Oven (Fig. 15).—One or more culture- or brood-ovens should be made, in the following style. A double box of thin metal should be made with an interior height of two feet and a breadth and depth of fourteen inches or more. This should be encased in an outer box so as to leave a space of an inch (B) between the walls. The box should be provided with a Centigrade thermometer (E) of 100 degrees extending through the top in such a manner that the bulb projects an inch or two into the interior of the inner box. A hole should be perforated also in the top of the outer box at one of the corners, so that a cork carrying a thermostat (G, F, H) can be inserted into it and the bulb containing the mercury extend some inches into the space between the walls of the outer and the inner box. This space is to be filled completely with water, so that the whole box, sides, top, and bottom, is surrounded with a layer of this fluid an inch thick. The whole apparatus is to be covered, except on the bottom, with thick felt or asbestos-paper (A), and the box is to be provided with two doors,—an inner glass door (D) fitting tightly and an outer hollow metallic door (C) carrying a layer of air an inch thick. This should also be capable of being tightly closed, and should likewise be covered externally with felt or asbestos-paper.

Fig. 15.



The temperature of the oven is regulated in the following manner. The tube from the gas-pipe is connected with one arm (F) of the thermostat; the other arm (G) of this instrument is also connected with rubber tubing which is attached at the other extremity to a small burner (I) capable of furnishing a small round gas-jet. The latter is to be protected from side-movements of the air by means of a mica or glass cylinder suitably supported. This burner is placed under the centre of the bottom of the oven, and the desired degree of temperature, recorded by the Centigrade thermometer, with 30 degrees and upwards of its scale projecting above the top of the oven, is obtained by regulating the thermostat by means of the fine screw (H). An exceedingly regular temperature is obtained by this means, especially if the gas-supply from the pipe be equalized by a proper regulator.

In hot climates, and in hot weather in temperate climates, the gelatin media commonly used solid become fluid from the high heat, and the great advantages offered by this culture medium are lost if there is no means of keeping it solid at temperatures sufficiently high to permit development therein. The culture-oven above described is capable of being used for this purpose, but its mode of preparation must in that case be modified. A current of water cooled by ice is made to pass through the cavity between the inner and the outer box, and the desired temperature of the air in the inner box is to be secured by regulating the rapidity of the flow of the current of ice-cold water.

CULTURE MEDIA.

Fluid Culture Media.—Most of the bacteria grow with exuberance in fluids containing various organic substances, especially animal. The most commonly used culture fluids are broths made from flesh of various kinds,—chicken, beef, mutton, veal. These broths are usually made in the proportion of one pound of flesh to a litre of distilled water. The flesh is chopped fine, the fatty portions having been removed, and is allowed to soak for a few hours in distilled water kept cold during that period by keeping in an ice-box. The water may then be poured off, and the juice squeezed out of the flesh by means of a meat-press is added to it. The mixture, which has an acid reaction, is then neutralized and thoroughly boiled for forty to sixty minutes, until all the coagulable albuminous substance is precipitated. It is then filtered into large sterilized flasks, a stock of the latter being constantly kept on hand. In the process of filtration it is possible that some living germs may have entered the fluid, which makes it necessary to sterilize completely. This is done by boiling for ten to fifteen minutes on five or six successive days; in the interim the flasks are kept in the culture-oven at a temperature of 35° C.

It is often desirable to add other substances to these broths, for the cultivation of certain species of bacteria. One to two per cent. of *peptonum siccum* and a half per cent. of chloride of sodium may be added. These are best added subsequent to the first boiling, for the presence of peptone in the broth darkens the color considerably, the intensity of the latter being increased by prolonged boiling. After the addition of the peptone and salt, it is necessary again to neutralize, boil, and filter, and the subsequent repeated boilings should not in this case extend over ten minutes at a time. Whenever any of this stock material is withdrawn it is necessary again to sterilize the remainder, for, however carefully the fluid may be withdrawn by means of sterilized pipettes, it is possible that a few bacteria may enter the flask accidentally from the air; and it is also advisable to boil the fluid at least once or twice after it has been transferred to the small sterilized vessels in which it is to be used.

For culture-purposes, the fluid may be placed in sterilized test-tubes, or small one-ounce sterilized flasks, and it is advisable to keep constantly ready for use a number of such vessels. The fluid should not more than half fill the flask, and the test-tubes should not contain more than one and a half inches of it.

Frequent use of this fluid is made for hanging drop cultures, and for this purpose it is necessary to have a number of object-glasses with a concavity ground out of one of the surfaces. Such cultures are to be made in the following manner. The object-glass and an ordinary thin, perfectly flat cover-glass are sterilized, best in the sterilizer by dry heat; a ring of vaseline is placed around the edge of the cavity, by means of a camel's-hair brush; the cover-glass is seized with the points of a delicate pair of sterilized forceps; a drop of the fluid is removed with the loop of platinum wire, previously heated to redness in the flame of the Bunsen burner or spirit-lamp, and is placed upon the centre of one surface of the cover-glass; the drop is now inoculated with the desired bacteria by touching it with the point of platinum wire upon which they have been removed from the material for observation. The cover-glass is immediately turned downward and placed upon the ring of vaseline in such a manner that the drop shall hang over the centre of the concavity in the object-slide. It is then gently pressed down until the vaseline forms an uninterrupted layer around the edge, entirely excluding communication between the surrounding air and that contained in the concavity. This hanging drop culture is now ready for observation, and can be studied constantly under the microscope at the ordinary temperature of the room, or upon a warm stage, or it can be set aside for development at the ordinary room temperature, or placed in the culture-oven and examined from time to time. After such drop cultures have been sufficiently studied in the fresh state, the cover-glass may be carefully removed and the fluid allowed to dry; the film thus formed may be fixed and artificially stained and mounted permanently in the manner previously described.

Solid Culture Media.—Solid culture media may be either transparent or opaque.

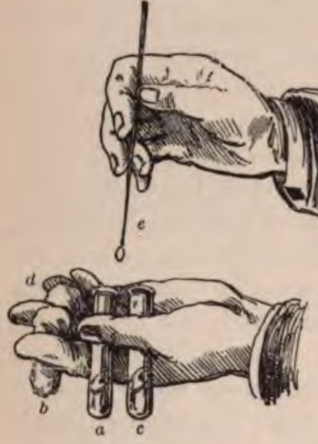
Of the transparent culture media, that containing animal gelatin is most generally used, and a stock of it is best prepared in the following manner. A sufficient quantity of meat juice has already been strongly boiled until the albumen is thoroughly coagulated. The best gelatin obtainable is added in the proportion of five to ten per cent. (The stronger percentage should be employed in mild weather.) Before adding to the fluid, which should be cold, the gelatin is to be chopped into fine pieces; it is then soaked in the cold fluid for half an hour or more until it has become well swollen; the whole is then heated until the gelatin becomes thoroughly melted. The peptone and salt are then added in the proportion already mentioned, and the mixture is neutralized with a strong solution of sodium bicarbonate. Care should be taken to have the reaction faintly alkaline rather than at all acid, as many forms of bacteria will not grow well or develop at all in acid culture media. It is well, however, for the cultivation of certain germs, to provide a stock of slightly acid culture media, both fluid and solid. This mixture is now well boiled for thirty minutes and filtrated through strong filter-paper previously well sterilized in the sterilizing-oven, the funnel supporting the filter having been also thoroughly sterilized. Before the mixture is poured upon the filter, the latter should be moistened with a small quantity of sterilized distilled water, and it is necessary that the filtering should be done while the fluid is very hot. In fact, in filtering through filter-paper it is advisable that the funnel should be surrounded by boiling water in a vessel properly formed to receive the funnel. The filtration, however, can usually be satisfactorily performed without such an apparatus, if instead of the filter-paper fine sterilized absorbent cotton is used. In this case the funnel must be kept quite warm, by throwing the flame of a Bunsen burner around it frequently. The filtered fluid is to be directly collected in a number of large sterilized flasks, and a stock of it constantly kept on hand. For immediate use, a convenient number of test-tubes are one-fourth filled by means of a sterilized pipette. It is sometimes found that the mixture thus obtained is not quite clear; and this can be determined from the first flow of the fluid through the filter. In this case it is necessary to stop the filtration and clarify the mixture by the addition of the white and shell of an egg. This albumen should not be added until the fluid has become cool. It is to be thoroughly disseminated throughout the mixture by shaking well, and the whole again subjected to hard boiling. It should be stated here that these boilings are best done in the steam cylinder. After the egg-albumen has completely coagulated, the mixture is again filtered in the manner just described.

It is finally necessary to sterilize the filtered fluid; and this is done by placing the various vessels containing it in the steam cylinder for ten to fifteen minutes on five or six successive days. In the interim the vessels should be kept in the culture-oven at a temperature of 35° C. The remark previously made should be repeated here, that whenever any of the stock material is removed from the flasks containing it, it is necessary to sterilize again that which remains, in order to prevent development of bacteria which may accidentally have found access while the cotton plug has been removed.

Test-tubes containing flesh-peptone-gelatin thus prepared are used for culture in various ways. The most common use is to inoculate them with pure cultures of bacteria by means of a puncture with the point of a platinum wire extending into the depth of the gelatin.

Another, and the most important, use of this gelatin culture medium is that introduced by Koch for obtaining pure cultures from mixtures of various species. The procedure is as follows. Three gelatin tubes are taken and the contents rendered fluid by gentle heat; after the fluid gelatin has descended to the temperature of body-heat, one tube is inoculated, by means of a previously sterilized platinum needle, with the material containing the various bacteria in question; after the inoculation, the tube is again immediately plugged and the fluid well but gently shaken, in order to diffuse the germs thoroughly and evenly throughout the fluid mass; from this, while still fluid (*a*), another tube (*c*) of the three is inoculated by transferring into it with the platinum wire loop (*e*) three drops in the manner indicated in Fig. 16; the second tube is then immediately plugged and well shaken, after the manner of the first. The third tube is inoculated from the second in the same manner. A sterilized glass plate is now carefully withdrawn from the metallic case enclosing it, care being taken to seize it by the edges between the thumb and finger; this is placed upon the ground-glass plate of the cooling apparatus already described (Fig. 12, D), and immediately covered with the bell-glass; in a few moments the glass plate has become sufficiently cold, and the gelatin from the first test-tube is now poured upon it and spread out, either by means of the lip of the tube or a sterilized glass rod, in an even layer in the form of an oblong square, care being taken that the fluid does not extend to the edge of the plate, and the latter is again quickly covered with the bell-glass. In pouring the fluid from the test-tube, care should be taken that upon removing the cotton plug the inner surface of the lip is well wiped. The external surface of the lip should be exposed for an instant to the flame of the Bunsen burner, but the fluid should not be poured out until the lips have become sufficiently cool, otherwise many of the germs contained in the inoculated tube may be killed by the action of the heat as the fluid flows out. In a few moments the layer of gelatin has become solid. The fluid contained in the other test-tubes is spread upon glass plates in a similar manner. The plates in regular succession are placed upon the benches in the moist chamber (Fig. 11) and set aside for development.

FIG. 16.



This flesh-peptone-gelatin becomes fluid below the body-temperature (at about 80° F.), and if it is desired to be kept solid during the growth of bacteria, it cannot be subjected to the heat of the culture-oven. The surrounding room temperature is sufficient for the development of most of the germs which will grow in this medium. High summer heat is frequently sufficient to melt the gelatin, and at these times this medium is not usually available for solid cultures, unless the culture-oven be used as a cooling-box.

In solidifying, the germs dispersed throughout the layer of gelatin upon the plate are fixed in the position in which they may happen to be caught at the time, and from each one capable of development a colony will be formed. It will be found that these colonies, visible under a low power of the microscope (fifty diameters) or to the naked eye, will have developed in thirty-six to forty-eight hours, or more, and it will be seen that in one of the gelatin plates the colonies are sufficiently distant from each other to permit of inoculations from individuals, by means of the point of the platinum wire, without danger of accidental contact with any of the others. This plate is now used for making pure cultures. A number of solid gelatin tubes at hand are inoculated from the different colonies and set aside for development, and, if sufficient care has been exercised in the procedure, it will be found that each tube contains a perfectly pure culture.

Instead of pouring the inoculated fluid gelatin from the tube upon a large glass plate in the manner described, a small quantity of it may be withdrawn by means of a sterilized pipette and spread upon an ordinary object-glass which has been previously sterilized. The layer thus formed, after solidification, may be placed in the moist chamber for development, and the growth of isolated colonies may thus be watched under the microscope from time to time.

A device which is now frequently used for plate cultures is even better than that just described. After the gelatin has been poured upon the plate, a mat with a large perforated centre is cut out of stiff paper one-eighth of an inch thick, the outer edge of the mat having the same dimensions as the plate. This mat, which has also been sterilized, is placed upon the plate, and another plate is clamped upon it. Thus we have a closed shallow chamber formed capable of being placed upon the stand of the microscope.

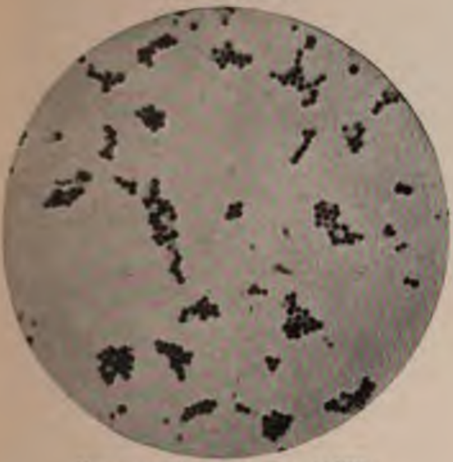
Anaërobic bacteria will not grow when exposed to free air. The surface of the gelatin upon the glass slide or upon the glass plate may be covered in whole or in part by extremely thin sterilized mica plates and thus protected from the air, when such bacteria may have an opportunity for development, in which case the colonies can be satisfactorily examined under the microscope, or used for obtaining pure cultures in the usual manner.

Another use of the gelatin layer upon the glass slide has already been alluded to, and is as follows. A sufficient quantity of sterilized flesh-peptone-gelatin is removed with a sterilized pipette and poured upon the surface of the glass slide and allowed to solidify. The gelatin is then inoculated by scratches with a platinum wire carrying the desired microbes, after the method of Brefeld already described. The slide is then placed in the moist chamber for development and subsequent examination.

Agar-Agar-Flesh-Peptone.—When it is desired to employ transparent culture media which remain solid at the body-heat, or at the ordinary temperature of the culture-oven, agar-agar is used, in the proportion of one to two per cent., instead of gelatin, in the following manner. The agar is obtained already ground, or is chopped up into fine pieces. The required quantity is added to the meat juice or broth, wherein it is allowed to soak until thoroughly swollen. After neutralization the whole is then well boiled for at least an hour, and the mixture is filtrated, through sterilized washed cotton, or through sterilized fine flannel, into two or three sterilized receiving-flasks of sufficient capacity. To one of the flasks is added sterilized glycerin in the proportion of six to eight per cent. To another of the flasks a similar quantity of glycerin is added with peptone and salt in the usual proportions. It is necessary again to boil the mixture in these flasks, and filter. The process of filtration is far more difficult in this case than in that of the gelatin mixture. The filtration should proceed while the vessels are subjected to the action of steam in the steam sterilizer. It is to be remarked that care should be taken to have the final fluids in each of these cases neutral or faintly alkaline. These mixtures solidify at 40° C., and become slightly opalescent; they are never so transparent as flesh-peptone-gelatin. After the filtration has been satisfactorily performed, the fluid should be distributed, as in the case of the gelatin mixture, in stock flasks of convenient size and in test-tubes for ready use. The sterilization of this material must be accomplished in the manner already described for the gelatin mixture. This material may be used for cultures in the same manner as the gelatin mixture, and, besides, the cultures may be made in the culture-oven and at temperatures even as high as the highest fever-heat.

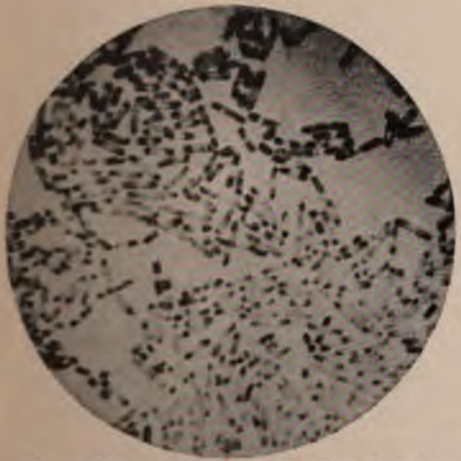
The agar mixture, besides as in the ordinary plate culture method, is employed for the isolation of mixtures of various species of bacteria in the following manner, after Esmarch. Three sterilized agar tubes are taken and the contents rendered fluid. After cooling down to about 42° or 43° C., these tubes are inoculated in the manner already described for the gelatin plates. A block of ice at hand, having a smooth upper surface which should be horizontal, is so grooved by scraping with a knife that a gutter six inches lóng and three-quarters of an inch wide and as much deep runs across the surface. The inoculated tubes in succession are now held horizontally and slowly rotated on their axes, in order that the fluid may spread evenly over the whole of the inner surface of the tube as far up as the stopper. This being done, the tube is placed horizontally in the ice gutter, where it is immediately set in rapid axial rotation by the hand. In this manner the film of agar lining the tube becomes quickly solid. The tubes

Photo. No. 1.



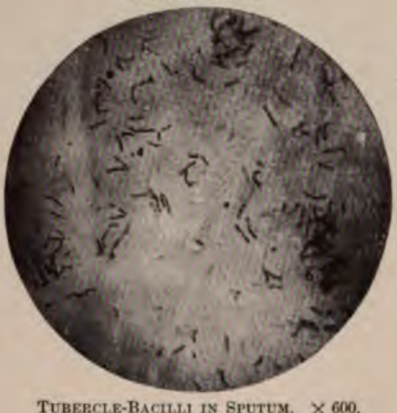
Micro- and Streptococci. × 1200.

Photo. No. 2.



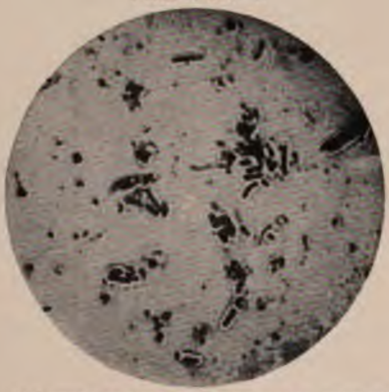
Typhoid Bacilli. × 1200. Pure culture from the spleen.

Photo. No. 3.



Tubercle-Bacilli in Sputum. × 600.

Photo. No. 4.

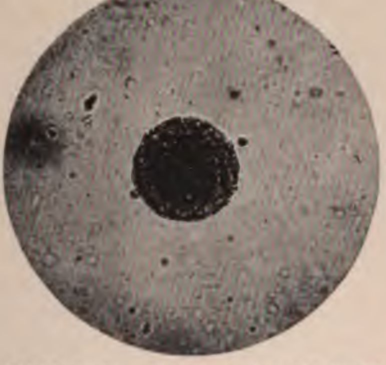


Bacteria of Intestinal Contents, showing Two Comma Bacilli.—Case of cholera Asiatica. × 1200.

Photo. No. 5.

ing Colony in Gelatin Plate of Cholera
kler).—Twenty-four hours' growth. × 30.

Photo. No. 6.



Non-liquefying Colony in Gelatin Plate of an Air Micrococcus. × 30.

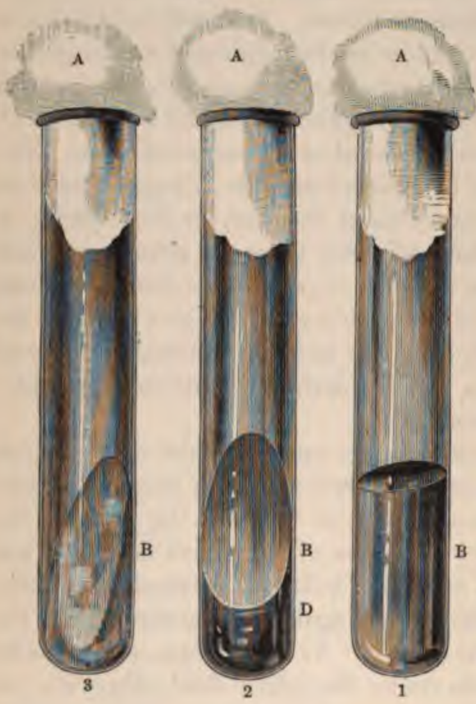
are now placed aside in a horizontal position for a half-hour; they can then be kept in the vertical position at the ordinary temperature of the surrounding air, or placed in the culture-oven if so desired, for development of the germs scattered throughout the layer of agar. From time to time the development of colonies in these tubes can be watched under the microscope by having an attachment upon the stage which will hold the tube in proper horizontal position and allow of its rotation and a to-and-fro movement, so that any desired colony may be brought into the field. After a sufficient time it will be found, as in the case of flat gelatin plates, that one of the Esmarch tubes shows the colonies sufficiently separate from one another to allow of one being touched with the bent point of a platinum needle and a portion of it removed and inoculated into any other desired medium, thus securing a pure culture therefrom. It should be stated that this method of cylindrical plate cultures is also applicable to the gelatin mixtures. The use of the Esmarch tubes for this purpose has several advantages, chief of which is the security against accidental inoculation of the culture medium from the germs suspended in the surrounding air. The colonies growing in such tubes can be observed for a much longer time than those upon glass plates in the ordinary moist chamber, for the capacity of the covering bell-glass is such that ordinarily there are always a certain number of germs suspended in the included air, and in the course of hours they will settle upon the surface of the gelatin plate and give origin to the growth of colonies starting therefrom. For isolating different species and obtaining pure cultures therefrom, the Esmarch tubes are now generally preferred to the former plate method.

Plate cultures are, however, used for other purposes, chief among which is the differentiation between colonies of different species by means of certain characteristics of growth. (Photos. Nos. 5 to 8.) For this purpose the gelatin plates are much more useful than those of cylindrical form. It was soon found by Koch in using this method of isolation of bacteria that many of the different species presented certain distinguishing characteristics in the aspect of colonies developed in the gelatin. As an example, some species while developing in such a medium render the gelatin fluid, others do not possess such an influence. Furthermore, certain species spread more or less widely over the surface of the gelatin, while others are heaped up upon it within narrow limits. Again, some of the colonies present regular circular outlines, others irregular. Some of them are more or less coarsely granular, others finely granular, still others more or less glairy, others powdery. Some present one color, some another. Some have the power of rendering the neighboring gelatin fluid, while developing evenly throughout the fluid mass, thus rendering it cloudy; others are limited more or less to the centre of the fluid portion, being surrounded by a fluid border entirely limpid. Some possess the power of disseminating a characteristic color in a narrow zone surrounding the developing colony, and so on. Thus the differences between many species are more or less marked and characteristic, so that for the recognition and differential diagnosis of numerous species of bacteria the gelatin plates furnish most valuable means. The aspects of pure cultures of these species are frequently characteristic also in gelatin tube cultures. (Photos. Nos. 9, 10.)

Whilst many of the species of bacteria present certain distinguishing characteristics when cultivated in agar-agar, in this medium they are usually far less marked than in the case of gelatin cultures, and in it the number of species possessing special characteristics is far more limited.

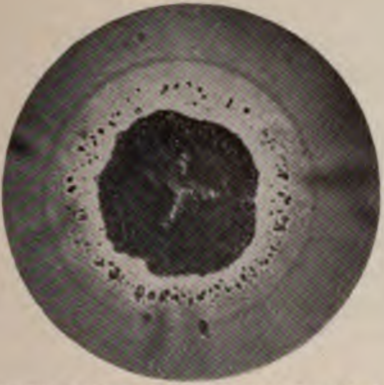
The agar-agar tubes for ordinary culture are usually prepared in such a manner that the surface of the agar is increased by inclining the tubes (Fig. 17, 2, B) before solidification in the same manner as that described for the blood serum, and, instead of punctured inoculations, scratches along the surface are usually made with the point of a needle.

FIG. 17.



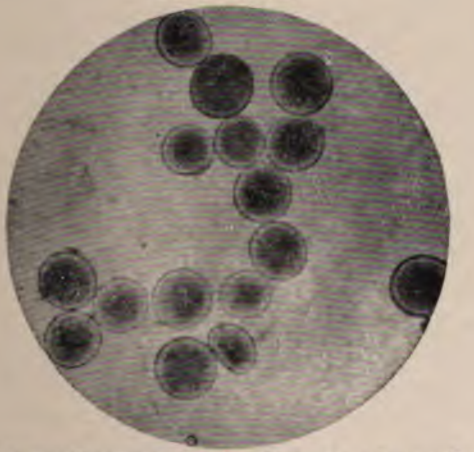
Opaque Culture Media.—Culture media of this kind have been long used. Perhaps the most common consists of sterilized bread-pap contained in small sterilized globular flasks. Potatoes either sliced or mashed and kept in similar vessels have also been more or less extensively used. The most frequent use of potato for this purpose has been as follows. Firm potatoes are selected, free from specks, their skins well scrubbed with a stiff brush, the eyes containing particles of earth are picked out, the potato is immersed for twenty minutes to half an hour in a solution of corrosive sublimate, 1 part to 1000, taken therefrom and placed in a colander with freely-perforated bottom, subjected for an hour to the action of steam in the steam sterilizer, then allowed to cool; after which blades of common knives (a sharp table-knife with a wooden handle answers the purpose well) are thoroughly sterilized by holding in the flame of the Bunsen burner, and allowed to cool. The hands are now well washed and dipped for a few moments in a solution of corrosive sublimate, 1 part to 1000. The potato is now firmly grasped between the first two fingers and thumb of the left hand and evenly divided with the sterilized blade of one of the knives. Each half is placed in the moist chamber with the cut surface up, care being

PHOTO. No. 7.



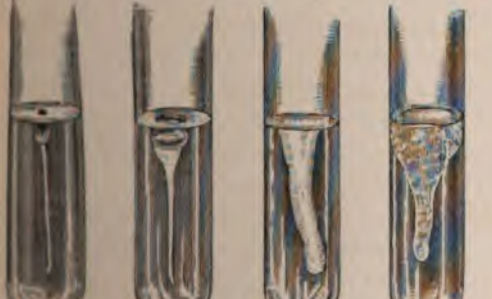
COLONY IN GELATIN PLATE OF COMMA BACILLUS OF KOCH.—Forty-eight hours old. × 30.

PHOTO. No. 8.



NON-LIQUEFYING COLONIES IN GELATIN PLATE OF A CURVED BACILLUS, RESEMBLING THAT OF KOCH, BUT NOT IDENTICAL WITH IT. × 30.

PHOTO. No. 9.



COMPARISON OF TUBE-CULTURES IN GELATIN OF COMMA BACILLUS OF KOCH (THE TWO TO LEFT) WITH THE CURVED BACILLUS OF FINKLER (THE TWO TO RIGHT).—Half natural size. (Copy after Koch.)

PHOTO. No. 10.

CULTURE IN GELATIN TUBE OF COMMA BACILLUS OF KOCH (FIFTY-TWO HOURS OLD), SHOWING APPEARANCE OF AIR-BUBBLE AT TOP, THE SURFACE OF THE SOLID GELATIN BEING INCLINED.—Natural size.

taken in this manœuvre that nothing shall come in contact with the surface. The potatoes thus prepared are covered as quickly as possible with the bell-glass, in order to limit exposure to the surrounding air. The middle of the cut surface of the potato thus prepared may now be inoculated with the platinum wire and set aside for development. The latter may take place at the ordinary surrounding temperature, or in the culture-oven, as may be desired. Many of the bacteria will be found to have a more or less characteristic growth upon this culture medium, and for a few of them indeed it offers the only known ready means of making a differential diagnosis. After some days, however, growths of fungi which have fallen from the air are apt to appear upon the surface of the potato and thus frequently interfere with the observation. In order to obviate this, a modification of the potato culture method has recently been introduced by Meade Bolton. A number of large test-tubes an inch in diameter are required. A long cylindrical punch of an inner diameter slightly less than that of the test-tube is needed; this also should be sterilized. A number of fresh potatoes are carefully selected, the two ends cut off with a sterilized knife. A plug is now punched out from the centre by means of the punch. This plug is divided by a sterilized knife in such a manner that the knife passes from one corner at the top obliquely downward to the opposite corner at the bottom. Each of these halves is placed in a test-tube with the thick end at the bottom (Fig. 17, 3, B). After a sufficient number of test-tubes have been thus filled, they are sterilized by subjecting them for an hour to the action of steam in the steam sterilizer. This may be repeated once or twice on successive days. The potatoes are then ready for culture, and the inoculations are to be made upon the middle of the inclined surface by means of a platinum needle. It is found that this method of potato culture not only secures greater protection from accidental contamination of the culture medium, but the growth of the inoculated bacteria can be closely watched from time to time directly through the walls of the tube.

Other vegetables and even animal substances of various kinds may be used for cultures in a similar manner. For the cultivation of bacteria which grow preferably, or better, in acid media, either fluid or solid, various substances have been used, such as prunes, quinces, apples, carrots, etc. Many of the natural animal fluids have also been employed, such as aqueous humor, lymph, pleuritic, pericardial, or abdominal fluids, urine, milk, etc. The agar medium to which five to eight per cent. of neutral glycerin has been added is a good substitute for blood serum.

For long preservation free from possible destruction by the accidental development of bacteria, the necks of the stock flasks and the tops of test-tubes can be hermetically closed by melting them in the flame of a powerful Bunsen burner, as suggested by Sternberg. This is a convenient means of providing one's self with requisite media to be transported long distances and used in distant scientific expeditions. But the most convenient means of accomplishing the same object—in fact, it is well to use it habitually, because of its simplicity—is that used at the Agricultural Department by Dr. Theobald Smith, and at the Army Museum by Dr. Wm. M. Gray. After the vessel containing the culture medium (flask or test-tube) is filled or inoculated, before stopping the vessel the end of the cotton stopper is dipped into very hot melted paraffine. The plug of cotton with its end saturated with the hot paraffine is now replaced immediately in the mouth of the vessel, which when cold is found to be hermetically sealed. When it is desired to withdraw the plug from such a closed tube, it is only necessary to heat the neck gently in the flame in order to melt the superficial portion of the paraffine.

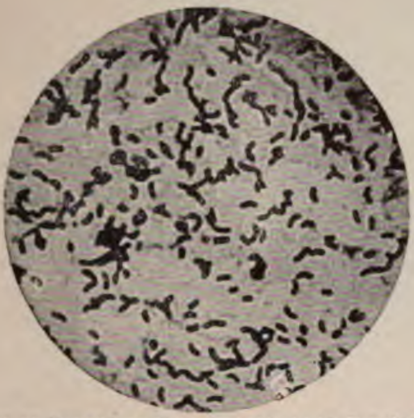
METHOD OF ENUMERATION OF BACTERIA.

The most frequently employed method of counting the bacteria contained in a given specimen of *water* is based upon Koch's method of isolation of microbes by resort to gelatin plate cultures. A certain small quantity of the water, *freshly obtained*, is *without delay* (for bacteria will multiply even in distilled water) well mixed with a certain considerably larger quantity of sterilized flesh-peptone-gelatin rendered fluid in a sterilized test-tube, and the mixture is poured upon a sterilized glass plate after the method already described for making gelatin plate cultures. After the layer of mixed gelatin becomes solid, the plate is set aside in the moist chamber for twenty-four to thirty-six or more hours to develop. After the colonies have sufficiently developed, the plate is placed upon a black sheet of pasteboard or of metal which is distinctly and regularly divided by a series of parallel cross-lines into small squares, and is then placed upon the stage of the microscope furnished with a low-power lens (twenty to thirty diameters), or upon the stage of a dissecting microscope. The number of colonies within a square can be counted without difficulty. When a number of the squares are thus examined and the average in each square ascertained, the data for a very close approximation of the number of bacteria contained in a cubic centimetre of the water in question are obtained; for each colony has developed from a single germ. It is unnecessary to remark that the number of bacteria in other fluids may be estimated in a similar manner. Enumeration of the bacteria in *air* and *earth* may be approximated by suitable modifications of this method.

CULTIVATIONS FROM ANIMAL TISSUES.

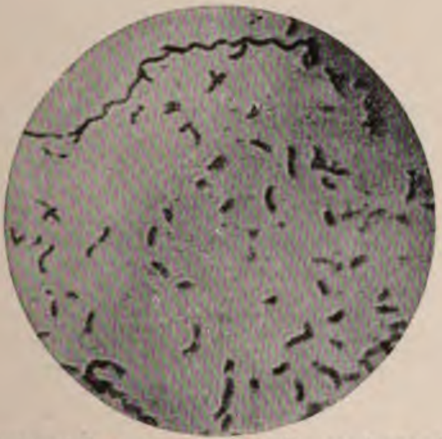
In seeking to obtain pure cultures of bacteria existing upon exterior surfaces or upon the surface of alimentary or other passages or open canals, which are constantly lined with numerous varieties of microbes, resort must be had to the method of plate culture, and the labor is usually great. In the case of search for bacteria in internal abscesses or solid lesions, the work of isolation is far less tedious, for the number of associated organisms is then much less.

Photo. No. 11.



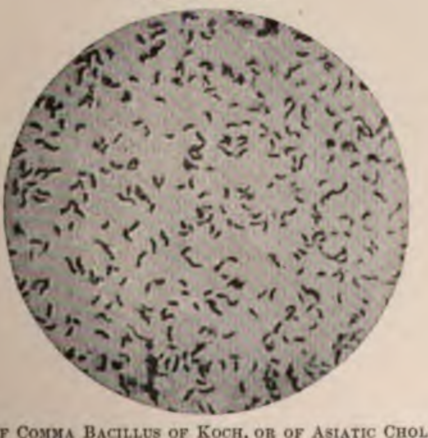
Pure Culture of Deneke, or Curved Bacillus of Old Cheese. × 1200.

Photo. No. 12.



Pure Culture of Finkler, or Cholera Nostras, Curved Bacillus, showing a Spirillum. × 1200.

Photo. No. 13.



Pure Culture of Comma Bacillus of Koch, or of Asiatic Cholera. × 1200.—
Obtained by plate culture from intestinal contents of same case as No. 4.

In the case of abscesses or enclosed fluids, a small quantity is withdrawn by means of a sterilized hypodermic syringe after previous sterilization of the surface through which the puncture is to be made. Such sterilization is secured in the following manner. The selected surface is thoroughly well washed and scrubbed with soap and water. It is next well washed with alcohol. Then it is washed with a solution of corrosive sublimate in sixty per cent. alcohol (1 part to 500), and, if possible, a wad of absorbent cotton moistened in the same fluid should be strapped closely upon the surface and kept there covered with oiled silk for several hours. If, however, more expedition is necessary, after the surface has been well washed first with the soap and water and the alcohol, and then with the germicide, the latter is washed off again with strong alcohol. The puncture with the sterilized hypodermic syringe may now be made and the desired fluid withdrawn. Three agar tubes should be at hand, and cylindrical roll cultures made after the manner of Esmarch already described. After the colonies have sufficiently developed, pure cultures of the various species included should be made in gelatin or other desired tubes in the usual manner.

To obtain cultures from solid lesions the following method should be pursued. The organ containing them should be removed entirely if *post mortem*, this being done with sterilized instruments and hands, care being taken that the organ in question shall be the first one removed. If transportation is necessary, it should be immediately enveloped in cloth soaked with a watery solution of corrosive sublimate, 1 part to 1000, and the culture should be made at the earliest possible moment. When ready to do this, a number of dissecting-knives with wooden handles, of small scissors, and of forceps large and small, should be thoroughly sterilized by heating in the Bunsen flame and subsequently allowed to cool. The hands should be sterilized, and the cultures now made as rapidly as possible in a still air. Three agar tubes should be at hand ready to make Esmarch plate cultures in the manner described. The organ is incised through the lesion by means of one of the sterilized knives by a long deep cut vertical to the surface of the organ. The cut surfaces are separated, and another cut through the lesion is made vertically to this surface with another sterilized knife. These new surfaces are separated, and, if possible, a third cut vertical thereto is again made with another sterilized knife. The first tube is now to be inoculated by means of the platinum needle from a desired point in the last surface, and the two other tubes are to be inoculated successively from the first in the manner already described; the tubes, after spreading the layer of agar over the entire surface and fixing it there by means of the block of ice, are set aside for the development of the colonies. Pure cultures are to be obtained from these in the usual manner. It is well to char the surface of the organ with a red-hot spatula, before puncture, when possible.

PATHOLOGICAL PROPERTIES OF BACTERIA.

The bacteria are now admitted to belong to the vegetable kingdom and to constitute the lowest forms thereof. Like the higher vegetables, a few species are noxious, most are harmless. The recognition of the noxious qualities of bacteria is to be made by actual experiment. This is done through contact, feeding, inhalation, and inoculation experiments. It should be unnecessary to remark that in performing these experiments two fundamental principles must be always kept in view: first, that the experiments be performed with absolutely pure cultures of the selected species, and, secondly, that they be performed in such a manner that no accidental contamination by other extraneous bacteria can occur. These two fundamental principles can be with considerable facility and perfection applied in the case of inoculation experiments. In the case of contact, feeding, and inhalation experiments, however, there is often unavoidably some association with other bacteria which naturally infest the surfaces in question and to some extent interfere with the purity of the experiments.

In inoculation experiments the bacteria may be inserted into the cutis, into the subcutaneous connective tissue, into the muscular or intermuscular tissue, into the depths of internal organs, etc., with comparatively little risk of accidental contamination. The preliminary procedure is very similar to that already described for obtaining material for culture from enclosed abscesses. The hair is to be first thoroughly removed from the surface, and the latter then sterilized in the manner mentioned, and in the case of experiments upon the lower animals it is well also to go a step further, in securing the absolute destruction of surface-germs by charring the point of the intended puncture by means of a hot plate of metal. Of course it is essential that all instruments used in the operation should be thoroughly sterilized by heat. The inoculation material may be introduced either by means of a hypodermic syringe or by means of the platinum wire loop inserted through the incision. After the operation is complete, the point of puncture or incision, including the charred surface if one be made, should be thoroughly protected by means of an impervious antiseptic covering.

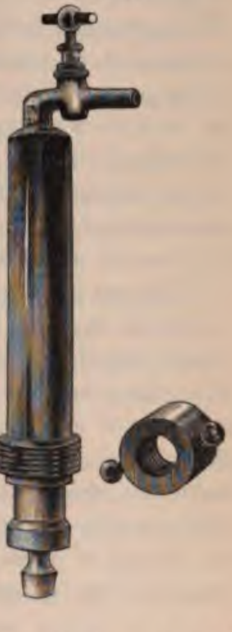
After a varying period of development or incubation, lesions corresponding to the affection in question should be produced in the animal experimented upon, *if the latter be* SUSCEPTIBLE *to the disease*, and *if the powers of the bacteria have not become* ATTENUATED: in these lesions the bacteria experimented with should be again recoverable by the usual methods.

ARTIFICIAL IMMUNITY FROM INFECTIOUS DISEASES.

With the knowledge of the characteristics of chicken-cholera, Pasteur also made known the important fact that in the microbe which was demonstrated to be the cause of the disease a means of artificial protection against subsequent attacks was furnished. Since this important practical discovery, the knowledge of analogous properties of a number of other specific microbes seems to indicate the existence of a law more or less general governing immunity from recurrent attacks of many, if not all, of the so-called infectious or contagious diseases which are known to leave behind them a greater or less, a longer or shorter immunity.

Whether this immunity is secured by direct and more or less mechanical action of the microbes themselves, or indirectly through the agency of a specific product of the specific microbe elaborated in the process of its growth or decay, was for a time a matter of purely theoretical dispute; but recent chemical examinations of the contents of pure cultures of various pathogenic and a few other bacteria have shown the existence therein of certain peculiar poisons not only possessing specific physiological action, but also having peculiar chemical properties and constitutions which ally them more or less closely to certain well-known poisonous vegetable *alkaloids.* These chemical bodies have been named *ptomaines.* Experiments demonstrating the activity of these bodies have been performed by numerous investigators in conjunction with those intended simply to demonstrate the pathogenic qualities of the specific microbes. Pathogenic bacteria proved to be the cause of the specific infectious disease to which they belong have been killed in their pure cultures by the action of heat. The culture medium then injected under all due precautions in certain instances likewise produces a series of symptoms characterizing the disease in question. Again, these pathogenic bacteria have been removed from the fluids of their pure cultures by means of filtration. This has been most perfectly done by means of the unglazed porcelain filter of Chamberland (Fig. 18). The filtered fluid, the sterility of which has been tested by culture experiments, has been inoculated with similar results. Furthermore, the chemical substances, or ptomaines, obtained from these culture fluids, when injected also cause analogous results. From these experiences it would seem probable that it is the alkaloid developed by the growth of the bacterium which is the specific active agent in the production of the disease.

FIG. 18.



In a certain number of specific infectious diseases it has been already demonstrated that artificial immunity can be produced by inoculations with cultures of the pathogenic microbe. Furthermore, the same end has been accomplished in a number of cases by using, instead, the products of pure cultures after the bacteria had been killed or removed by filtration. Again, the same effect has been accomplished for a number of diseases by the use of the ptomaines alone.

The value and the utility of this knowledge of the means of securing

s diseases are scarcely to be calcu-
rily begun investigations in this

ating infectious disease by inocu-
auses it is already considerable:
been given a more or less wide
ing against the destruction by in-
and smaller domestic animals. But
measures has been found by practi-
artificial production of the disease
hich may be the starting-point of a

r disappears and the objection based
the active living germ—the essential
—is substituted the employment of its
, which recent experimentation indi-
fficacious for the production of artificial

ledge already gained that the immunity
attack or an artificial production of the
the tissues of the specific microbe *through*
probably due both to the restraining effect
evelopment of the specific bacterium which
analogous to the effect of alcohol, generated
in arresting at a certain stage the growth of
, and to the establishment of a tolerance by
poisonous alkaloid. When these properties
ines shall become thoroughly known and well
and safe control of epidemic diseases will prob-
rtainty.

the future applications of the products of bacteria
a demography is scarcely now conceived, for most
m to indicate that the time may not be far distant
at the bedside will be enabled to control or cut short
ous or contagious disease already started, through the
he ptomaine elaborated by the growth of the specific
auses it, or of its direct physiological antidote, and the
er or veterinarian stop or prevent an epidemic, without
ating infectious centres, by inoculations of the chemical
ptomaines of the specific bacteria.

MATERNAL IMPRESSIONS.

By WILLIAM C. DABNEY, M.D.

From time immemorial there has been a popular belief that impressions made upon the mind of a pregnant woman would cause defects in the child with which she was pregnant at the time.

In the well-known instance related in Holy Writ there seems to have been no expectation on Jacob's part that the Almighty would interfere *directly* to cause the flocks of Laban to bring forth young "ringstraked, speckled, and spotted," but the device to which Jacob resorted is mentioned in such a way as to show a belief in maternal impressions at that time.

It was only comparatively recently, as the present age of scepticism approached and thinking men came to doubt the truth of those things which they could not understand, that the power of these maternal impressions began to be questioned. Whether maternal impressions bear a causative relation to fœtal defects is *one* question: *how* such impressions act in producing the effect is another and a totally different question.

I propose in this article to review the evidence upon which the theory of "Maternal Impressions" rests, and to see what grounds there are for the popular belief which is common to "all nations and kindred and people."

Much of the testimony upon which the theory is based is worthless; some because it is "hearsay," and some because the "witnesses" were not competent to form an opinion and to give an exact account of what they saw. All of this character I have endeavored to omit, and to take into consideration only that which we have from reliable sources.

Much confusion exists also as to the nature of the defects attributable to maternal impressions, as well as to the nature of the impressions themselves. It is important, therefore, that the subject should be systematically studied before any definite conclusions can be reached.

There are two classes of defects which have been attributed to Maternal Impressions,—*mental* defects, and *bodily* defects. These should be considered separately.

Mental Defects.—That mental defects in the child may be due to violent emotional disturbances of the mother during her pregnancy is generally acknowledged by those who have given most attention to the subject; and yet the cases of this character which have been recorded are few in number. History and tradition, it is true, furnish a number of instances of the kind, but they lack the weight which is given by careful scrutiny of all the attendant circumstances.

Sir Walter Scott, for instance, mentions that James I. could not stand the sight of a drawn sword, and a gallant gentleman who was knighted by James makes the same statement, and acknowledged afterwards that he was apprehensive at the time lest the king should let the sword fall upon his shoulders with the wrong side down. The monarch's apprehension was attributed to the fact that prior to his birth his mother had seen Rizzio cut down in her presence.

Dr. E. Séguin, well known as an authority on the subject of mental affections, stated[1] that it was a well-known fact that an officer of the First Napoleon, as to whose courage there could be no question, became pale when he saw a naked parlor sword, the explanation being that his father in a fit of jealousy had nearly killed his mother with such a weapon during her pregnancy with Napoleon's future officer.

Dr. Séguin reported at the same time another case which came under his own observation and care. A girl, who at the time he knew her was twelve or thirteen years old, was a congenital idiot; the other members of the family, which was a large one, were above the average in point of intelligence. The mother was pregnant with this idiotic child during the civil wars of Paris, and was harassed with anxiety for the safety of her husband.

In a paper read before the Obstetrical Society of London, May 7, 1884, Dr. Arthur Mitchell, Commissioner in Lunacy for Scotland, stated that in 6 cases out of 443 in which an effort was made to establish a cause for idiocy, the influence of maternal impressions was clearly traceable.

The well-known statement of Baron Larrey with respect to the siege of Landau in 1793 is most striking, and yet it is by no means certain that the results were attributable solely to the fear and distress of the women, for the privation and suffering were also extreme. Of 92 children born in the district soon afterwards, 16 died at birth, 33 died within ten months, 8 became idiotic (or rather it should have been stated, perhaps, *were* idiotic), and 2 were born with several bones broken.

So far as we are able to judge from the limited data at hand, it would appear that a *prolonged* impression is far more liable to influence the fœtus than a short one, even though the latter may be more violent: it is especially difficult to reach any conclusion on this point, however, because in many instances a sudden and violent shock was followed by a long period of distress. In Mitchell's cases, for example, it is to be observed that in four of the six the women suffered from prolonged distress in consequence of the death of a near relative, and in one of the other two, while the fright was but temporary, the impression was very enduring.

---

[1] Phila. Med. Times, December 23, 1876.

The *character* of the impression is of great importance. *Anxiety* and *grief* seem to hold the first place, and *fear* the second, with respect to the frequency with which maternal impressions influence the mental characteristics of the child. So far as I can learn, no case of sudden or excessive joy has produced any appreciable effect.

The data are insufficient also to establish the *period* of *pregnancy* at which maternal impressions are most liable to cause mental defects in the child; and additional difficulty is placed in our way here by the circumstance that the impressions are usually prolonged. It would seem most probable, however, that the mind of the child would be most readily affected in the later months of pregnancy. "The permanent cerebral convolutions are formed from the seventh month onward" (Landois).

It is useless to speculate upon the manner in which maternal impressions influence the mental development and characteristics of the child. Upon this point we *know* absolutely nothing, and mere speculations would be out of place here.

We know but little also as to the *frequency* with which mental defects in the child are to be attributed to impressions made upon the mother during her pregnancy. Mitchell found but 6 cases in 443 in which the connection was clearly manifest. Certain it is that in proportion to the number of cases in which women receive violent mental shocks or are subjected to great grief and anxiety during pregnancy, the number of cases in which the mental faculties of their children are impaired is exceedingly small. During our late civil war, for example, the anxiety and grief of the women whose male relatives were in the field were beyond description, yet, so far as I can learn, there was no perceptible increase in the proportion of idiotic or otherwise mentally defective children among those who were born in those troublous times.

Bodily Defects.—Far more cases of bodily than of mental defect have been attributed to maternal impressions; and the reason for this is obvious. The bodily defect is apparent at the birth of the child; the mental defect is obvious only at a later period, when the child's mind should have undergone development; and by that time the various causes of anxiety or mental distress during pregnancy have probably been forgotten.

As has been mentioned heretofore, the doctrine of maternal impressions, so far as the production of *bodily* defects is concerned, has met with vigorous opposition. Some of those who have been most strenuous in their opposition have, however, acknowledged that malformations may be caused by psychical impressions, such as "unaccustomed agitation and fright" (Foerster).[1]

Rokitansky, whose vast experience and sound judgment give weight

---

[1] Die Missbildungen des Menschen, p. 4.

[illegible] arguments, says, "The question whether mental emotions do in-[illegible] development of the embryo must be answered in the affirma-[illegible] undoubtedly have occurred of such maternal impressions—[illegible] particularly—when violent giving rise to malformations." [1] He [illegible] that it is just *conceivable* that the connection may be acci-[illegible]. He refers also to a fact with which all anatomists are familiar, that [illegible] vascular system are more common than those of any other [illegible]. But the heart and blood-vessels are so far shut in from [illegible] observation, that the influence of maternal impressions in the produc-[illegible] anomalies has scarcely been noticed. Peacock alone of all the [illegible] subject calls attention to the probable connection between [illegible] made upon the pregnant woman and congenital defects of the [illegible].

[illegible] strongest arguments against the influence of maternal im-[illegible] the child in utero, in the opinion of the opponents of the [illegible], is that all deformities are due to errors of development. [illegible] difficulties in the way of this objection. It presupposes [illegible] which have been attributed to maternal impressions were [illegible] development" or deformities in the common acceptance of the [illegible] the case; we shall see that in a considerable proportion [illegible] have been reported by reliable physicians there was no [illegible], but a mark or marks which evidently occurred late [illegible] the development of the child was practically complete. [illegible] a very large proportion—a large majority, indeed—of [illegible] were plainly due to errors of development does not in [illegible] against the doctrine of maternal impressions, provided it [illegible] the impression was made at a period of pregnancy when [illegible] the deformed part of the body was not complete. It is [illegible] how maternal impressions produce deformities, but [illegible] produce them.

[illegible] has to be considered from a number of different [illegible] definite conclusions can be reached; and it will be well to [illegible] farther, what those different points are.

[illegible]:

[illegible] pregnancy at which the impression was made. This [illegible] determine whether the impression was made at a [illegible] development was possible.

[illegible] the defect in the child to the object making the impression [illegible].

[illegible] necessary for the woman to be *conscious* of the impression [illegible] result.

[illegible] of the character of the impression made before the birth of the child, and the proportion of cases in which such ante-partum statement has been made.

---

[1] [illegible] Anatomy, vol. i. p. 11.

[2] Malformations of the Heart, pp. 165, 166.

5. The channels through which impressions are received by the mother.

6. The duration of the impression necessary to produce the effect.

7. The character of the impressions which are most liable to produce results.

8. A brief consideration of the objections which have been urged against the doctrine of maternal impressions.

9. The practical deductions to be drawn from a consideration of the subject.

For convenience of reference I shall tabulate the 90 cases which have been collected from various sources, and which seem to me worthy of credence. I have already stated that many were excluded from the list because they did not seem to me to be reported with sufficient clearness to be worthy of belief.

The table will show (1) the name of the reporter, (2) the journal or work in which the report may be found, (3) the period of pregnancy at which the impression was made, (4) the cause or nature of the impression, and (5) the nature of the defect in the child.

It will be observed that nearly all the cases included in the General Table have been reported within the past twenty years. This is not due to the absence of reports of cases prior to that time, but to the greater accuracy and care with which cases of every character have been reported during the past quarter of a century. Those who are in the habit of really *studying* the reports of cases have doubtless been struck with the fact that the character of these reports is very different now from what it formerly was. Of late the *facts* connected with cases have been reported without being obscured by theories and conjectures: hence these cases are far more valuable, for purposes of study and comparison, than those reported in former times.

During the latter half of the eighteenth and the first half of the nineteenth century there appeared quite a number of works on Maternal Impressions: the object of nearly all was to show that there was no ground for the belief in the doctrine which was evidently prevalent at that time. These works are valueless at the present day, because the arguments which they contain are based not upon facts, but upon theories, many of which are now known to be false.

I desire to state with reference to the cases reported in the General Table that as far as possible I have examined the reports in the journals or periodicals where they were originally published, and I wish to acknowledge my obligations to the officers and employees of the "Library of the Surgeon-General's Office" for courtesies shown me while prosecuting my studies there. In a few instances it was impossible to get access to the original reports, and I was compelled to take such information as was furnished in abstracts of those reports, published in other journals.

GENERAL TABLE.

No. of Case.	Reporter.	Where Reported.	Period of Pregnancy.	Cause or Nature of Impression.	Nature of Defect.
I.	Brydon.	Brit. Med. Jour., July 17, 1886.	2 months.	Mother stated before knowing the nature of the defect that she had seen a picture of a child without a neck.	The child had no neck.
II.	A. M. Brown.	Brit. Med. Jour., Feb. 20, 1886.	Not stated.	The mother had her ears pierced, and was much disturbed afterwards for fear of effect on the child.	Child born with holes in the lobules of the ears.
III.	T. Graham.	Brit. Med. Jour., March 6, 1886.	Not stated.	The mother was frightened by a rat.	Three fingers of the right hand webbed; nails like claws.
IV.	Brydon.	Brit. Med. Jour., April 3, 1886.	3 months.	Mother dreamt her big toe was bitten off by a rat.	Child born with one big toe missing.
V.	Barrett.	Brit. Med. Jour., April 10, 1886.	From time of marriage.	The milkman, whom the mother saw daily, had one finger amputated.	Child had only four fingers on one hand.
VI.	Addenbrooke.	Brit. Med. Jour., May 13, 1871.	5 or 6 months.	Woman saw her mother suddenly stricken with paralysis on one side.	Child born with facial paralysis on one side.
VII.	Addenbrooke.	Brit. Med. Jour., May 13, 1871.	5 or 6 months.	Woman saw her mother suddenly stricken with paralysis on one side.	Child born with facial paralysis on one side.
VIII.	Huntley.	Brit. Med. Jour., Mar. 13, 1875.	4 months.	Saw a child killed, the teeth being driven up into the jaw.	Child born with hare-lip.
IX.	Graham.	Brit. Med. Jour., Jan. 18, 1868.	4 months.	Frightened by a rabbit, the two subsequent children normal.	Child born with hare-lip.
X.	Bolton.	St. Louis M. and S. Jour., Oct. 1881.	4 months.	Woman saw an albino at a circus.	Child born with a patch of white hair on its head.
XI.	Bolton.	St. Louis M. and S. Jour., Oct. 1881.	3 months.	Mother saw a man with hare-lip, and was much impressed. (Previous children normal.)	Hare-lip.
XII.	Bolton.	St. Louis M. and S. Jour., Oct. 1881.	3 months.	Young opossum thrown in woman's lap; she was much startled. No *expectation* of defect.	Child had ear like an opossum.
XIII.	Williams (quoted by Dewey).	St. Louis M. and S. Jour., Dec. 1881.	Before and during pregnancy.	Mother formed a most ardent attachment for a lady living in her house, who had one leg shorter than the other, and one blue and one dark eye.	Child had precisely similar peculiarities.
XIV.	Fairbrother.	St. Louis M. and S. J., Aug. '81.	3 months.	Mother saw a man with two fingers of the right hand amputated.	Child had only three fingers on the right hand.

XV.	Rowland.	St. Louis M. and S. Jour., Aug. 1881.	3 months.	Mother saw a child with an arm crushed by a cog-wheel.	Child had singular marks as if made by cog-wheel on arm and shoulder.
XVI.	Furman.	St. Louis M. and S. Jour., May 5, 1880.	2 or 3 months.	Mother frightened by a jackass.	Child had head and ears like a jackass.
XVII.	Furman.	St. Louis M. and S. Jour., May 5, 1880.	2 months.	Mother saw a terrapin killed; was greatly shocked.	Child had claws like a terrapin.
XVIII.	Trenholme (quoted by Furman).	St. Louis M. and S. Jour., May 5, 1880.	4½ months.	Mother saw her child brought home with forehead badly cut; was greatly distressed.	Child born with a mark on the corresponding part of its forehead.
XIX.	Trenholme (quoted by Furman).	St. Louis M. and S. Jour., May 5, 1880.	Not stated.	Mother saw a man with both legs amputated; was greatly impressed.	Child born with both legs absent.
XX.	Maughs.	St. Louis M. and S. Jour., Dec. 1882.	4 weeks.	Woman dreamt her child would be hermaphrodite; so informed her husband at the time.	Child was hermaphrodite. (Form of sexual defect not mentioned.)
XXI.	Scott.	St. Louis M. and S. Jour., Dec. 1882.	2 months.	Mother frightened by the sight of the frog-faced woman.	Child "like the frog-faced woman."
XXII.	Stockard.	St. Louis M. and S. Jour., July, 1881.	2 months.	Mother saw a fœtus without ears.	Child had only one ear.
XXIII.	Atkinson.	Philada. Medical Times, Aug. 8, 1874.	Not stated.	Mother frightened by lightning, and grasped left arm just below the elbow.	Left arm ended with rudimentary fingers just below the elbow.
XXIV.	W. L. Atlee.	Philada. Medical Times, Aug. 8, 1874.	"Early period."	Mother saw a man with hare-lip.	Hare-lip.
XXV.	W. T. Taylor.	Philada. Medical Times, Feb. 11, 1882.	"Early period."	Woman visited her mother, who had cancer between the eyes; greatly distressed.	Child born with large nævus between the eyes.
XXVI.	D. W. Prentiss.	Philada. Medical Times, March 11, 1882.	"Early period."	Woman saw friend with large nævus on face; mother slapped herself on right buttock, and said if child was marked it would be there.	Child born with large nævus on right buttock.
XXVII.	W. T. Taylor.	Philada. Medical Times, Nov. 25, 1876.	"Early period."	Mother saw beggar with fingers of one hand missing; greatly impressed.	Child had no fingers on the right hand.

GENERAL TABLE.—*Continued.*

No. of Case.	Reporter.	Where Reported.	Period of Pregnancy.	Cause or Nature of Impression.	Nature of Defect.
XXVIII.	W. T. Taylor.	Philada. Medical Times, Nov. 25, 1876.	"Early period."	Mother saw personification of the devil.	Child had very short forearms, and only two long fingers and a thumb on each hand.
XXIX.	W. T. Taylor.	Phila. Med. T., Nov. 25, 1876.	During pregnancy.	Mother looked with "unaccountable delight" on her father-in-law's bald head.	Child had a bald spot on its head.
XXX.	W. T. Taylor.	Phila. Med. T., Nov. 25, 1876.	"Early period."	Woman dressed wounds on her husband's thighs.	Child had scars at corresponding parts of its thighs.
XXXI.	Editorial.	Philada. Medical Times, Dec. 2, 1882.	"Early period."	Woman saw girl pulling a little boy's penis violently; alarmed lest she should pull it off.	Child born without prepuce.
XXXII.	Hammond.	Quart. J. Psych. Med., Jan. 1868.	2 months.	Woman saw her husband with severe wound in his face; greatly shocked.	Scar on face corresponding in site to father's injury.
XXXIII.	Hammond.	Quart. J. Psych. Med., Jan. 1868.	Not stated.	Woman dreamt that she saw a man with part of one ear missing.	Child had part of one ear missing.
XXXIV.	Burchman.	Med. and Surg. Reporter, July 30, 1881.	"Early period."	Woman saw a man with the right thumb amputated.	Thumb attached by slender pedicle.
XXXV.	Doty.	Med. and Surg. Reporter, July 2, 1881.	4 months.	Woman attacked by a ram and greatly alarmed.	Long head covered with black wool, which extended down the back of the neck and on the arms; large round eyes; two large front incisor teeth; cry bleating.
XXXVI.	Heywood Smith.	Med. and Surg. Reporter, May 31, 1881.	3 months.	Mother frightened by a monkey.	Girl with face singularly like a monkey's.
XXXVII.	Purefoy.	Med. and Surg. Reporter, May 31, 1881.	3 or 4 months.	Woman attempted to raise a calf by hand, of which the right ear, right eye, and both forelegs were absent.	No right ear; no right eye, orbit indicated by slight depression; arm and forearm on right side absent, but there was an abortive hand attached to the scapula.
XXXVIII.	Rawlins.	Med. and Surg. Reporter, June 11, 1881.	Not stated.	Mother impressed by sight of man with one leg.	Leg missing from middle of the thigh.

XXXIX.	Hill.	Med. and Surg. Reporter, Jan. 27, 1877.	Not stated.	Woman saw a leg amputated.	One leg from knee down, and fingers of one hand, missing.
XL.	Baker.	Obstetric Gazette (Cin.), Feb. 1879.	"Early period."	Mother lived next door to a man with hare-lip, and was apprehensive lest child should have same defect.	Hare-lip.
XLI.	Baker.	Obstetric Gazette (Cin.), Feb. 1879.	2 months.	Mother impressed by sight of man with hare-lip; very apprehensive lest child should be similarly deformed.	Hare-lip.
XLII.	——	Obstetric Gazette (Cin.), Feb. 1879.	2 months.	Mother saw brother's body; head greatly swollen and eyes protruding (killed in railway accident).	Immense head; large eyes; space of about two inches above eyebrows and ears where bones were wanting.
XLIII.	Delacaux (quoted by Hammond).	Quart. J. Psych. Med., Jan. 1868.	2 months.	Mother impressed by sight of man with cleft palate.	Cleft palate.
XLIV.	Daly.	London Lancet, Jan. 16, 1869.	During first three months.	Woman greatly worried by rats, and feared child would be like one; asked at its birth if it was.	No neck; no face; long snout like a rat in a line with the body. (Child born dead.)
XLV.	Griffith.	London Lancet, Nov. 3, 1877.	"Early period."	Woman frightened by a decrepit old man; feared child would be deformed.	Child deformed (nature of deformity not stated).
XLVI.	Goodell.	Amer. J. Obstetrics, May, 1871.	2 months.	Mother greatly excited by her husband assisting at circumcision; expected "mark."	No prepuce, but granulating surface like recent circumcision.
XLVII.	Wright.	Amer. J. Obstetrics, Jan. 8, 1878.	7 weeks.	Mother visited brother in jail for serious crime. Saw prisoner brought in with manacles on hand and feet tied together. Greatly shocked and impressed.	Child born at 5 months; hare-lip; fibrous cord connecting one hand with the other and similar cord connecting the feet.
XLVIII.	Jameson.	Amer. Practitioner, Aug. 1878.	Before marriage.	Mother worked in factory with man with cleft palate; apprehensive during pregnancy lest child should have similar defect.	Cleft palate.
XLIX.	Jameson.	Amer. Practitioner, Aug. 1878.	"Early period."	Mother saw beggar with hare-lip; very anxious lest child should have similar deformity, and expressed fears before labor. No heredity.	Hare-lip.
L.	Storer.	Amer. J. Med. Sciences, Apr. 1853.	"Beginning."	Saw a hen's leg knocked off with a stone; greatly excited about it.	One foot missing.
LI.	Adams.	Amer. J. Med. Sciences, Apr. 1853.	"Early period."	Woman dressed stump of amputated arm for her brother.	One arm absent.

GENERAL TABLE.—*Continued.*

No. of Case.	Reporter.	Where Reported.	Period of Pregnancy.	Cause or Nature of Impression.	Nature of Defect.
LII.	[illegible]	Jour. de Méd. Chir. et Pharm., Oct. 1880.	4 months.	In a scuffle with a man who attempted undue liberties with her the woman noticed that he had but one ear.	Child had but one ear.
LIII.	Wendel.	Louisville Med. News.	Not stated.	Woman struck by appearance of stump; no expectation of defect in the child.	Child had only one arm.
LIV.	Wendel.	Louisville Med. News.	Not stated.	Woman struck by appearance of stump; no expectation of defect in the child.	Child had only one arm.
LV.	Wall.	New York Medical Record, 1881.	"Early period."	Woman impressed by sight of child with double hare-lip.	Two children in succession born with hare-lip. (No heredity.)
LVI.	Roth.	Virchow's Archiv, Band xci. Heft 3.	3d month.	"Error" (or impression). (Virchow.)	Spina bifida.
LVII.	Roth.	Virchow's Archiv, Band xci. Heft 3.	3d month.	"Error" (or impression). (Virchow.)	Hare-lip.
LVIII.	Roth.	Virchow's Archiv, Band xci. Heft 3.	2d month.	"Error" (or impression). (Virchow.)	Hare-lip.
LIX.	Roth.	Virchow's Archiv, Band xci. Heft 3.	3d month.	"Error" (or impression). (Virchow.)	Hare-lip.
LX.	Roth.	Virchow's Archiv, Band xci. Heft 3.	3d month.	"Error" (or impression). (Virchow.)	Hare-lip.
LXI.	Roth.	Virchow's Archiv, Band xci. Heft 3.	3d month.	Very severe shock.	Hare-lip.
LXII.	Roth.	Virchow's Archiv, Band xci. Heft 3.	2d or 3d month.	Impression. (Virchow.)	Hare-lip.
LXIII.	Roth.	Virchow's Archiv, Band xci. Heft 3.	2d or 3d month.	Impression. (Virchow.)	Hare-lip.

LXIV.	Roth.	Virch. Archiv, Bd. xci. Heft 3.	3d month.	Impression. (Virchow.)	Hare-lip.
LXV.	Roth.	Virch. Archiv, Bd. xci. Heft 3.	3d month.	Impression. (Virchow.)	Cleft palate.
LXVI.	Roth.	Virch. Archiv, Bd. xci. Heft 3.	2d or 3d month.	Stab in the neck.	Cystic tumor of the neck.
LXVII.	Roth.	Virch. Archiv, Bd. xci. Heft 3.	4th month.	Sight of an injury.	Nævus.
LXVIII.	Roth.	Virch. Archiv, Bd. xci. Heft 3.	Not stated.	Slight shock.	Hare-lip, which healed of its own accord.
LXIX.	Roth.	Virch. Archiv, Bd. xci. Heft 3.	Not stated.	"Impression."	Hare-lip.
LXX.	Minot.	Boston M. and S. Jour., Nov. 24, 1870.	"Early period."	Woman saw man with all the fingers of one hand missing; expressed apprehension for her child.	No fingers (only a thumb) on one hand; exactly like object causing impression.
LXXI.	O'Reilly.	Med. Herald, Aug. 1883.	1 month.	Woman saw, at circus, a hydrocephalic cat preserved in alcohol.	Hydrocephalus.
LXXII.	Paget (quoted by T. Smith).	London Lancet, Aug. 16, 1867.	3 months.	Monkey jumped on woman's back in the street; alarmed her greatly.	Left upper extremity and greater part of neck and body covered with coarse hair one or two inches long; "whole shoulder and limbs much like a monkey's."
LXXIII.	Sedgwick.	Proc. Roy. Med. and Chir. Soc. London, vol. viii.	"Early period."	Woman had seen a man with one finger missing, and the second and third fingers of one hand united and having a single nail. Stated apprehensions before labor.	Child had precisely similar defects.
LXXIV.	A. Thompson.	Trans. Obst. Soc. London, 1877.	17 weeks.	Saw friend's breast roughly handled by surgeon.	Child had four mammæ.
			20 weeks.	Saw horse killed with pole-axe.	Child had scar on its head.
LXXV.	——	St. Thomas' Hos. Reports, vol. xiv.	Beginning.	Woman saw representation of the "two-headed nightingale."	Child precisely like the "two-headed nightingale."
LXXVI.	B. R. Johnston.	Brit. Med. Jour., Mar. 28, 1885.	Beginning.	Woman saw the "two-headed nightingale," and fainted at the sight.	Child united from neck to hips in front.
LXXVII.	Wilson.	Obst. Jour. Gr. Brit., June 15, 1880.	Last few days of pregnancy.	Woman received burns upon her hand.	Child born with fresh-looking blebs upon its hand, corresponding in position to the mother's burns.
LXXVIII.	Swift.	New York Med. Jour., Oct. 9, 1886.	8 months.	Woman greatly shocked by seeing her son with injury to his thumb; nail black. No expectation of defect in child.	Child born three weeks after with thumb-nail black, and nail came off same time as its brother's.

GENERAL TABLE—*Continued.*

NUMBER.	REPORTER.	WHERE REPORTED.	PERIOD OF PREGNANCY.*	CAUSE OR NATURE OF IMPRESSION.	NATURE OF DEFECT.
LXXIX.	Fordyce Barker.	Gynæcol. Trans., 1886.	Early period.	Favorite daughter had her ears bored; mother had no expectation of defect	Holes in the lobules of the ears.
LXXX.	Fordyce Barker.	Gynæcol. Trans., 1886.	Beginning.	Mother saw three hare-lip people at dinner-table; greatly shocked. No heredity.	Hare-lip.
LXXXI.	Fordyce Barker.	Gynæcol. Trans., 1886.	Beginning.	Woman had her left hand violently pressed by her husband's elbow; finally fainted.	First and second phalanges of all the fingers and the thumb of the left hand missing.
LXXXII.	Brayton Ball.	Gynæcol. Trans., 1886.	2 or 3 months.	Woman saw a child with large protruding tongue; impression strong.	Child had large tongue protruding from mouth.
LXXXIII.	T. W. Shaw.	Gynæcol. Trans., 1886.	6 or 8 weeks.	Mother saw her child's hand run through with a pitchfork, and thought it was cut off at the middle.	Hand missing from about the middle.
LXXXIV.	J. A. Robeson.	Gynæcol. Trans., 1886.	Paternal impression (?).	Man's first wife killed in railway accident; thighs cut off. Married again.	First child of second wife had thighs and legs missing from same point.
LXXXV.	Busey.	Gynæcol. Trans., 1886.	Few weeks.	Woman frightened by attempts of a pet squirrel to bite her.	Child had compact mass of hair, extending back from eyebrows over head and neck, "closely resembling a squirrel," also two well-developed lower incisor teeth.
LXXXVI.	Fearn.	Trans. Alabama Med. As., 1850.	Not stated.	Woman saw metacarpal bone removed from her husband's hand.	Corresponding bone in child's hand missing.
LXXXVII.	Parker.	Gynæcol. Trans., 1886.	3 months.	Woman saw a pig's belly struck against a projecting piece of fence and ripped open; greatly shocked.	Anterior abdominal walls a thin film.
LXXXVIII.	Stewart.	Gynæcol. Trans., 1886.	Not stated.	Woman saw a man with supernumerary fingers.	Child had supernumerary fingers.
LXXXIX.	———	Gynæcol. Trans., 1886.	Not stated.	Shocked at sight of her husband with part of his scalp torn off.	Bald patch on child's head.
XC.	Hunt.	Amer. J. Med. Sci., Jan. 1881.	8½ months.	Mother burnt extensively on body and limbs; child born about thirty-six hours afterwards.	Child born dead. Large, fresh-looking blebs corresponding in site to burns of mother.

NOTE.—The expression "period of pregnancy" refers to the time at which the impression was made.

An examination of the table will show that these 90 cases may be divided into several different classes according to the part of the body involved; and with a view of studying the defects, with especial reference to the development of the different parts of the body, I shall make the following classification:

				Cases.
1.	Hare-lip or cleft palate			21
2.	Defects of the upper extremities			21
3.	"	"	lower "	8
4.	"	"	ears	8
5.	"	"	eyes	4
6.	"	"	head, neck, and trunk	20
7.	"	"	skin and hair	15

In several instances there was more than one defect, and in such cases, as a rule, each defect has been placed in its appropriate class, so that it will be observed that the sum of the different defects is 97, while the total number of cases is but 90.

I shall arrange these different classes of cases in tabular form, for greater convenience of reference and study.

CASES OF HARE-LIP AND CLEFT PALATE.

Case.	Period of Pregnancy.	Case.	Period of Pregnancy.
I.	4 months.	XII.	3 months.
II.	4 months.	XIII.	2 months.
III.	3 months.	XIV.	3 months.
IV.	Early period.	XV.	3 months.
V.	Early period.	XVI.	3 months.
VI.	2 months.	XVII.	2 or 3 months.
VII.	2 months.	XVIII.	2 or 3 months.
VIII.	7 weeks.	XIX.	3 months.
IX.	Before marriage.	XX.	3 months.
X.	Early period.	XXI.	Commencement of pregnancy.
XI.	Early period. (Occurred in two successive pregnancies.)		

If we examine now these cases of defects of structure of the lips and palate, it will be observed that in all but 2 of the 21 cases the impression was made within the first *three* months of pregnancy, and in the other two it was made during the *fourth* month. This is a point of great importance; for, while it is impossible with our present light to understand *how* these defects can be produced by maternal impressions, it has been justly claimed by the opponents of the doctrine that it is well-nigh, if not altogether, impossible to conceive how a defect of development could occur when the development itself was practically completed. That a "retrograde" process of absorption may occur is within the bounds of possibility, certainly, but it is far more improbable than a failure of proper development.

Roth quotes Meckel with respect to bodily defects brought about by maternal impressions, to the effect that "it is impossible that such a causal connection could exist later than the first months of intra-uterine life;" and Roth himself, who is a pronounced believer in the power of maternal impressions, says that the most probable time in which they are effective is during the first three months, or, more exactly, from the second to the third month, for after the third month the plates become closer to each other, so that a separation at that time would scarcely be possible.

The remarkable experimental investigations of Dareste[1] on the artificial production of monsters in the lower animals have proved beyond a reasonable doubt that these monstrosities have their origin in the early periods of embryonic life; and it is an interesting and suggestive fact that the two conditions which would most surely affect the fœtal or rather the embryonic blood-supply were just those which always modified the processes of evolution,—namely, (1) contact of the egg with a source of heat at a point near the cicatricula but not coinciding with it, and (2) the production of temperatures slightly above or below that of normal incubation.

It is important to remember, however, what has already been remarked, that there is a great difference between those defects of *development* which have been attributed to maternal impressions and certain defects which have been attributed to the same cause, such as scars, defects of the hair, and certain nervous defects, which may and probably do have their origin at a much later period of intra-uterine life. The hair, as we shall see hereafter, is developed quite late at any rate, and it is entirely possible, and probable even, that such defects as facial paralysis, of which two most striking cases are given in the General Table, would occur at a late period of pregnancy. Furthermore, with respect to scars and marks, it is well-nigh certain that they would occur late; for not only is the skin comparatively late in developing (about the fourth month), but we shall see that there is almost conclusive proof that these scars are or *may be* the result of disturbances of an inflammatory character. We shall see also farther on that as a matter of fact the scars, etc., which have been attributed to maternal impressions have had their supposed cause in operation, as a general rule, at a much later period of pregnancy than the "deformities" in the common acceptation of the term; such, for example, as hare-lip and cleft palate. Indeed, this seems to me an argument in favor of the truth of the doctrine of maternal impressions; for in many instances the persons who have reported these cases have been manifestly ignorant of the details of embryology and teratology, and yet they have, as a rule, attributed defects of *development* to impressions made at an *early* period of pregnancy, and "scars" and "marks" to similar impressions made at a much later period.

With respect to the special forms of deformity or defective development which we are considering just now,—hare-lip and cleft palate,—we are told by embryologists that the superior maxillary processes of the first branchial arch come together during the first eight or ten weeks of fœtal life, and at the ninth week or soon afterwards the hard palate is closed and on it rests the septum of the nose.

---

[1] Comptes-Rendus, Nov. 3, 1873.

A glance at the table will show that in the main the maternal impressions which produced or which were supposed to have produced these deformities occurred at this very period.

Roth reports a case which I have included in the General Table which, viewed from a developmental stand-point, would find its appropriate place here: it was a case of tracheal cyst of the neck; the "maternal impression" was made at the second or third month of pregnancy, and was due to seeing a person stabbed in the neck. It is scarcely possible, however, to attribute to the influence of a maternal impression a case reported by Mr. Ashburton Thompson to the Obstetrical Society of London on April 4, 1877. A woman seven *months* pregnant went to the door to answer a knock: she was shocked to see a man who could not speak, and from whose windpipe projected a tracheotomy-tube. Two months afterwards the child was born with a tracheal cyst and a fistulous opening leading into it. It seems scarcely possible that there could have been any connection between the impression and the defect in this instance, on account of the evident error of development to which the defect was due and the late stage of pregnancy at which the impression was made.

DEFECTS OF THE UPPER EXTREMITIES.

CASE.	PERIOD OF PREGNANCY.	NATURE OF DEFECT.
I.	Not stated.	Three fingers webbed; nails like claws.
II.	From marriage.	Only four fingers on one hand.
III.	3 months.	Three fingers on one hand.
IV.	3 months.	Marks like those of cog-wheel on shoulder.
V.	2 months.	Claws like a terrapin.
VI.	Not stated.	Rudimentary fingers, below left elbow.
VII.	Early period.	No fingers on one hand.
VIII.	Early period.	Only two long fingers and thumb on each hand.
IX.	Early period.	Thumb attached by slender pedicle.
X.	3 or 4 months.	Right arm and forearm absent; abortive hand attached to scapula.
XI.	Not stated.	Fingers of one hand missing.
XII.	7 weeks.	Fibrous cord connecting the hands.
XIII.	Early period.	One arm absent.
XIV.	Not stated.	One arm absent.
XV.	Not stated.	One arm absent.
XVI.	Early period.	No fingers (only the thumb) on one hand.
XVII.	Early period.	One finger missing, two webbed.
XVIII.	Commencement.	Phalanges on one hand missing
XIX.	6 or 8 weeks.	Hand missing from about the middle.
XX.	Not stated.	Supernumerary fingers.
XXI.	Not stated.	One metacarpal bone missing.

In the table of cases of hare-lip and cleft palate it was unnecessary to state the special deformity in each case, but in the other tables it has seemed best that this should be done.

There are 21 cases in the table of defects of the upper extremity, but three of these might with propriety be placed in a different category,—namely, Case IV., in which there were marks like those made by a cog-wheel on the arm and shoulder, which should probably be placed more correctly with the "defects of the skin and hair;" Case IX., in which the thumb was attached by a slender pedicle, which was almost certainly a secondary defect and not due to an error of development; and Case XII., in which there were fibrous bands uniting the two hands and spreading out over one so as to bind the fingers together. A case somewhat like the one just mentioned has been reported by Kidd,[1] in which the bands were certainly of secondary origin and had caused amputation of one leg. Indeed, with respect to the absence of limbs or parts of limbs much caution is necessary before arriving at a positive conclusion as to the cause. In many cases the presence of rudimentary fingers or toes leaves no doubt that the deformity is due to a defect of development, but in other instances there can be just as little doubt that the defects are due to intra-uterine amputation by bands or by the umbilical cord.

Leaving out, then, the three cases which I have already mentioned, there remain *eighteen* of deformities of the upper extremity; of these, *eight*—namely, I., V., VI., VIII., X., XVII., XX., and XXI.—are clearly due to errors of development, and cannot be attributed to the effects of constriction by bands or by the cord. In four of these eight cases the period of pregnancy at which the impression was made is not stated; in two others it was made at an "early period" of pregnancy, in another at the second month, and in the other at the third or fourth month.

Of the ten cases in which a part or the whole of a limb was missing,—and which it is best to consider separately, because such a defect may have been secondary,—the period of pregnancy at which the impression was made is not stated in *three;* in all the others it occurred at an early period, in one only later than three months, and in five it is distinctly stated to have been within eight weeks.

If we turn now to the development of the upper extremity, it will be found that the limbs are apparent at a very early stage,—from the thirty-fifth to the forty-second day,—but the humerus shows its first centre of ossification (in the shaft) at the eighth or ninth week, the radius and ulna at the third month, the metacarpal bones and phalanges at the end of the third month.

These facts render it quite evident that so far as the period of pregnancy is concerned there is nothing to justify the conclusion that the deformities in question are *not* due to maternal impressions. On the contrary, it will be well to reiterate here what has been previously stated, that the connection is rendered more probable by the fact that the observers were often not aware of the importance of the correspondence between the period of pregnancy and the time of the impression, and hence were not biassed by this circumstance at least in arriving at the conclusions at which they did.

It is well enough to state here, what might with propriety have been stated earlier, that there may be an *excess* as well as an *arrest* of development. Supernumerary fingers furnish an illustration of the former.

---

[1] Obst. Jour. of Great Britain, vol. ii. p. 737.

DEFECTS OF THE LOWER EXTREMITIES.

CASE.	PERIOD OF PREGNANCY.	NATURE OF DEFECT.
I. . .	3 months.	Big toe missing.
II. . .	Before and during pregnancy.	One leg shorter than the other.
III. . .	Not stated.	Both legs absent.
IV. . .	Not stated.	Leg absent from middle of thigh.
V. . .	Not stated.	One leg, from knee down, absent.
VI. . .	7 weeks.	Fibrous cord uniting feet.
VII. . .	Commencement.	One foot absent.
VIII. . .	Paternal impression?	Both legs missing from middle of thighs.

The same remarks are applicable to defects of the lower extremities as to defects of the upper.

Of the eight cases in the table it is possible—from the entire absence of rudimentary parts—that the defect in all except one (Case II.) is attributable to an amputation of the member by the cord or by bands.

In Case II. one leg was much shorter than the other, a common enough defect in a slight degree, but the singular features of this case were that in addition to this shortness of one leg the child had one blue and one brown eye, precisely similar peculiarities to those of the lady by whom its mother had been so much impressed.

In three of the cases the period of pregnancy when the impression was made is not mentioned, but in all the others it was *early*, only one being as late as three months. One of these cases is most remarkable, and suggests, what reflection will show is not so improbable as would appear at first sight, that the impression was made upon the father and by him communicated to the child through the spermatozoa. It is equally probable, however, that the second wife dwelt upon the tragic end of her predecessor, and that the impression was made in this way. (See Case LXXXIV. of the General Table.)

The period of development of the lower limbs is nearly the same as that of the upper, and requires no comment.

DEFECTS OF THE EARS.

CASE.	PERIOD OF PREGNANCY.	NATURE OF DEFECT.
I.	Not stated.	Holes in the lobules of the ears.
II.	3 months.	Ear like an opossum.
III.	2 or 3 months.	Ear like a jackass.
IV.	2 months.	One ear absent.
V.	Not stated.	Part of one ear absent.
VI.	3 or 4 months.	One ear absent.
VII.	4 months.	One ear absent.
VIII.	Early period.	Holes in the lobules of the ears.

It will be observed that in *two* of the eight cases of defect of the ears the period of pregnancy at which the impression was made is not stated; in *one* it was at three or four months; in *one*, four months; in *one* it was stated to have been at an early period, and in the other *three* it was three months or less. In two instances in which the ears were like those of the animal causing the impression, the period of pregnancy was three months in one and two or three months in the other. Of two cases in which there were holes in the ears, the period is not stated in one, and in the other it is said to have been early in pregnancy. In all the other cases, four in number, the ears were absent in whole or in part.

Embryology teaches that, as a rule, the outer ear appears as a low projection at the seventh week, and at the third month the external ear is usually well formed. It is evident, therefore, that if the impression in these cases was effective at all, it must have been in the stage when the ears were just forming or had just formed, when they were in such a soft and plastic state that any interference with their further development would lead to their atrophy and disappearance.

The possibility of the removal of the ears by intra-uterine amputation must be exceedingly remote; for their close approximation to the head of the fœtus would render their inclusion in bands or in a loop of the cord well-nigh impossible.

Of the two cases in which there were holes in the lobules of the ear at birth, the period of pregnancy at which the impression was made was not stated in *one*, and in the *other* it was merely stated to have been early. How these holes were produced it is impossible to understand.

DEFECTS OF THE EYES.

CASE.	PERIOD OF PREGNANCY.	NATURE OF DEFECT.
I.	Before and during pregnancy.	One blue and one dark eye.
II.	3 or 4 months.	Right eye absent.
III.	4 months.	Eyes large and round "like a sheep's."
IV.	2 months.	Eyes very large.

There were, it will be noticed, but few cases in which there was any defect of the eyes. Two of the four cases, however, are very striking when taken in connection with the other defects which were present in the same cases, and compared with the objects giving rise to the impression in each instance.

Case I. has already been described sufficiently in detail when speaking of defects of the lower extremities.

It is worthy of note with respect to Case II. that the child was not a cyclops, for the left eye was well developed, and the orbit of the right was indicated by a slight depression.

Case III. was probably one of intra-uterine hydrocephalus, for, in addition to the "very large eyes," the head was large, but the bones were absent for about two inches all around.

So far as defects of development are concerned, none of the four eye-cases are of especial interest; in two the eyes were large and round, but a peculiarity of this character taken by itself would have no significance.

In one (Case II.) there was a very evident and singular defect of development. The impression was made during the first three or four months, when the woman was attempting to rear by hand a calf which presented defects singularly like those which her child presented at its birth some months afterwards. There is nothing in the period of pregnancy at which the impression was made in this case which militates against the view that the defect was due to the impression.

DEFECTS OF THE HEAD, NECK, AND TRUNK.

CASE.	PERIOD OF PREGNANCY.	NATURE OF DEFECT.
I.	2 months.	No neck.
II.	5 or 6 months.	Facial paralysis.
III.	5 or 6 months.	Facial paralysis.
IV.	2 or 3 months.	Head like a jackass.
V.	Early period.	Large nævus between the eyes.
VI.	Early period.	Large nævus on the buttock.
VII.	4 months.	Long head (with black wool).
VIII.	3 months.	Face like a monkey's.
IX.	2 months.	Immense head; bones wanting for space of two inches above eyebrows and ears.
X.	First 3 months.	No neck; no face; long snout like a rat.
XI.	2 months.	Spina bifida.
XII.	1 month.	Hydrocephalus.
XIII.	17 weeks.	Four mammæ.
XIV.	Commencement.	Children united from neck to hips in front.
XV.	Commencement.	Children united from neck to hips in front.
XVI.	2 or 3 months.	Large and protruding tongue.
XVII.	3 months.	Anterior abdominal wall a thin film.
XVIII.	2 months.	Like the "frog-faced woman."
XIX.	4 weeks.	Hermaphrodite.
XX.	2 or 3 months.	Cystic tumor of the neck.

For the sake of convenience, the head, neck, and trunk have been grouped together, though they include organs which are developed at different stages of intra-uterine life. Of the twenty cases, there was facial paralysis in *two*, the period of pregnancy being the same in each. The cases are exceedingly striking ones: two women at about the same period of pregnancy were assisting their mother, who was an invalid, to the night-stool, when she was suddenly paralyzed on one side; the daughters were of course greatly shocked, and at full term each gave birth to a child with facial paralysis. In another case (VII.) the child was born with a long head and black wool; the period of pregnancy at which the impression was made in this instance was four months. In yet another case (XIII.) the child was born with four mammæ; the impression in this case was made at the seventeenth week. In all the other cases the impression was made at an early period of pregnancy; in fourteen of them certainly it was not later than three months; in the other two the reporters merely state that it was at an *early* period. It is unnecessary to study in detail the development of the fœtus in the class of cases now under consideration; it is sufficient to observe that in every instance, except the four mentioned above, the period of pregnancy at which the impression was made was *early*, when the embryo was in a plastic state and when the different organs and parts of the body were as yet undeveloped. It is especially to be remarked, furthermore, that in three at least of the other four cases, if not in all of them, the defects were of such a character as might very probably occur at a late stage of pregnancy. The case which seems most doubtful in this respect was the one of supernumerary mammæ: the impression here was definitely fixed at the seventeenth week; "the development of the human mamma begins in both sexes during the third month; at the fourth and fifth months a few simple tubular glands are arranged radially around the position of the future nipple, which is devoid of hair" (Landois). It may be well to call attention here to a fact in connection with the case that has just been mentioned (LXXIV. of the General Table) which is worthy of consideration. The mother in this instance was subjected to two impressions, one at the seventeenth week and one at the twentieth, and each is supposed to have caused a fœtal defect.

DEFECTS OF THE SKIN AND HAIR.

CASE.	PERIOD OF PREGNANCY.	NATURE OF DEFECT.
I.	4 months.	Patch of white hair on the head.
II.	4½ months.	Mark on the forehead.
III.	During whole of pregnancy.	Bald spot on the head.
IV.	Early period.	Scars on the thighs.
V.	Early period.	No prepuce.
VI.	2 months.	Red scar on the face.
VII.	4 months.	Black wool on head, back of the neck, and arms.
VIII.	2 months.	No prepuce.
IX.	3 months.	Back of neck and body covered with hair one or two inches long.
X.	20 weeks.	Scar on the head.
XI.	Last few days of pregnancy.	Blebs on the hands.
XII.	8 months.	Thumb-nail black and came off soon after birth.
XIII.	Few weeks.	Compact mass of hair extending back from eyebrows over head and neck.
XIV.	Not stated.	Bald patch on the head.
XV.	8½ months.	Large blebs on body and limbs.

Of the fifteen cases of defect of the skin and hair, *nine* were of the former and *six* of the latter. I have placed them together because the history of embryonic development would lead us to infer that these defects would occur from impressions made at a later date than those which we have considered heretofore. An examination of the table will show that this is the case. In *seven* of the fifteen cases it is distinctly stated that the impression was made after the fourth month of pregnancy, in *one* it continued during the whole of pregnancy, and in *another* the time is not stated. In the other six cases the impression was made at an early period. In *two* cases the impression was made during the last few days of pregnancy, and in each instance it was similar in character. In one (Case XI.) a woman during "the last few days" of her pregnancy received several burns upon her hand: her child was born with blebs, full and rounded and fresh-looking, at the corresponding parts of one of its hands. In the other (Case XV.)—one of the most remarkable cases on record—a woman was severely burnt upon her body and limbs. She was taken to the Pennsylvania Hospital, and thirty-six hours after her admission she was delivered of a child upon whose body and limbs were full and fresh-looking blebs corresponding almost exactly in situation to those on the mother.

With respect to scars and marks upon the skin, it is impossible to form any definite opinion as to their mode of production. Suffice it to say that they are almost certainly produced by some disturbance of the circulation, and that this disturbance may and most probably does occur usually comparatively late in the pregnant state, and may induce quite rapidly the changes that are observed at birth. This view is rendered highly probable, furthermore, by Case XII. in the table of defects of the skin and hair. A lady eight months pregnant was greatly shocked by seeing her little boy brought in with one of his thumbs severely crushed; there was an extravasation of blood under the nail, and it soon became black: her child was born soon afterwards, and the corresponding thumb-nail on its hand was black, and finally came off on the same day with that of its elder brother. In each of these cases the disturbance leading to the "mark" was clearly of an *inflammatory* character.

In those cases in which there was some defect in connection with the development of the hair it is to be observed that in *three* of the six cases the period of pregnancy was four months or over, in *one* it was three months, in *one* it was a few weeks, in *one* it is not stated. Now, "the hair appears upon the forehead at the nineteenth week; at the twenty-third to twenty-fifth week the lanugo hairs appear free" (Landois).

In order to determine the relationship which existed between the time of the impression and the stage of development, it was necessary to divide the cases into different classes; but this will be unnecessary in investigating the other points of interest, and we will return to a consideration of the General Table.

In what proportion of cases is the defect in the child similar to the object causing the impression upon the mother?

It has been urged as one of the objections to the doctrine of "Maternal Impressions" that the defect in the child does not usually correspond with the object producing the impression,—indeed, that it but seldom does so.

An examination of the General Table will show, however, quite a close correspondence in 69 of the 90 cases which I have collected. In some of these cases, indeed, the correspondence was exceedingly close,—for example, in Hunt's case of extensive burns (No. XC.), and also in Wilson's case (No. LXXVII.), in which there were blebs corresponding in situation to the burns on the mother's hand. Also in the remarkable case reported by Purefoy (No. XXXVII.) the correspondence was most striking. So close, indeed, is the correspondence in all of these 69 cases that any argument against maternal impressions based on a want of correspondence between the impression and the defect would seem to be worthless.

The strength of the argument in favor of maternal impressions drawn from a similarity between the object causing the impression and the nature of the

[illegible] stance that in some cases there were
[illegible] corresponding closely with a number
[illegible] the impression. For example, in
[illegible] the impression had no right ear,
[illegible] no right eye, nor had the child;
[illegible] the arm and forearm of the child
[illegible] was an abortive hand attached to
[illegible] ratological grounds that in these cases
[illegible] of *one side;* but such defects, to
[illegible] and the similarity is such that its
[illegible] vable to my mind. So in the case of
[illegible] not (in each of them) a *single* mark
[illegible] object causing the impression, but a
[illegible] arks alike in character, but on different
[illegible] however, corresponded in situation to an
[illegible] in a case reported by Mr. Ashburton
[illegible] two separate and distinct impressions,
[illegible] defects in the child.

[illegible] *be conscious of an impression and to*
[illegible] *cause?*

[illegible] there can be, I think, no doubt, that it
[illegible] a defect in the child for such a defect
[illegible] ntal or bodily. For example, in the case
[illegible] does not seem to have expected that her
[illegible] ar to those of the calf which she attempted
[illegible] as, of course, so often before her eyes and in

[illegible] of a defect, indeed, in Swift's case (No.
[illegible] r saw her little boy with his thumb in-
[illegible] with the corresponding thumb affected. In
[illegible] that the woman could have given any
[illegible] of her own injuries upon the bodily for-
[illegible] rings were, of course, intense, and death was
[illegible] might be multiplied in which there seems
[illegible] between the impression and the defect, but
[illegible] entirely unconscious of any impression at
[illegible] ught that it would influence the child which

[illegible] *made by the mother, before the child is born,*
[illegible] *of the defect which she anticipates?*

In not a few [illegible] mother has stated before the birth of the child what the [illegible] and what she believed would be the nature of the defect in the [illegible] ample, in Daly's case (No. XLIV.) a woman during the first [illegible] her pregnancy lived in a house which was infested with [illegible] was greatly annoyed by them, and at the birth of the child, before she knew of any defect, she asked if it was like a rat: the child had no neck, and no face, but a long snout projecting from between the shoulders and in a line with the body. In two cases also (XX. and XXIII.), where the impression was due to a dream, the nature of the impression was clearly and distinctly stated months before the birth of the child, and in each instance the defect corresponded thereto in a most remarkable manner. Evidence of this sort, however, should be very carefully weighed before acceptance, unless the defect corrresponds very closely with the impression, for it is a notorious fact that many women expect defects in their children, and often have very definite conceptions as to what those defects will be, and yet at birth the children are normally developed in all respects and are free from any "marks" whatsoever.

Through what channels are impressions made upon the mother?

The channel through which impressions are usually received by the mother is that of sight; but it is difficult to say how much is due to the simple sight of the object, and how much to the emotional disturbance caused by viewing it. It is probable that the latter is really the effective cause, for in some instances the impression has been caused in other ways and yet the result has been the same. For example, in three cases (IV., XX., and XXXIII.) the impression was caused by a dream; in another (XLVI.) a woman was greatly excited because her husband—a physician—was invited to assist at the circumcision of a neighbor's child. In still another case (LXXXI.) a woman had her hand violently pressed by her husband's elbow, the pain being so great that she finally fainted. In this case, and in at least two others,—those of Hunt and Wilson (XC. and LXXVII.),—the impression was evidently caused by violent pain.

What duration of the impression is necessary in order to produce a result?

There seems to be no definite rule on this point, nor is it by any means easy to arrive at a conclusion with regard to it.

In Case XC. the period of time which elapsed between the "impression" and the "effect" could not have been *more* than twenty hours, for the woman was delivered of the child about thirty-six hours after her injuries were received, and the fœtal heart-sounds had become inaudible some hours before.

In a number of cases the shock was sudden, but the mental impression resulting therefrom was far more enduring, and it is impossible to say whether the defect would have resulted if there had been nothing to induce it but the sudden and fleeting shock. There seems to be a general impression among writers on the subject that the impression is more likely to produce an effect if it is of considerable duration; but Case XC. shows that this is not invariably true. In a case reported by Bolton (XII.), in which an opossum was thrown into the lap of a young woman three months pregnant, it is distinctly stated that, while she was startled at the time, the circumstance was soon forgotten.

It is probable that either a sudden and violent impression or one which is slighter in degree but operative for a longer time may produce a similar effect.

What character of impressions is most liable to produce defects?

In the vast majority of cases the impression is due to some emotional disturbance, and in nearly all the cases included in the table the emotion was of an unpleasant character. *Fright* and the mental impressions resulting therefrom would seem to be by far the most common of all causes. *Physical suffering* must have been the cause in two cases (XC. and LXXVII.). It was *pity*, doubtless, that led the woman to attempt to rear a deformed calf by hand, and it is probable that this was the emotion that led to the defects in the child in this case (XXXVII.). In a few instances the emotion was of an agreeable character: for example, a lady was in the habit during her pregnancy of looking at the bald head of her father-in-law with "unaccountable delight:" her child was born with a bald patch on its head (Case XXIX.). In the case (XIII.) of the lady whose child was born with one leg shorter than the other and with a difference in the color of the two eyes, the emotion causing the impression was one of great affection.

It is singular, in view of the frequency with which defects are attributed by the general public to "maternal longings" for certain articles of diet, that so few cases of this character should have been reported by physicians; and I have been able to find none which it seemed proper to include in the table. There would seem to be no good reason, however, why a strong impression should not be produced in this way. A friend of mine has told me that during one of his wife's pregnancies her craving for oysters was such that she was "moved to tears" when she found they could not be had; and when the child was born there was a mark upon its foot which to the eyes of the father and mother was precisely like an oyster. I have never seen the child myself, and mention the case here only to show how strong such "cravings" may be, even in the case of sensible women, to which class this lady belongs.

Before bringing this chapter to a close, it may be well to consider very briefly the objections which have been urged to the doctrine of "Maternal Impressions." They are as follows:

1. Abnormalities may occur without fright.

2. Deformities generally occur before pregnancy is certain or before the mother is conscious that she is pregnant.

There can be no question of the truth of both of these propositions. We have already seen that in a considerable number of cases, where the similarity between the object causing the impression and the defect in the child was most striking, there had been no *conscious* impression made upon the mother. But, aside from this, all that the most ardent advocates of maternal impressions claim is that they are *one* of the causes of defects or deformities, but by no means the only cause.

3. Abnormalities may occur in animals.

This is not a valid objection, for animals possess emotions as well as mankind: what boy has not seen a cur dog with a tin pan tied to his tail exhibit the most abject terror? Nor are instances wanting in which abnormalities in animals appeared to be due to maternal impressions. Furman has reported[1] one in point occurring in Henderson, Kentucky: there passed through the town a menagerie with which was an elephant; a sow pregnant a short time saw this elephant, and one of her pigs born some time afterwards had skin, ears, and *trunk* similar to those of an elephant. He states that a similar case had occurred in Shawneetown, Illinois. Now, unless we deny the facts, the conviction that the relationship in these cases is that of cause and effect seems almost irresistible.

4. Several children of the same parents often present bodily abnormalities.

In a number of cases of deformity heredity has been the cause, and the deformity may differ somewhat from that of the parent. For instance, Mr. Lucas reported to the Clinical Society of London in 1887 three cases in which the absence of one upper lateral incisor tooth in the parent was followed by hare-lip in the child, and at a meeting of the same Society on April 1, 1887, a case was shown by Mr. Parker and Dr. Robinson in which the two inner toes were united and the three outer similarly united; the child's grandmother had a similar defect, and sixteen of this old woman's descendants were deformed in precisely the same way.

The late Dr. W. T. Taylor, of Philadelphia, reported[2] a very curious case which had been related to him by Dr. Garretson, in which a lady gave birth to five children in succession, each of whom had cleft palate. In the first instance the defect was attributed to a maternal impression; and, as the defect was slighter in each child than in the preceding one, Garretson supposes that the impression was gradually effaced. This view of an "overlapping" impression—if I may use such a word—is at least a plausible one.

The objection that deformities are due to defects of development has already been considered.

Why in the case of twins one should be deformed and the other not, in cases of supposed maternal impressions, is unknown; and it is useless to speculate on the subject just now.

The fact that fright and emotional disturbances of other kinds are common in pregnant women, and deformities comparatively rare, is not a just ground for unbelief in the power of maternal impressions. It would be as unreasonable to say that scarlet fever is never conveyed by milk, because but few cases of the kind have been reported, as to say that maternal impressions never cause deformities, because such a connection can rarely be established. The *fact* that scarlet fever is sometimes conveyed in milk was well established long before the nature of the disease was definitely understood, and it was not rejected because no explanation could be given of the manner in which it was brought about; nor should the fact that maternal impressions sometimes produce deformities be rejected because we cannot understand how they act.

---

[1] St. Louis M. and S. Jour., May 5, 1880.

[2] Phila. Med. Times, Nov. 25, 1876.

part of this whole subject yet to be should be guarded from strong ring her pregnancy, for fear of the light before us, there can, I think, Few as are the instances, relatively able to maternal impressions, they ntly distressing when they occur, pregnant woman; and I cannot but sician to warn his pregnant patients motions of every kind, and especially r.

llowing conclusions with respect to be warranted:

gnant woman are capable of causing d.

ts are *often* (comparatively speaking)

al impressions may be either errors are apparently due to circulatory or in-

development have, as a rule, been at- riod of pregnancy when such errors of

have, as a rule, been attributed to of pregnancy, when circulatory and most reasonably expected.

the cases there is a striking similarity ssion and the defect in the child.

man to be *conscious* of the impression, to occur.

rtion of cases the woman has stated the anticipated defect before the birth of

y due to emotional disturbances which character, but physical pain is capable induce defects.

violence may produce an impression —but, as a general rule, the duration is

apable of producing defects in the lower

l impressions are sufficiently numerous to necessitate the avoidance by any tional disturbances, especially those of

DISEASES OF THE FŒTUS.

By BARTON COOKE HIRST, M.D.

In the brief space assigned this article it will be possible to give only a sketch of the most important pathological conditions affecting fœtal life.

On account of the nature of the work, most attention will be paid those intra-uterine diseases which more especially affect the subsequent extra-uterine existence.

The various conditions that unfavorably influence the fœtus in utero will be considered in the following order:

I. Diseases referable to maternal influences.
II. Diseases referable to abnormal conditions of the father.
III. Syphilis.
IV. Infectious diseases.
V. Non-infectious diseases.
VI. Traumatism.
VII. Diseases of the fœtal appendages which react injuriously or fatally upon the fœtus itself.

Fœtal Diseases referable to Maternal Influences.—The catalogue of these affections is a long one. Nervous disturbances, high temperature, defective nutrition, diseases of the womb and of its adnexa and lining membrane, alteration in the blood-pressure, the presence within the blood of soluble poisons, or that subtle influence which we call heredity, may all be accountable for fœtal disease or fœtal death.

The Influence upon the Fœtus of Nervous Disturbance in the Mother.—No one has demonstrated a direct nervous connection between mother and fœtus, yet no one will deny the remarkable sympathy between the two. Mental peculiarities, acquired perhaps only during pregnancy, are not rarely stamped indelibly upon the fœtus. The mother of Jesse Pomeroy, the well-known moral monstrosity of New England, took delight, while carrying this child in utero, in watching her husband, a butcher, ply his trade. The boy's irresistible inclination to torture and slay may well have had its origin in his mother's perverted taste during pregnancy. But more wonderful still is the occurrence of physical defects or peculiarities in the fœtus, photographic reproductions of objects that have produced a strong impression upon the mother during pregnancy. I had occasion once to administer many hypodermic injections to a woman in the early months of gestation, producing in several instances small abscesses which left conspicuous scars. The child was born with spots upon it identical in appearance and situation with those upon its mother's arm. Still more extraordinary examples of maternal impressions have been seen by others.[1] The fatal effect in some instances upon the fœtus of strong emotions in the mother has seemed to me explicable in the light of recent discoveries as to the formation of leukomaines and ptomaines: perhaps the powerful nervous disturbance acts upon the blood like an electric current upon a chemical solution, altering its composition. It would be difficult to explain by this theory, however, cases of congenital idiocy which may be traced to emotions of fear, anger, or disgust during pregnancy. I have been recently told of a remarkable case of this kind. A lady was obliged to pass the bridal night with an intoxicated bridegroom; conception occurred, and the child became an idiot; three subsequent children were also mentally defective, although there was no taint of insanity on either side of the house. The impression of deep disgust experienced at the first conception exerted an influence on the development of the subsequent children. A great fright during pregnancy, if it does not kill the child outright, may much diminish its mental capacity. Down[2] says that he can refer to a number of cases of feeble-mindedness which were the outcome of the siege of Lucknow, and the same author refers to an incident of the siege of Landau (1793): "In addition to a violent cannonading, the arsenal blew up with a terrific explosion which few could hear with unshaken nerves;" of ninety-two children born in that district within a few months afterwards, eight became idiots. We must frankly admit that an explanation of the susceptibility displayed by the fœtus to violent impressions upon the maternal nervous system is beyond our power; we are obliged, notwithstanding, to allow that the fact is as well established as any in medicine.

The Influence of Elevated Temperature upon the Fœtus.—It used to be thought that fever of itself in a pregnant woman was highly dangerous to the fœtus. This idea was generally adopted after the well-known experiments of Runge,[3] who found that if the body-temperature of a pregnant rabbit was raised to 105.8° F., the young within it died. Doléris[4] in 1883 pointed out a fault in these experiments; the temperature was too rapidly raised: with a gradual elevation of body-heat to 105°–106° F. the young of pregnant animals were not at all injuriously affected. Indeed, a fœtus has been known to endure extraordinary heat without destruction. Preyer[1] once found a temperature of 111.2° F. in the anus of a young guinea-pig in utero. I have been told by a chicken-breeder that on one occasion the temperature of his incubator was found to be 115° F., and yet only half his chicks died in consequence. Usually, however, as Runge found in his second series of experiments, a maternal temperature of 109.5° F. must be fatal to the fœtus, even though this heat be gradually produced. The practical deductions to be drawn from these experiments—and they are in accordance with clinical experience—are that a sudden rise of temperature to 106° F. will probably kill the fœtus; that a gradual elevation to this point, on the contrary, need not be feared; and that a temperature as high as 109° F. will, even though gradually produced, destroy fœtal life.

---

[1] For the best modern paper on this subject, see Fordyce Barker, Trans. Amer. Gyn. Soc., vol. xi., 1886.

[2] Mental Affections of Childhood and Youth, London, 1887.

[3] Arch. f. Gynäk., 1877, Bd. xii. u. xiii. Ss. 16, 123.

[4] Comptes-rend. hébd. des Séances de la Société de Biologie, Nov. 28, 29. Doléris' results have been confirmed by Doré, by Negri, and by Runge himself in a second set of experiments.

Defective Nutrition.—Defective nutrition in the mother, with its consequent anæmia, either is fatal to the fœtus in utero or else is accountable for the birth of puny, wretched children, who die early or drag through a sickly childhood. The causes of the maternal malnutrition are many: among the more serious are chronic diseases, as cancer, phthisis, malaria, nephritis;[2] chronic poisoning, as by lead or perhaps tobacco; inability to retain food, as in the vomiting of pregnancy; inability to obtain food, as during siege and famine: the "enfants du siège" of Paris were for some time distinguishable from the children born before and after them. The treatment of fœtal ill health from maternal anæmia is of course to improve the mother's impoverished blood: remove the cause of the trouble, if possible, administer iron, and prescribe moderate exercise in the open air, with perhaps change of climate, and the birth of a vigorous infant can sometimes be secured which will perhaps contrast strongly with its predecessors which were not treated in utero.

Diseases of the Endometrium, the Womb and its Adnexa.—These need only be mentioned here, for their most frequent effect is the premature expulsion of the ovum. I have known, however, a great inflammatory thickening of the endometrium to exist throughout pregnancy, with the result apparently of diverting nutriment to itself which should have gone to the child, which was born a feeble creature and lived only a short time.

Alteration in the Maternal Blood-Pressure.—Runge[3] found that sudden alterations of the blood-pressure in pregnant animals were fatal to their young. What practical bearing this discovery may have upon disease and death of the human fœtus has not been determined.

Poisons in the Maternal Blood.—Any soluble substance absorbed into the maternal circulation may pass from mother to fœtus.[4] To the presence within the mother's blood of poisonous material may be attributed certain cases of feebleness and ill-development at birth. Paul[1] observed one hundred and twenty-three cases of saturnism in pregnancy: sixty-four of these ended in abortion, four in premature labor; five children were still-born, and of the whole number only ten survived the age of three years. In Europe it is claimed that tobacco-workers give birth to feeble children; here this is denied.[2] For some poisons a fœtus acquires remarkable tolerance. Recently I administered large doses of morphine for a long period to a pregnant woman without apparently affecting the fœtus. To the influences of some other substances the fœtus in utero is very sensitive. For instance, the bile salts seem to have a most pernicious action upon fœtal health and life.[3]

---

[1] Physiologie des Embryo, Leipsic, 1884.

[2] E. Cohn stated at a meeting of the Berlin Obstetrical Society that eighty-six per cent. of the children from mothers with nephritis would be born still or too feeble to survive long.

[3] Arch. f. Gynäk., Bd. xiii. S. 488.

[4] Chloroform, ether, salicylate of sodium, benzoate of sodium, atropine, strychnine, morphine, quinine, corrosive sublimate, iodide of potassium, urea, the bile salts, soluble salts of

Heredity.—The fœtus in utero may acquire from its mother certain tendencies to disease which may be manifested only in after-life: the most remarkable example of this is found in the transmission of hæmophilia through a female to her male offspring. Another extraordinary instance of a tendency to disease acquired in utero, but manifested only in adult life, has recently been reported. A young woman with a violent attack of chorea in pregnancy told her physician that her mother had been affected with the same disease while pregnant with herself. Nothing is more familiar in nature than the transmission of mental, physical, and moral peculiarities from parent to child; and this fact must be taken into account by all clinicians. The question as a whole, however, is too large for consideration here, and it must be passed by with the brief mention it has received.

Diseases of the Fœtus referable to Abnormal Conditions of the Father.—It sometimes happens that the spermatic particle, while capable of fertilizing the ovum, is unfit to perform its share in the work of building up a healthy, well-developed fœtus. If the father is too young or too old, the subject of some debilitating disease, a victim of chronic poisoning, or a drunkard, his fertilizing element may produce an embryo that will die before maturity or else be born at term a defective, unsound infant. As saturnism in the mother is disastrous to the fœtus, so also a man saturated with lead seems almost incapable of procreating healthy children. Of thirty-nine pregnancies in women whose husbands were sufferers from chronic lead-poisoning, eleven ended in abortion, there was one still-birth, and only nine of the children survived early infancy.[4] Men afflicted with nephritis,[5] diabetes,[5] phthisis,[6] or cancer[7] have been found, in some instances, unable to produce a fœtus capable of normal growth, while their widows,

---

lead, and tobacco are some of the substances that have been known to pass from the maternal into the fœtal blood.

[1] Thèse de Paris, 1861.

[2] Hirst, Amer. Syst. of Obstetrics.

[3] See Valenta, Oesterreichische Jahrb., 1869, Bd. xviii. S. 163.

[4] Paul, loc. cit.

[5] Priestley, Lumleian Lectures on Intra-uterine Death, London, 1887.

[6] D'Outrepont, Neue Zeitschr. f. Geburts., 1838, Bd. vi. S. 34.

[7] Jacquemier, Dict. encycl. des Sci. Méd., art. "Avortement."

subsequently married, have borne healthy children. Drunkenness in the father is not infrequently a cause of ill-development in the fœtus. Down[1] has observed twelve cases of sporadic cretinism in England, the majority of which could be traced to drunkenness in the father at the time of procreation. Matthews Duncan[2] has also called attention recently to the evil influence upon the fœtus of intoxication in the parents.

Syphilis.—This disease of fœtal life is put in a separate section chiefly on account of its great importance and relative frequency.[3] It is separated from the other infectious diseases because its manner of invading the embryo and fœtus is peculiar. If a woman is syphilitic, every ovum within the ovary is diseased, and if fertilized will contaminate the resulting embryo. On the other hand, each fertilizing element from a man with this disease carries in itself the seed of the disorder to infect the ovum which receives it, although the maternal organism, as a whole, may remain unaffected. Again, if the syphilitic poison is introduced into the body of a pregnant woman previously healthy, the disease may be transmitted to the fœtus in utero. This doctrine of the modes in which an embryo may become tainted with syphilis has not yet met with general acceptance, although it can be supported by strongest proofs. No one, of course, now denies the fact that a woman infected before or at the time of insemination will probably produce syphilitic offspring. That the disease can be transmitted to the fœtus in utero, or that the ovum alone can be infected while the mother remains, for a time at least, free from the disease, are statements not so universally admitted. A prominent authority in this country says, in a recent edition of his work on obstetrics, "The syphilitic poison will not traverse the septa intervening between the fœtal and the maternal vascular systems." Neumann,[4] however, has seen this very thing occur in five out of twenty women who were infected with syphilis during pregnancy. In the Maternité at Bordeaux,[5] of twelve women infected with syphilis in the first four months of pregnancy, all gave birth to dead children; in those cases in which infection occurred from the fourth to the sixth month about half the children were still-born, and in seven cases of infection during the last three months there were four still-births. I have attended, in the Philadelphia Hospital, a woman who acquired a chancre in the third month of pregnancy: her child, still-born, had on it unmistakable evidences of syphilis. This cannot excite much surprise; for it becomes every day more clear that the syphilitic poison is "a particulate and living virus,"[6] and we shall presently offer ample evidence to prove that disease-breeding germs can pass from mother to fœtus. Collerier, Notta, Follin, Charnier, Mireur, Langlebert, Corry,

---

[1] Op. cit.

[2] Edin. Med. Jour., April, 1888.

[3] Ruge estimates that eighty-three per cent. of premature births and still-births may be traced to syphilis in one or both of the parents. (Zeitschr. f. Geburtsh., Bd. i.)

[4] Wien. Med. Presse, xxix., xxx., 1885.

[5] Hirigoyen, abstract in New York Med. Record, April 12, 1887.

[6] J. Hutchinson, Brit. Med. Jour., 1886, i. 279.

[illegible] said that they do not believe the
[illegible] the mother is syphilitic; but of
[illegible] Charpentier, Priestley, and many
[illegible] transmission of syphilis to the ovum
[illegible] the previous infection of the woman.
[illegible] syphilitic poison develops with its
[illegible] infected in her turn directly from
[illegible] septum.[3]

[illegible].—The infection of the fœtus may be in-
[illegible] either parent had acquired syphilis at a
[illegible] ation. There is no doubt that the like-
[illegible] diseased children somewhat diminishes
[illegible] safety has not been discovered. Lomer[4]
[illegible] infant ten years after the infection of
[illegible] a latent syphilis of twelve years' dura-
[illegible] been pursued, however, four years should
[illegible] If a woman should acquire a chancre during
[illegible] disease attacking the fœtus must not be
[illegible] sign of syphilis in the fœtus is occasionally
[illegible] the ovum is infected by the spermatic par-
[illegible] perfectly healthy till the middle of preg-
[illegible] syphilis may appear, without the slightest
[illegible] sore. In such cases the poison of the disease
[illegible] fœtus to mother.

[illegible] fœtal syphilis can be looked for only in the
[illegible] from the uterus, and much may depend upon
[illegible] is, however, not always easy to reach. The
[illegible] ignorance or design, may be entirely negative; the
[illegible] no distinctive sign upon its body; if it is living,
[illegible] characteristic eruptions during the first few weeks
[illegible] enough to the hereditary taint; if the child is dead,
[illegible] more easily made, unless maceration has proceeded
[illegible], however, there is one sign that may be regarded as

[illegible] of fœtal death it is important to ascertain the cause of
[illegible] order to prevent its recurrence in subsequent pregnancies.
[illegible] is a trained pathologist, the detection of syphilis should
[illegible]. The [illegible] eruptions on the skin, the condylomata
[illegible] of the [illegible] membranes, the inflammations of the

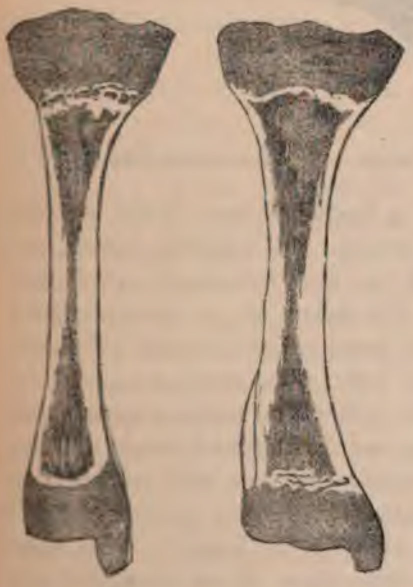
---

[illegible] t. ii. p. 36.
[illegible] xxiii., 1886.
[illegible] loc. cit.; J. Hutchinson, Brit. Med. Jour.,
[illegible] 1885; G. S. West, Amer. Jour. Obstet., 1885, p. 182.
[illegible]
[illegible] Jahr [illegible]

[6] Fournier, Syphilis et Mariage.

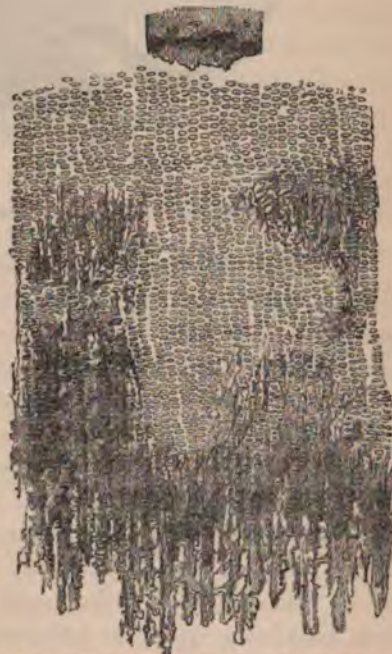
serous membranes, the gummatous and miliary deposits, and the morbid growth of connective tissue in the brain, lungs, pancreas, kidneys, liver, and spleen, and in the coats of the intestines and walls of the blood-vessels, along with a characteristic osteochondritis, should demonstrate the character of the disease. It often falls to the lot of the general practitioner, however, to observe cases of repeated fœtal death the cause of which is obscure, although suspicion naturally rests upon syphilis. Thanks to the investigations of Wegner,[1] Ruge,[2] Lomer,[3] and others, it is now well established that syphilis can be recognized in the fœtus by a few signs easily found, quite characteristic, and requiring for their detection no special training in the methods of pathological research. Wegner was the first to call attention to a curious condition of the dividing-line between diaphysis and epiphysis of the long bones of a syphilitic infant. Instead of a sharp, regular, delicate line formed by the immediate apposition of cartilaginous to bony tissue, as in a healthy fœtus, there may be seen in syphilitic cases a jagged, rather broad line of a yellow color separating bone from cartilage. A microscopic study of this portion of the bone shows that there has been a premature attempt at ossification which has ended in fatty degeneration. For more than a year I carefully looked for this sign in every case of unmistakable fœtal syphilis that occurred in the Philadelphia and Maternity Hospitals,

FIG. 1.



Tibia showing syphilitic osteochondritis. (Wegner.)

FIG. 2.



Microscopic appearance of syphilitic osteochondritis. (Wegner.)

---

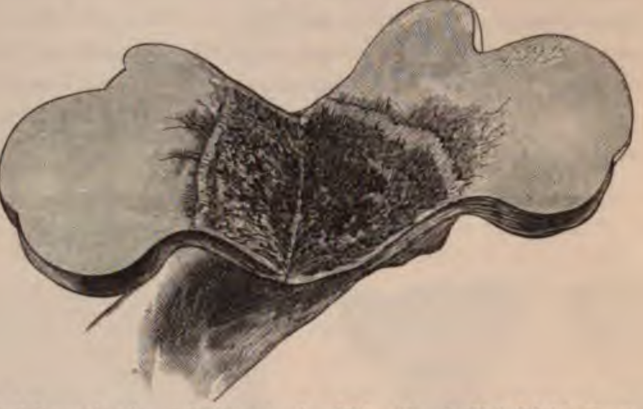
[1] Virchow's Archiv, Bd. l. S. 305.

[2] Zeitschr. f. Geburtsh., Bd. i.

[3] Ibid., Bd. x.

and never failed to find it, while in doubtful cases it proved a valuable aid to a correct diagnosis. In the Frauenklinik at Berlin[1] this sign was also carefully investigated, with a result wholly favorable to its distinctive character.[2]

FIG. 3.



Head of femur showing syphilitic osteochondritis. (Case in Philadelphia Hospital.)

According to Ruge,[3] the liver of a healthy infant should constitute about one-thirtieth part of the body-weight. In syphilitic infants, however, this proportion is much exceeded, the liver forming in extreme cases one-eighth of the total body-weight. The spleen, too, is much enlarged in syphilis: this organ, which in a normal fœtus at term should be in weight one-three-hundredth part of the whole body, often much exceeds its due proportion. Upon these three signs, the yellow line between epiphysis and diaphysis, the increased weight of liver, and the increased weight of spleen, all easily discovered, the diagnosis of syphilis may rest with reasonable certainty. If one would push the investigation further, perhaps the next surest indication of syphilis might be found in the lungs.[4] These organs will manifest a syphilitic infection in three ways: by an interstitial overgrowth; by the presence of gummata; by a peculiar catarrhal inflammation, resulting in what is called white pneumonia. The first of these is the most common: the connective-tissue overgrowth about the blood-vessels and the alveoli gives the lungs greater weight and more solidity than they should possess; their color is often dark red; if the infant has breathed, as it commonly does, although imperfectly, for a short time after birth, the lungs will not float buoyantly, although they do not usually sink outright.

---

[1] Lomer, loc. cit.

[2] Zweifel thus describes the progress of the disease: "There is formed, in a certain region of the cartilage, granulation-tissue, insufficiently supplied with blood-vessels and ill nourished. There results necrosis of this tissue, with an attempt at exfoliation and an accompanying suppuration."

[3] Loc. cit.

[4] For an exceedingly interesting paper on this subject, see Heller, Die Lungenerkrankungen bei angeborener Syphilis, Deutsch. Arch. f. Klin. Med., Bd. xlii. S. 159.

Microscopically it may be seen that the alveoli are much encroached upon by the interstitial thickening, and that lung-expansion and adequate respiration are impossible. The catarrhal pneumonia in utero due to syphilis is rare. The lungs in this form of disease are large and heavy; they quite fill out the thoracic cavity and bear upon their external surface the imprint of the ribs; in color they are white, the whole organ having undergone a more or less complete fatty degeneration. This condition is quite incompatible with extra-uterine life: the infant never breathes.

Prognosis.—The chances for a syphilitic embryo reaching a healthy maturity are very slim. Charpentier found in an analysis of six hundred and fifty-seven cases that more than a third of the pregnancies in syphilitic women ended in abortion, while a large proportion of the children born at term were dead. Add to this the low vitality of syphilitic infants and the high mortality among them, and it will be found that, fortunately for the race, hereditary syphilis is not so common as one might expect, if it is looked for in children of more than a year's growth.

Treatment.—The treatment of fœtal syphilis is best begun before the embryo is called into existence, by eradicating the disease from the parents. If only one is affected, the treatment of the other is of course superfluous. In case of doubt, however, both man and woman should be put on a long course of antisyphilitic remedies. The direct treatment of the embryo or fœtus after conception, while not so satisfactory, should not be neglected, if there is good reason to believe it syphilitic.

Both mercury, in its soluble salts, and iodide of potassium will pass into the fœtal circulation and may modify or entirely prevent the morbid processes characteristic of the disease. Along with these remedies it might not be amiss to give chlorate of potassium. In most cases the placenta will be diseased and the effective area for oxygenating the fœtal blood much diminished; and it is in such cases that this drug does good, and has been recommended by Simpson, Barker, Penrose, and others, although it may be doubted if the explanation formerly offered would account for its favorable action,—that it increased the oxygenating power of the maternal blood.

Infectious Diseases.—These affections are produced by the entrance into the body and the development there of some low form of life: this has been conclusively proved of many infectious diseases; of the rest it may be surely inferred, although the exact nature of the *materies morbi* has in some instances not yet been demonstrated. The only medium of communication with the outer world possible to the fœtus is the maternal blood. If, therefore, the fœtus is attacked by an infectious disease, the micro-organism that produces it must have passed from the maternal into the fœtal portion of the placenta, and so have traversed the septa intervening between the fœtal and the maternal blood. Many observers, however, deny the possibility of this transmigration. Brauell[1] and Davaine[2] experiment-ing with the bacilli of anthrax saw large colonies of them heaped up on the maternal side of the placenta, while the fœtal structures were entirely free from disease. Straus and Chamberland[1] failed to infect animals by injecting the blood of a fœtus taken from an animal that had died of anthrax. Runge of Dorpat inoculated a number of pregnant rabbits with tuberculosis, but failed absolutely to detect the characteristic bacillus in a single fœtus. Chambrelent[2] quotes Budin, Tarnier, Charpentier, Hoffmann, Jossinsky, and Fehling as denying the possibility of the passage of microbes from mother to fœtus. V. Ott,[3] in a recent article, after giving a résumé of the literature on the subject, expresses his disbelief in the passage of solid particles into the placenta, and supports his statement by describing experiments of his own which altered the constitution of the mother's blood without in any way affecting that of the fœtus. Wolff[4] infected a number of rabbits and guinea-pigs with anthrax, but failed to find a trace of the disease in their young. Curt Jani,[5] an assistant of Prof. Weigert, having an opportunity to examine the body of a woman who had died in the fifth month of pregnancy from general miliary tuberculosis, found not a trace of the bacilli in placenta or fœtus, although every maternal organ swarmed with them. Urvitch[6] inoculated seven pregnant mice with the microbes of mouse-septicæmia: the experiment was negative as regards the young. Bompiani[7] and Morisani[8] have observed pregnant women with anthrax while their infants remained unaffected. Krukenberg[9] experimented with the bacillus prodigiosus: he could not in a single instance discover that it passed into the fœtal portion of the placenta. On the other hand, careful experiments and clinical observations bear witness to the fact that microbes can pass from mother to fœtus. In 1882 Arloing, Cornevin, and Thomas[10] showed that the anthrax-bacilli could pass from mother to fœtus; in the same year Straus and Chamberland recalled their first-expressed opinion and announced their belief in the transmission of disease-germs to the fœtus in utero.[11] Chambrelent[12] was able to cultivate the microbes of chicken-cholera from the fœtal blood and to inoculate other animals with the cultures. Mars[13] of Cracow after injecting putrid solutions into the mother animal found large numbers of micro-organisms in the fœtus; and Dr. Pyle,[14] working in the Pathological Laboratory of the University of Pennsylvania, obtained the same results; he also claimed to have found micro-organisms in a fœtus that had been removed by Cæsarean section from a woman dying of septicæmia. Koubassoff, working under the supervision of Pasteur in his laboratory in Paris, asserted that he never failed to find the anthrax-bacillus in the fœtus when the mother had been thoroughly infected with the disease, except in one instance, where of two fœtuses one was partially macerated and its placenta the seat of hemorrhagic extravasations, while the other was well developed. In the former of these two no bacilli were found, but in the latter they were present in large numbers. Upon this experience Koubassoff bases the conclusion that the placenta can offer effective resistance to the passage of microbes only when its condition is pathological. Sangalli[1] and Ahlfeld[2] have each observed a case of anthrax in pregnancy in which the fœtus acquired the disease. In Ahlfeld's case, however, the infection probably occurred during labor. A case is recently reported in which the fœtus was infected by the diplococcus pneumoniæ (Fränkel) during an attack of pleuro-pneumonia in the mother;[3] in connection with this report is detailed an experiment in which the young of a pregnant rabbit were infected with the meningococcus taken from a case of cerebro-spinal meningitis and injected into the mother animal. Jaquet's[4] experiments showed that the micrococcus tetragonus could pass from mother to fœtus. Rosenbach[5] has reported a case of congenital osteomyelitis, cysticerci have been found in the new-born of pigs, and filariæ have been discovered in fœtal puppies.[6] Collections of pus have also been found in the new-born infant.[7] Malvoz[8] infected with anthrax four pregnant rabbits, containing thirty-two fœtuses: two of the latter were infected in utero. Experiments with the bacillus prodigiosus and micrococcus tetragonus were negative. Malvoz believes that only those microbes which produce some lesion in the placenta can pass into the fœtal circulation.[9] In Birch-Hirschfeld's[10] experiments on pregnant animals with anthrax-bacillus the results were in the majority of cases favorable to the view that micro-organisms can pass from maternal into fœtal blood.[11] An interesting study has lately been made of the passage to and from the blood in the kidneys of micro-organisms, which has a direct bearing upon the passage of microbes from mother to fœtus.[1] It was found that bacilli did pass not only from the blood into the uriniferous canals, but also back again from urine to blood. It seems that we must concede to these minute creatures the ability to penetrate the placenta. Indeed, in no other way could we explain the occurrence of infectious diseases in the fœtus, and the list of these is already a long one. Perhaps the microbes are carried over to the fœtus from the maternal circulation enclosed in wandering white blood-corpuscles.

---

[1] Virch. Archiv, 1838, p. 459.

[2] Bulletin de l'Académie de Médecine, 1867.

---

[1] Comptes-rend. de la Société de Biologie, 1882, p. 689.

[2] Recherches sur le Passage des Eléments figurés à travers le Placenta, Paris, 1883.

[3] Arch. f. Gynäk., Bd. xxvii.

[4] Virch. Arch., Bd. cv. S. 192.

[5] Ibid., Bd. ciii. S. 522.

[6] Inaug. Dissert., St. Petersburg, 1885, p. 77.

[7] Annal. di Ostet., May–June, 1887.

[8] Abstr. in Centralbl. f. Chirurg., vii., 1887.

[9] Arch. f. Gynäk., Bd. xxxi. H. iii. S. 313.

[10] Compt.-rend. des Séances de l'Acad. des Sciences, 1882, t. xcii. p. 739.

[11] See Koubassoff, ibid., t. c. p. 373.

[12] Op. cit.

[13] Arch. de Tocol., 1883, p. 381.

[14] Med. News, Aug. 30, 1884.

---

[1] Gaz. Med. Ital.-Lombard., 1883, Nos. 4, 5.

[2] Bericht. u. Arbeit. aus d. Geburts.-Gynäk. Klinik zu Marburg, 1885–86.

[3] Foà and Bordoni-Uffreduzzi, La Riforma Med., 1887, No. 39.

[4] At a meeting of the Berlin Obstetrical Society, Feb. 10, 1888.

[5] Quoted by Watson Cheyne, Brit. Med. Jour., 1888, vol. i. p. 452.

[6] Bouchut, Pathologie générale.

[7] In the Lancet, 1887, vol. ii. p. 859, is an account of an abscess of the vestibule opened fourteen hours after birth. My friend Dr. De Schweinitz tells me that in superintending a post-mortem examination of an infant delivered in my service in the Philadelphia Hospital, he saw in the anterior mediastinum a fluid closely resembling pus. The mother was in a dying condition from septicæmia when the child was born.

[8] "Sur le Mécanisme du Passage des Bactéries de la Mère au Fétus." Successful thesis, Brussels, 1887.

[9] Ann. de l'Institut Pasteur, 1888, No. 3.

[10] München. Med. Wochenschr., No. 42, 1888.

[11] See also Wolff, "Ueber Vererbung von Infectionskrankheiten," Virch. Arch., Bd. cxii, S. 136.

Variola.—The occurrence of variola in utero has long been a fact beyond dispute. The fœtus, however, is not always affected, even though the mother has the disease badly; on the other hand, the mother may transmit the disease to the child in her womb although she remains healthy; or a light attack of varioloid in the mother may be associated with virulent small-pox in the fœtus. Again, it has been noted that, of twins, one or both may be affected. Many observers have tried the effect of vaccinating a pregnant woman: in the majority of cases a subsequent vaccination of the child "took;" occasionally, however, it was a failure, apparently because the virus had affected both mother and fœtus.[2]

Rubeola.—The transmission of measles from mother to fœtus is a rare occurrence, but is not unknown. Thomas[3] was able to collect six cases from medical literature. There are also cases recorded of measles appearing in the first few days after birth, making it probable, from the short period of incubation, that infection had occurred in utero.

Scarlatina.—There are a few well-authenticated cases of children born with an unmistakable scarlatinous rash upon them, accompanied by fever and followed by desquamation and albuminuria. Those reported by Leale[4] and Saffin[5] are quite typical.

Erysipelas.—Kaltenbach,[6] Runge,[7] and Stratz[8] have reported cases that were in all probability erysipelas in utero. Lebedeff,[9] however, has presented convincing evidence of the possibility of intra-uterine erysipelas. In the subcutaneous tissues of a child born of a mother in the midst of an attack of the disease were found Fehleisen's micrococci. The child had lived only ten minutes.

Malaria.—Many practitioners have reported cases of periodic exacerbations of temperature in the new-born apparently due to malaria acquired during intra-uterine life. I had such a case myself recently, in which the temperature of a new-born infant rose on two successive afternoons to 103° F., the fever being preceded by great uneasiness; quinine to the mother in large doses promptly cured the child. Unfortunately, the germs of Labaran were not looked for. Quite recently I have had under my care in the Maternity Pavilion of the Philadelphia Hospital a woman and her new-born infant in both of whom the resident physician, Dr. Preston, discovered the characteristic bodies in the blood-corpuscles.

---

[1] Schweizer, "Ueber das Durchgehen von Bacillen durch die Nieren," Virch. Arch., Bd. cx. S. 255.

[2] For an extensive bibliography of intra-uterine variola and vaccination, see Tarnier et Budin, op. cit.; Wolf, Virch. Arch., Bd. cv.; Chambrelent, loc. cit.

[3] Ziemssen's Handbuch, vol. ii. p. 50.

[4] Med. News, 1884, p. 636 (good bibliography).

[5] New York Med. Record, April 24, 1886.

[6] Centralbl. f. Gynäk., Nr. 44, 1884.

[7] Ibid., Nr. 48, 1884.

[8] Ibid., ix. 213.

[9] Zeitschr. f. Geburtsh., xii. 321.

Tuberculosis.—Curiously enough, the transmission of tuberculosis to the fœtus in utero is an exceedingly rare occurrence. Demme once found the tubercle-bacillus in the macerated fœtus of a tuberculous woman: this is the only instance of fœtal tuberculosis that I know of, in human pathology at least. There is a similar case in veterinary medicine: Johne on one occasion found tubercles in a still-born calf, in which were discovered the bacilli of tuberculosis. While, therefore, the passage of tubercle-bacilli from mother to fœtus is a possible occurrence, it must be regarded as very exceptional.

Septicæmia.—The possibility of the transmission of septic micro-organisms from mother to fœtus has been denied by many, but the occurrence of septic infection in utero has been strongly affirmed by Koubassoff, Chambrelent, Pyle, Mars, Von Holst, and others. Von Holst,[1] after an extensive search through medical literature, asserts positively that although septicæmia in utero is rare, it has undoubtedly occurred.

Cholera.—Tarnier[2] says there is nothing to justify a belief in the occurrence of intra-uterine cholera, and Queirel[3] asserts that it is doubtful whether cholera directly affects the fœtus in utero; but nevertheless early abortion is the rule, or if the child is born alive it survives only a few days.

Typhoid Fever.—The most serious effect upon the fœtus of typhoid fever in pregnancy is usually a premature expulsion of the ovum: this occurs in sixty-five per cent. of the cases.[4] It would seem, however, that the disease can directly attack the fœtus; for Neuhaus[5] on one occasion found what are supposed to be the specific micro-organisms of this disease in the lungs, spleen, and kidneys of a four-months' fœtus from a woman who was convalescing after a prolonged attack of typhoid fever.

Articular Rheumatism.—There are two instances on record of the transmission of this disease from mother to fœtus,—one described by Pocock,[6] the other by Schaeffer.[7] In both instances a woman affected with articular rheumatism gave birth to a child presenting in one case at once, in the other at the end of three days, unmistakable signs of the same disease.

Recurrent Fever.—Albrecht[8] has described three cases of congenital recurrent fever, and in one fœtus he found the spirilla.

---

[1] Dissert. Inaug., Dorpat, 1884.

[2] Op. cit.

[3] Nouvelles Archives d'Obstét. et de Gynéc., 1887, p. 157.

[4] Duguyot, Thèse de Paris, 1879.

[5] Berlin. Klin. Wochenschr., 1886, S. 389.

[6] London Lancet, 1882, vol. ii. p. 804.

[7] Berlin. Klin. Wochenschr., 1886, S. 79.

[8] St. Petersburg. Med. Wochenschr., 1880, Nr. 18 u. 1884, S. 129.

Yellow Fever.—Dr. Bemiss,[1] of New Orleans, says, "The pregnant woman being attacked by yellow fever and recovering without miscarriage, immunity from future attacks is conferred upon the offspring contained in the womb during the attack." If this is true, it certainly seems that the fœtus too must have been infected by the disease.

Pneumonia.—Cases of fœtal pneumonia are reported not infrequently; in the great majority of cases they are no doubt the interstitial pneumonitis of syphilis. Geyl has shown, however, that if a fœtus be deprived of the necessary amount of oxygen for its blood, it will make inspiratory efforts in utero, drawing into its lungs liquor amnii with whatever that fluid may contain, a sufficient irritant in some cases to arouse a catarrhal inflammation. I have placed fœtal pneumonia in the list of infectious diseases on account of that observation already referred to in which the diplococcus pneumoniæ (Fränkel) was found transmitted from mother to fœtus during an attack of pleuro-pneumonia in the former.

Non-Infectious Diseases.—Under this heading are grouped rather loosely a number of heterogeneous affections. Inflammations of the serous membranes not dependent upon syphilis, with its accompanying exudations; some congenital skin-affections, as ichthyosis, hypertrichosis, albinism, purpura hæmorrhagica, and elephantiasis; intra-uterine diseases of the brain, which may consist in sclerosis, atrophy, lack of development, tumors, cysts, or inflammation of the membranes; diseases of the liver, whether multicystic or sclerotic; cystic disease, cirrhosis, or hypertrophy of the kidneys, and the congenital tumors, whether solid or cystic, malignant or benign, need simply be mentioned as in this class. There are other diseases, however, deserving a more extended notice.

Rachitis.—Intra-uterine rachitis is not common, but there is abundant evidence to prove that the disease may occur in utero. Indeed, it appears that in Europe at least congenital rachitis, in its minor grades, is by no means rare.[2] As the etiology of infantile rachitis is not at all clear, so the causes of the disease in the fœtus are all the more obscure. Most likely the nutrition of the mother is at fault; and not only improper or insufficient food, but also other unfavorable conditions of life, as cold, dampness, lack of light and ventilation, may play a part in the production of fœtal rachitis. In the more advanced degrees of the affection, an inspection of the product of conception after its expulsion from the womb can leave no doubt as to the true condition. A stunted growth, heavy joints, limbs bent in curves or angles and abnormally short, a distended belly with a "pigeon-breast," the large square head with gaping sutures and fontanels, and the bowed spine, all point unmistakably to this curious disease of the bones. A closer inspection will show that the long bones are either abnormally hard and tough, or that, while unusually thick, they are very fragile and may be snapped across by the slightest exercise of force; in this latter condition the medullary spaces have much encroached upon the external layer of hard bone, which may be reduced to the merest shell. This indicates a more active stage of the disease; but if, on the other hand, the long bones are firm, tenacious, and unbending, set, perhaps, in the unnatural shapes they have acquired in utero while in a more pliable condition, it is evident that the intra-uterine disease has run a longer course and has passed from an active process of destruction to one of attempted repair (and cure). The diagnosis of the disease in the fœtus during pregnancy is, of course, impossible; therefore no treatment will be attempted.[1]

---

[1] Parvin's Obstetrics, p. 222.

[2] "Die Rachitis, eine congenitale Krankheit," Felix Schwarz, abstr. in Allgemein. Wien. Med. Zeitung, Jänner, 1888, S. 6. This author claims to have found signs of rachitis in four hundred and three out of five hundred new-born infants examined.

Anasarca.—One rarely sees marked anasarca of the fœtus, occurring perhaps in connection with general dropsy of the mother, or as an entirely independent condition untraceable to any maternal affection. This disease of the fœtus usually determines its premature expulsion, most often between the fourth and eighth months, and the infant, even though it reach a viable period, is commonly born dead. Fœtal anasarca has been attributed to dropsy in the mother, to syphilis, in two instances to fœtal leukæmia,[2] in another to obstruction of the umbilical vein,[3] and Steinwirke[4] describes a case under the name of elephantiasis congenita cystica. The serous infiltration of the skin is often accompanied by collections of fluid in the abdominal and pleural cavities, and the placenta is often œdematous.

Spontaneous Fractures in Utero.—A syphilitic osteochondritis results not uncommonly in a separation of epiphysis and diaphysis in the long bones, simulating fracture. Advanced rachitis in the fœtus is undoubtedly the commonest cause of intra-uterine fractures occurring independently of violence during pregnancy and labor.[5] Link,[6] however, describes a peculiar "uncomprehended intra-uterine fœtal bone-disease" associated with extreme brittleness of the long bones: in a case reported by this observer, ribs, clavicles, and the long bones of the extremities were broken.

Luxations and Ankyloses.—These affections of the joints in fœtal life are not common. Dislocations have been found more frequently in females than in males, and are more commonly seen in the lower than in the upper extremities.[7] If in a breech presentation the presenting part is detained for a long time in the pelvic canal, there may be an apparent ankylosis of the hip- and knee-joints for some time after birth,—the limbs rigidly retaining the position they occupied during labor. True ankylosis is chiefly of interest in connection with the study of dystocia.

---

[1] See Tarnier et Budin, op. cit., p. 255; Schorlaw, Monatschr. f. Geburtsh., Bd. xxx. S. 401; Gräfe, Arch. f. Gynäk., Bd. viii. S. 500; Fehling, Arch. f. Gynäk., Bd. x.; Trans. of the Meeting of German Naturalists and Physicians, 1886; Virch. Arch., Bd. c. S. 256. Dr. Hamill recently presented a very typical case of fœtal rachitis to the Philadelphia Obstetrical Society.

[2] Klebs, Prag. Med. Wochenschr., 1878, Nr. 49; Sänger, Arch. f. Gynäk., Bd. xxxiii. H. 2, p. 161.

[3] Breslau. Klinik, Bd. i. S. 260.

[4] Dissert. Inaug., Halle, 1872.

[5] See Heinrich Braun, Arch. f. Klin. Chirurg., Bd. xxxiv. S. 668.

[6] Arch. f. Gynäk., Bd. xxx. S. 264.

[7] Tarnier et Budin, op. cit.

Intra-uterine Amputations.—Complete severance of some portion of the body from the trunk can almost always be traced to a disease of the amnion with the formation of constricting amniotic bands. The theory that a part encircled by the umbilical cord will be cut through is not tenable. Carl Braun points out that although the soft tissues may be constricted to the bone by a loop of the cord, yet the cord itself must yield before the osseous tissue is cut through. Ectromelic monsters have been erroneously described as the victims of intra-uterine amputations; and the writer once heard a pathologist hazard the opinion that an acardiac monster might be the result of the intra-uterine amputation of the head!

Perforation of the Intestine.—Paltauf[1] has recently reported five cases of death in the first few hours after birth due to perforation of the large intestines and escape of meconium into the peritoneal cavity. In three cases the rupture of the bowel was found at the sigmoid flexure; in another, at the splenic flexure; and in the fifth the point of perforation was in the transverse colon. In two of the cases there was a good opportunity to study the morbid process that resulted in this lesion, for several spots were found in the large bowel exhibiting the different stages of the diseased condition until complete perforation. The muscularis first gives way, next the serosa, and last of all the mucosa. A microscopic examination of the spots from a beginning in an extravasation of blood and rupture of the muscular coat until complete perforation showed a process of tissue-necrosis.

It will be noticed that rupture occurred four times out of five at the flexures of the colon, where an accumulation of meconium might exert the most pressure, and this seems the most probable explanation: just as a large collection of fæces in the lower bowel can end in fistula, so it would seem an unusually large collection of meconium in the colon might result in perforation. Although the accident occurred in Paltauf's cases after birth, the diseased condition of the bowel must have had its origin in utero; in fact, there are two records of intra-uterine rupture of the colon. In one a child was extracted after craniotomy; its abdominal cavity was filled with meconium which had escaped from a small perforation found at the junction of the ascending and the transverse colon: there were evidences of peritonitis with exudation.[2] In the second case there was discovered in a fœtus a peculiar abdominal tumor, which turned out to be a collection of meconium, encapsulated; hence the rupture of the intestine must have occurred in utero at a period quite remote from birth.

Fœtal Traumatism.—In spite of a position which secures it the greatest possible immunity from external violence, the fœtus has been seriously and fatally injured. In cases of gunshot, stab, or other perforating wounds of the abdomen in pregnant women, the fœtus has been also wounded.[1] The trocar, plunged into what was thought to be an ovarian cyst, has penetrated the child in utero, and wounds have been inflicted with sharp and dull instruments ignorantly used to bring on an abortion, or in the hands of physicians who overlooked the condition of pregnancy; even the examining finger has injured the child's head.[2] Falls from a height, blows and kicks, or a crushing force upon the mother's abdomen, have killed the child within her womb. The damage done the fœtus by this indirect violence is manifold: the abdominal viscera may be almost disintegrated,[3] the skull may be fractured,[4] there may be intracranial hemorrhage, leading, perhaps, as in one case, to intra-uterine hemiplegia.[5] A fatal injury to a fœtus from violence done the mother by another person might raise an important medico-legal question: the offence, if it could be proved, should be considered a grave one.[6]

---

[1] Virch. Arch., Bd. cxi. S. 461.

[2] [illegible], Monatschr. f. Geburtsh., 1863, Bd. xxi., supplem. hf. (quoted by Paltauf).

Diseases of the Fœtal Appendages which react injuriously or fatally upon the Fœtus itself.—The fœtus is essentially a parasite, depending for its well-being upon the health of its host and the normal condition of the tissues that put it into communication with its source of oxygen and nourishment,—the maternal blood. Diseases, therefore, of the placenta, cord, and membranes must exert a malign influence upon the health and growth or even the life of the product of conception. Degenerations of the placental villi, apoplexies of the maternal capillary loops that surround the villi in early intra-uterine life, thrombosis of the blood which moves in a sluggish current through the maternal lacunæ, retro-placental effusions which separate a certain portion of the placenta from the uterine wall, syphilitic overgrowth of the placental decidua which crowds in upon the inter-villous blood-spaces, must all abrogate the vital functions of the placenta to a greater or less degree, with the result either of destroying the fœtus outright, or else, half starving and strangling it, of producing at term a puny, wretchedly-developed infant. Even should the placenta be in perfect condition to perform its part in the physiology of the fœtus, the umbilical cord may fail to convey the blood to and from the fœtal body in a natural manner; the circulation in it may be obstructed by knots, although these by no means invariably cut off the blood-current; the cord may be compressed in other ways, wound tightly about some portion of the body, or caught between the child's limbs. The calibre of the vessels may be diminished also by disease of their walls, by the great growth of connective tissue encircling both arteries and vein that is commonly seen in syphilis, or the vessels may be almost occluded by a cellular infiltration of the cord-substance, which is also to my mind a valuable sign of syphilis. The umbilical vein shows constantly in one portion of its course, near the umbilicus, a physiological constriction: according to Leopold, fœtal death can in rare instances be traced to an exaggeration of this narrowing until almost complete atresia is produced, and this, too, in cases not syphilitic. The fœtal circulation may be disturbed, if not entirely suspended, by hemorrhage from the vessels in the cord; the escape of blood, however, into the cord-substance is necessarily limited by the narrow area in which it is confined; but in contrast to this is the bleeding that may follow rupture of the large branches of the umbilical vein spread out under the amnion on the fœtal surface of the placenta. I recently had the privilege of examining a very interesting specimen of this sort presented to the Philadelphia Obstetrical Society by my friend Dr. Hamill: pretty much all the blood of the fœtal body was collected in an enormous clot under the amniotic covering of the placenta, and the fœtus had evidently bled to death at some time prior to its expulsion, from a ruptured branch of the umbilical vein. This specimen, to the best of my knowledge, is unique. Diseases of the deciduæ need hardly be considered here, for their influence is usually felt early in pregnancy and is manifested by the premature expulsion of the ovum. Cystic degeneration of the chorion, too, almost invariably involves the destruction of the embryo or fœtus; yet cases have been reported of healthy, well-developed infants born at term with rather extensive cystic disease of the chorion villi. Abnormalities of the amniotic secretion have a very decided influence upon the growth and well-being of the fœtus.

---

[1] Hays, Ann. de Gyn., 1880, t. xiii. p. 153; Fennell, Trans. New York Path. Soc., vol. iii. p. 249; Tarnier et Budin, op. cit., p. 345; Guelliot, Gazette des Hôpitaux, 1886, p. 405.

[2] Dohn, Zeitschr. f. Geburtsh., Bd. xiv. S. 366.

[3] Von Hoffmann, Wien. Med. Presse, xxvi., 1885, Nrs. 18, 20, *et seq.*

[4] Hirst, Amer. System of Obstetrics, New York Med. Jour., 1888.

[5] Gibbs, Lancet, 1858, vol. ii. p. 497.

[6] For an illustration, see the case reported by Gorham, Wien. Med. Presse, Bd. xxvi. S. 170.

Hydramnion is so often associated with and dependent upon some defect or disease in the fœtus or mother that it would give an incorrect idea of the influence exerted simply by the increased quantity of liquor amnii to present the statistics of fœtal disease and death associated with this affection. And yet it is impossible to ignore the fact that a large quantity of fluid distending the uterine cavity must of itself become a mechanical hinderance to the free access of maternal blood to the placenta, while the increased intra-uterine pressure must exert an unfavorable influence upon the fœtus. There are a few who, believing the fœtus derives nourishment from drinking the liquor amnii, would look to the composition of that fluid for an explanation in some cases of fœtal malnutrition; but I am not of that number. The fœtus, it is true, swallows liquor amnii, perhaps in considerable quantities but from time to time there are born children with a lack of continuity i the upper part of the alimentary tract, and yet they are well nourished an full-grown. The quantity of albumen, too, in the liquor amnii is again the theory that the fluid can support life.

The amniotic fluid plays an important part in the growth of the fœ by distending the uterine cavity, allowing room for the free play of fœtal movements, and preventing injurious pressure by the uterine walls. Therefore an insufficient quantity of fluid will prove a disadvantage to the fœtus. Schatz[1] has reported a good illustrative case,—an infant born with ulcers on the internal malleoli and inner surface of the knees, due to an extraordinarily small quantity of liquor amnii. Some curious deformities in the fœtus may be traced to the same cause.[2]

The study of fœtal disease is a large and interesting one, of no little practical importance. To obtain a thorough grasp of the subject it would be well to consider carefully normal growth, development, and existence in utero, in order intelligently to contrast pathological with physiological processes. It would be an advantage to begin the observation of the fœtus as well as its treatment a hundred years before its procreation, in a study of antecedent generations; but all this is beyond the limitation imposed upon me by the title of my chapter. Even a superficial examination, however, of simply the diseases that affect intra-uterine life cannot fail, I think, to clear up much that would otherwise be obscure, perhaps inexplicable, in the life-history of the young infant, the child, and the adult.

---

[1] Arch. f. Gynäk., Bd. xix. S. 329.

[2] Tarnier et Budin, op. cit., p. 294.

THE CARE OF THE CHILD AT AND IMMEDIATELY AFTER BIRTH IN HEALTH AND DISEASE.

BY R. A. F. PENROSE, M.D., LL.D.

THE CHILD IN HEALTH.

As soon as born, the child should be placed on its *right* side, since this position, for evident reasons, favors the prompt closure of the foramen ovale. Its face should be turned *from* the maternal organs, thus avoiding the possibility of any sudden discharges from these organs, as blood, coagula, or placenta, entering or obstructing the respiratory orifices. Finally, the child must be placed sufficiently near the mother's body not to put the umbilical cord on a stretch and thus, prematurely, drag on the placenta.

When respiration is thoroughly established—a circumstance that, under ordinary conditions, happens immediately after delivery—the cord must be ligated and the child separated from its maternal connection. Should the business be left to Nature, something of this sort would happen. In the course of a variable period (from a few minutes to several hours, or even days) the placenta would be expelled from the vagina, and the pulsations in the cord would gradually cease, since the functions of the placenta are now no longer performed, and the act of respiration has caused the blood of the child to circulate in new channels and organs. This is what takes place after delivery in animals. The young animal is born with its placenta attached; the mother separates it by biting the cord, and, in some cases, devouring the placenta, leaving only a part of the cord adhering to the umbilicus of its offspring. In other words, the mother divides the cord by a sort of natural écraseur, and thus prevents hemorrhage from the vessels of the stump. This stump speedily dries and sloughs, since both placenta and cord derive their organic supplies from the maternal vascular system, and, necessarily, die when the placenta becomes separated from its uterine connection. However, the human female, unlike the cow, does not look upon the placenta of her offspring as a *bonne bouche*, and we therefore separate it by other means than by making a meal of it.

We begin by applying a ligature to the cord. Before doing so, the child should be brought fairly in view, and the medical attendant should have the assistance of some other person. It has been suggested that these manipulations should be performed *under* the covering protecting the mother, so as to avoid *unseemly* exposure of the woman's person. All such advice is based on a sentimental mock modesty, more nearly related to immodesty than to real purity.

The child should then be brought fairly in view, and, as respiration is completely established, it may lie on its back, as a more convenient position. Before applying a ligature, we examine the root of the cord, to ascertain if it is in a normal condition; since, should umbilical hernia exist, it will be necessary to reduce the protruding abdominal structures before ligation. Again, should the cord be a thick one, a "fat cord," it is desirable to press away with the fingers the gelatinous matter surrounding the blood-vessels, at the point we propose to tie, before applying the ligature, since, should this precaution be neglected, it may happen that the gelatinous matter will ooze from the cut extremity of the cord, reducing the bulk of the terminal part of the stump, and the consequence may be slipping off of the ligature, and subsequent hemorrhage from the vessels of the stump.

We apply this ligature, as I have just remarked, as soon as the function of respiration becomes fully established,—that is, when the child breathes well, and, in most instances, has cried lustily. This assures us that the placental functions are no longer necessary, and we separate the child from its placenta, even though the blood-vessels of the cord still pulsate strongly. Should we wait, as has been suggested and urged, this would happen. The pulsations would gradually become more and more feeble, and finally would cease altogether, failing to be felt first at the placenta. No good, however, would attend or follow this over-sensitive conservatism. Much time would be lost, thus delaying the attentions necessary for the comfort and welfare of the mother; while the child would be exposed to the risk of "taking cold," etc.

The ligature may be any strong string not too thick. Nothing answers as well as a skein of common thread, which should always be prepared beforehand. This skein should be cut, the ends tied, and a ligature secured of the proper thickness, and so strong that it cannot break. I never use any other material. The string usually furnished for this purpose in the "baby-basket" is so rotten and weak that it *generally* breaks at the time of tying, not only causing annoying delay, but also taxing severely the patience of the often wearied medical attendant.

It has been suggested not to tie the cord at all; and many evils have been assumed as consequences of ligation. Diseases of the blood-vessels of the stump of the tied cord, diseases of the liver, and the jaundice incident to such affections, are by some attributed solely to ligation. As every medical practitioner of judgment and experience well knows, these evil results of ligation are absolutely imaginary; while dangerous or even fatal hemorrhage from the unclosed blood-vessels of the cut cord is too real a trouble to be forgotten or ignored.

Undoubtedly, if the cord were divided by an écraseur, or by biting, instead of by a sharp instrument, the danger of hemorrhage would be greatly lessened, but it would never be so completely obviated as by the application of a ligature,—an expedient as safe as it is sure.

The ligature should be applied about the breadth of three fingers from the child's body; that is, far enough to allow for re-tying should any accident render this necessary, and not so near as to cause the true skin of the child to be caught in the tie.

It has been advised to apply a second ligature about two inches nearer the placenta than the first, or child's ligature, and to divide the cord between the two. This practice should be limited to cases of plural births; and the reason for its employment in these is, that, where there are two or more fœtuses in the cavity of the uterus, the placental circulations of the children sometimes, though rarely, communicate, and, as a result of this communication, after the birth of the first child, should the second ligature be omitted, hemorrhage *may* take place from the placenta of the child or children still in the cavity of the uterus. When, however, we realize that we have to do with a single child only, we should not apply the second ligature. It must be borne in mind that the blood which escapes, for a few minutes, freely from the placental extremity of the cut cord is useless blood, —is fœtal blood, not maternal. It is blood that has done its work, and, like the placenta, of whose mass it forms a large part, its life and usefulness are ended. This placental blood is not needed by the child, whose vascular system has retained all that is necessary for the welfare of the individual, and any attempt to secure for the child a portion of this blood will be useless, or worse than useless, since, if successful, the delicate blood-vessels of the child would become too full, and a plethora would be produced that might have the most disastrous consequences,—a plethora which, even under the most favorable circumstances, would be very much more undesirable than its opposite condition of anæmia. When the placental extremity of the cut cord is not ligated, blood flows freely from it for a few moments after the vessels are divided. This escape of now useless placental blood is important in the physiology of the third stage of labor. In consequence of its escape, the mass of the placenta is greatly reduced in volume, perhaps to a size of less than one-half of what it would have been had a second ligature been applied and the blood retained. This great shrinking of the after-birth, prior to its expulsion, undoubtedly helps greatly the speed and ease of its delivery.

The next thing, after ligation, is to cut the cord. The best instrument for this purpose is a pair of strong, *sharp*, blunt-pointed scissors.

Here again we are advised, in the same vein of mawkish prudery that I have already referred to, to seize the cord in a particular manner, *i.e.*, between certain fingers and the thumb, and to cut it, thus guarded between them, *under* the clothes, so as to *avoid exposure.* The great objection to this highly moral and modest method is that at times the embarrassed attendant has cut off the fingers or toes, or, still more unfortunately, the penis, of the unlucky infant, instead of amputating its umbilical cord. The best method is to direct the nurse, or some intelligent assistant (the child lying on its back), to hold the cord, thus guarding the child from any sudden movement of its limbs; and then the scissors can be employed to sever the cord with absolute safety.

In cutting the cord, we do so about a half-inch from the ligature, thus furnishing a little nubbin or button beyond the ligature which serves as a guard to keep it from slipping.

Having cut the cord, we wipe the cut surface of the stump with a towel to assure ourselves that the occlusion of the blood-vessels is complete, and then the child is ready to be handed to its temporary nurse.

Some caution is desirable in handling a new-born infant: the surface of the child is very slippery, and, should this be forgotten, the infant might easily escape from the grip of the careless attendant. The child may be seized and handed to its nurse, securely and safely, by the following methods. Place one hand under the anterior part of the thorax, so that in lifting the child its head and limbs will hang flexed; or, supporting the back of the head and neck with one hand, grasp the inferior extremities with the other. The nurse, or some one *especially* detailed for the service, should receive the child in a piece of *well-warmed* flannel or other suitable material, and, wrapping it well up, should hold it in her lap or arms, preferably on the right side, until the time for its toilet. I say she should hold it in her lap, since I have known, especially in instances where the people were poor, and the apartment small,—I have known a child to be wrapped up in a piece of old blanket, or some old garment, then to be placed hurriedly and thoughtlessly on the mother's bed, or on a sofa, or a dressing-table, or a wash-stand, or a rocking-chair, or even on the floor. If it should happen to be a quiet child, and not cry, and should any circumstance direct especial attention to the mother, as hemorrhage, presently it will be forgotten. Small, and wrapped in its old covering, it looks, lying on the sofa or the floor, as if it were merely one of the many old towels or other old things used during the labor, and not the precious baby,—the cause of the whole business. I have known, under such circumstances, the new-born infant to be sat upon; to be rolled off the bed; to be trodden upon. Hence the great importance of the direction I have given,—that the new-born child is to be held by some responsible person, on its right side, from the time it is separated from its maternal connections until it receives its first toilet.

We find the surface of the child, at birth, covered, more or less thickly, with a sebaceous coating, the product of the glands of the skin, looking as if it had been plastered with a mixture of lard and tallow. This covering, whatever may have been its other uses, has served admirably to protect the delicate surface of the fœtus from the macerating influence of the liquor amnii, in which it has been soaking during gestation. Some children are much more thickly covered or plastered over with this substance than others; and in all children it is found in greater quantity in certain localities, as the head, about the genitals, etc. The soap and water employed to cleanse the infant from the accidental foulings incident to delivery, as blood, fæces, etc., will not remove it. It should not be suffered, however, to remain. If left on the skin, presently it dries, cracks, and finally causes more or less irritation of the surface. We remove this sebaceous matter by anointing the infant, before washing, with some substance in which it is soluble. It is readily dissolved by animal oils and fats, by albumen, by alcohol. The substance usually employed to dissolve it is hog's lard.

We can now intelligently consider the toilet of the new-born child, and, as this toilet relates to both its health and its comfort, I will describe it specifically and minutely. If the weather be cold, this toilet must be made in a *very warm* room,—preferably in front of an open fire or grate. It must never be forgotten that the child, until the moment of its birth, has always been in a temperature of 98° to 100° Fahrenheit, and that any prolonged exposure to cold after birth may be followed by disease and even death.

The nurse should prepare beforehand and have within easy reach the following: a cup or tumbler of clean cold water; a large basin or a tub of hot water of not less than 100° Fahrenheit, while it will often be better to have the water of the temperature of 105° or even 110°; some *bland* soap, as old white Castile or pure palm; a teacupful of fresh hog's lard; soft wash-rags or sponges; some soft warm towels; some soft old rag: muslin is as good as linen for the purposes needed, or even better.

Sitting in front of the fire,—should there be one,—the baby on its back, and an assistant at hand to lend her aid if necessary,—the toilet is begun and conducted as follows.

The nurse should begin at the mouth, and with a clean rag over her finger wash out the buccal cavity; having removed the epithelial and mucous accumulations of gestation, the infant may be given a few drops of water to swallow, or a little sweetened water, taking it by sucking from a piece of clean rag, or from the finger of the nurse, or from a teaspoon.

The next step is to remove the sebaceous matter from the child's skin. For this purpose the nurse takes a piece of old rag,—nothing better than a piece of soft old flannel,—and, keeping the child well wrapped in its covering, begins at the head, rubbing the surface briskly with the lard by means of the rag; instantly the ceruminous coating disappears, dissolved by the lard; the capillary circulation, stimulated by the brisk friction, becomes active, and the surface in consequence a bright red color. Similar applications of lard to the other parts of the surface secure similar results; and presently the infant is ready to receive the detergent benefits of soap and hot water.

It is well to follow a systematic method in giving this bath. The nurse should begin with the head, keeping the body well covered. The hot water and bland soap quickly remove the lard and the sebaceous matter it has dissolved, together with the soiling incident to labor. The nurse should be cautioned to apply the soap prudently about the eyes. Conjunctivitis in the new-born child may be due to other causes than the acrid secretions of the maternal parturient surfaces. Among these causes are exposure to cold, to too bright light, and last, but by no means least, the careless application of soap to the eyes during the first bath.

After the head is washed and carefully dried, the same detergent application of soap and hot water is made successively to the other parts of the body, the child still lying on its nurse's lap, and still covered, save the part undergoing the cleansing process. Speedily the parturient soilings are all removed, and the infant is then ready for its grand and final hot bath. For this purpose it is best to have a large wash-basin or a bath-tub containing hot water that has not been contaminated by the cleansing wash just described. The temperature of this water should be the same as I have suggested as desirable for that used in the detergent wash,—that is, from 100° to 110° Fahrenheit.

Into this hot bath the whole body of the infant, save its head, is now to be plunged; and this immersion may be prolonged from half a minute to one or two or more minutes,—in other words, until the child is well rinsed, and, if need be, well stimulated by this bath of clean, hot water.

Usually, during the various manipulations I have described, but almost certainly during the final plunge, the child cries vigorously. This apparently mere automatic action is highly beneficial in completely establishing the respiratory function, stimulating, at the same time, in a most salutary manner the general as well as the capillary circulation.

The infant, on removal from its bath, should be enveloped in hot, soft, absorbing towels, and gently and thoroughly dried. The nurse should now change the apron she has worn during the bath for a fresh dry one, before proceeding with the dressing of the infant. Before applying the cloths, the stump of the umbilical cord must be "dressed." "Dressing the cord" means enveloping the stump of the cord in some soft absorbing material which will soak up the putrid fluids incident to the process of sloughing. Usually some old linen rag is furnished for this purpose. This linen rag is in no respect to be preferred to old muslin rag, and often is not so soft or absorbing as the old muslin, and therefore not so suitable for the object in view. The medical attendant should "dress the cord" himself, after which the nurse may put on the child's garments. There are several styles of dressing the cord. The usual one is to take a square of old rag (three or four inches square), composed of two or more layers; a hole is cut in the centre of this square, through which the stump of cord is passed, and is placed on the surface of the abdomen: or the cord may be dressed with borated or sublimated absorbent cotton. The cut surface of the cord is directed to the chin of the child; the square is folded over the stump, and then folded laterally, and thus the cord is enveloped in several layers of absorbing rag, which keep it well protected until the sloughing is accomplished. This covering may be kept more securely by tying it to the cord with a soft string. Another method of dressing the cord is to prepare a small roller, say a foot long and an inch wide, and apply this to the stump until it is protected by several layers of the material. The cord, being by one of these methods properly enveloped, is then to be secured to the surface of the abdomen, both for the child's comfort and to prevent injury to the blood-vessels of the umbilicus. This may be done simply by turning the end of the stump towards the child's chin, and securing it by means of a couple of strips of adhesive plaster,—a most excellent method. The usual mode, however, is to apply the "binder." This binder should be made of flannel, wide enough to reach from the hips to the axillæ, and long enough to go twice around the child's body; it should not be hemmed, as it usually is prepared, since the hem makes a cord, and when applied presses uncomfortably on the skin. In applying the binder the stump is turned towards the chin, and is secured by rolling the binder twice round the child's body and fastening with safety-pins. Nurses often apply this binder too tight, intending by so doing to prevent umbilical and other forms of hernia: really, however, the practice favors the development of hernia, or aggravates it if already existing. The binder should be applied as loosely as is consistent with the purpose for which it is used. It might be remarked here that a healthy infant, properly dressed, should be entirely comfortable, a good deal fatigued, and very ready for a good, quiet sleep, immediately after its first toilet. Hence, when an apparently healthy child, after being dressed, is restless and uneasy and disposed to cry, the difficulty, usually, is not "colic," which is almost always given as the cause of these symptoms, but some error in the dressing; and the physician should examine the child carefully to ascertain what it is. The best way to do this is to place the infant in the middle of a bed, then to have it undressed, scrutinizing closely each garment and the way it has been applied and secured; and it will often be found that the cause of the "colic" is some pricking pin, or too tightly applied binder, etc., the removal of which will be speedily followed by the disappearance of all the unpleasant symptoms.

The stump of the cord will slough off in from four to seven days, leaving, in a healthy child, a superficial and healthy ulcer which quickly heals. Should the raw surface be disposed to become sluggish, some mild astringent may be applied, as the oxide of zinc, or iodoform, or a mixture of both of these. During the process of sloughing the stump becomes more or less offensive, sometimes quite disagreeably so. An excellent corrective application is, daily, after the child's bath, indeed, in some instances, oftener than once daily, to envelop the stinking stump and its original covering (which should not be interfered with) with several layers of well burnt and charred old rag; in other words, to apply several coatings of freshly-prepared charcoal. This acts most happily as an absorber of offensive gases, as an antiseptic, and, what is of equal or even greater importance, as a dryer; rapid desiccation of the stump being important not only to the speedy, but the safe, separation of the decomposing structure.

Before putting on the garments the child should be examined carefully all over, to ascertain the presence or absence of any vice of conformation.

The medical attendant is not usually consulted regarding the clothes the infant is to be dressed in. All that he is called upon to look after is that, in cold weather, the child is clad sufficiently warmly. This can roughly be determined by feeling its hands and feet: if these are warm, its clothing is usually sufficient; if cold and clammy, the child needs more covering. It must always be borne in mind that heat means life to the new-born. It is best to err by having too much rather than too little of it.

In view of the active circulation in, and the rapid development of, the child's brain after birth, no covering is applied to the head. Formerly this was not the practice, and all children had the head covered with caps from birth. The present practice of omitting the cap, for the reasons I have given, seems to be attended by such good results that it is to be commended.

The child's toilet is now completed. If the mother have recovered sufficiently from the fatigues of labor, it should now be put to the breast; indeed, this early application to the breast is so very desirable, for both mother and child, that no ordinary circumstance should be permitted to postpone it. For the mother it is valuable in securing prompt and continued contraction of the uterus, thereby preventing post-partum hemorrhage, excessive bloody lochial discharge, and after-pains. For the child it is important in furnishing some nourishment, and in affording an opportunity for early exercise of its automatic faculty of sucking, a matter of no little moment, since it sometimes happens, when the application of the infant to the breast is postponed for several days, that the faculty seems to be lost, and the child, ever after, refuses to take the breast. Should the child for any reason not be put to the breast, it may be given a few teaspoonfuls of warm, sweetened water, and then may be placed in its cradle or crib, on its right side, well covered, and out of all draughts. Its eyes should be protected from exposure to all bright lights, natural and artificial, and its surrounding atmosphere should be as pure as the possibilities of the situation admit of.

I have said that the child should be placed in its crib or cradle: I advise a cradle. It is the fashion of our generation, it was not of our fathers and grandfathers, to proscribe cradles. It is held that the rocking movement of the cradle is injurious to the child;—how injurious does not seem to be definitely determined.

This theory I am convinced, through long observation and large experience, is absolutely without foundation. Nay, more, I hold that the results of the soothing rocking movements of the cradle are positively beneficial. I consider a nursery lacking a cradle as destitute of an article not only of great present comfort to the child, and very great relief to its care-takers, but also of no little importance to the future welfare of the infant. The dreadful increase in affections of the nervous system which the present generation experiences is undoubtedly due to many and complex causes; and I here throw out the suggestion, would not the soothing influences of a cradle on the nervous system of infancy have rendered that system less irritable, and, consequently, less disposed to be injuriously affected by the innumerable causes of nervous disease incident to the whirl of modern life?

THE CARE OF THE CHILD AT BIRTH IN ABNORMAL CONDITIONS.

We must not expect to find the child at birth always in health, and presenting the appearances I have just described as characterizing that condition: it is not always plump and red, with a cry whose pitch and volume at once suggest the lungs of a youthful Stentor.

Sometimes the child is in a condition of debility, sometimes not only weak, but the victim of disease, sometimes apparently dead, sometimes really dead,—still-born.

The causes of these abnormal conditions are many and varied. They are the results of diseases of the fœtus during gestation, or the results of the accidents of gestation and parturition.

The product of conception evolves, during gestation, from a cell to a matured fœtus, and, in this evolution, passes through changes and metamorphoses of the most extraordinary nature; and yet, in healthy gestation, it accomplishes these changes and metamorphoses with a precision and exactness as mathematically accurate as the crystallization into definite and well-known forms of a saline solution. Hence, it is evident that if the *building-material*, out of which the future man is to be erected, be good, from it will evolve a structure that will be correspondingly good. The evolution of a healthy, well-developed infant is, then, not a matter of chance or accident; but it takes place as the result of laws as unerring and precise as the laws of crystallization.

Healthy men and healthy women, inheriting themselves good constitutions, and living healthy moral and healthy physical lives, *cannot* have any but healthy children. But, unfortunately, all men and all women are not healthy; unfortunately, they have either inherited or acquired bad constitutions. Still worse, few men and few women lead typically healthy moral and typically healthy physical lives; and the inevitable consequence of it all is that, when these imperfect men and imperfect women marry, if they have children, they must *necessarily* be more or less imperfect children.

The study of prenatal diseases shows that these conditions, to which I am now referring, cause all sorts of abnormal evolutions in the embryo and fœtus, and lead to a great variety of diseases, deformities, and monstrosities; they constantly cause the death of the product of conception during gestation, and, hence, Abortion, the great accident of gestation, is frequently due to them.

Over the threshold of life is written the declaration of Nature's righteous and inexorable law, "The fittest shall survive;" and this law, so just, so stern, so merciless in its unpitying exactions, is *the* law which governs, not only life's beginning, but life's progress and life's end.

Man's intellect may enable him to elude the workings of this law *for a time*, but ultimately its majestic omnipotence triumphs; ultimately the fittest *alone* survive.

Innumerable children die before birth, or at birth, or shortly after birth, or even *years* after birth, not because our science or skill is valueless, but because Nature's doom was pronounced at the moment of conception; and that wise and holy fiat, by which alone a perfect race of men can be possible, "The fittest shall survive,"—*that* fiat proves their destruction.

It is evident, therefore, that we are not to mourn, nor even to regret, every child that is born dying or dead; indeed, often this, apparently sad, termination of gestation is a matter for congratulation rather than grief; and though we are professionally bound to do all we can for our suffering charges, we may comfort ourselves, when the results are unfavorable, with the reflection that man cannot contend successfully against the laws of Nature.

Sometimes, then, children are born dead, sometimes apparently dead, sometimes in a condition of asthenia or debility. Let us consider first the condition of Asthenia or Debility.

DEBILITY IN THE NEW-BORN CHILD.

There is no difficulty in recognizing asthenia in the child. The infant is pale, at times blue; its features shrivelled; if the victim of prenatal disease, often more or less emaciated; though just born, often presenting the appearance of age and decrepitude.

In these cases, we listen in vain for the welcome music of the child's first cry; instead, we notice the convulsive gasp, or hear low moans, and perhaps the gurglings of air, as it is painfully and laboriously drawn through the mucous accumulations in the larynx and trachea. The child breathes imperfectly, either because it is too feeble to expand its lungs, or because, being a premature child, these organs are not sufficiently developed. Hence its blood is not aërated; hence it is blue; hence it is cold; hence it cannot cry.

In the treatment of the new-born child, in such conditions, we must carefully bear in mind the possible causes of the asthenia.

Perhaps we are most frequently called to treat the debility in premature children; children born, more or less, before full term. We must be careful not to exhaust the feeble or fainting child by our washing, etc. A weak child might die if subjected to manipulations most desirable for a strong and healthy one. The child may be too weak to rub with lard and wash. If possible, however, it is best to grease and wash it. Under such circumstances, the water should be as hot as can be used, from 110° to 120° Fahrenheit; and sometimes it is best to use whiskey, or whiskey and water, at the same temperature. At times the child is so weak that it is not prudent to dress it; and all that can be done is to envelop it in hot flannel, or, better still, in hot carded cotton or wool.

There are three elements, each of which is essential to the proper management of these feeble children. These essentials are, the removal of all obstructions to respiration, a very high external temperature, and the use of nourishment and internal stimuli.

Obstructions to respiration in the mouth should be removed by wiping out the buccal cavity: those in the larynx and trachea are not so easily got rid of. An expedient, at times, of value is to hold the child by the lower extremities, with its head down, and then to shake it briskly, or spank it sharply on the nates: a sudden inspiration, followed by a cough, may remove the whole trouble.

Should such efforts fail, nothing is left but to wait, in the hope, too often vain, that the child will ultimately acquire strength sufficient to take a full inspiration, and thus get rid of the obstruction.

The second essential is a *very high* external temperature. We must recollect that these feeble children breathe more or less imperfectly; hence they do not inhale nearly enough oxygen to aërate their blood; hence they must be cold; hence they must be weak.

As such children cannot make heat for themselves, we must supply it from without. Should we permit the child's temperature to fall much below normal, it will certainly die: many feeble children die from this cause alone, who, treated in the way I am about to describe, would as certainly have survived.

It is difficult to define with precision the exact amount of heat demanded or borne under such circumstances. Each case requires such careful and constant attention and watching, that the temperature may be increased or diminished as may seem to be necessary.

In many cases the temperature should be *paradoxically* high. The body of the child should be kept at a temperature of not less than 98° to 100° Fahrenheit, and, to secure this, may demand almost literal "roasting." Hot bottles and bags, etc., are to be used; so also is radiated heat from an open fire. The surrounding atmosphere should be *very hot;* in this way the blood is warmed through the lungs, as well as external temperature maintained. Cases are reported where feeble new-born children have been given up as dead, have been left hopelessly in front of a very hot fire, and, after a prolonged "toasting," have been discovered to be alive, and have subsequently done well.

The third essential, in these cases, is the use of nourishment and internal stimuli. The child is too feeble to take nourishment by sucking, and it should be administered by a mop or a teaspoon. It should be given in small quantities, a few teaspoonfuls at a time, *very hot,* and should be frequently repeated. Hot milk mixtures and hot dilute spirit are best. A good formula for the dilute spirit is, a teaspoonful of spirit (brandy or whiskey) added to five teaspoonfuls of hot water slightly sweetened. Of this mixture, the child may be given one or more teaspoonfuls from every few minutes to every hour or more, as may be necessary. I think I have saved the lives of some feeble children by intoxicating them with hot spirit-and-water, and keeping them mildly drunk for some time after.

By treating feeble children in this way we often have the great satisfaction of saving lives otherwise doomed. But often they die, in spite of all our efforts. They gradually become colder and colder; their faces and hands bluer and bluer; their respiration more and more gasping and feeble, until it finally ceases. Such cases die, not because the treatment has not been the best and most efficient, but because all treatment would have been useless.

THE NEW-BORN CHILD APPARENTLY DEAD.

Children are sometimes born not merely in a condition of asthenia, but in a state of apparent death; which apparent death speedily becomes real death, unless proper means are used to prevent it, and often in spite of all remedies. We find children born in this condition of apparent death presenting very different appearances. Sometimes the face and upper part of the body are red; sometimes marked with bluish spots, and swollen; the eyes are prominent and injected. Again, the child may be pale, and may exhibit marked evidences of profound prostration.

These varied appearances are produced by very different lesions, and are designated by a variety of names by different authors. Some speak of them as the "apoplexy and syncope" of the new-born; others, as the "congestive and simple asphyxia" of the child. Others reject these terms as very imperfectly designating the pathological conditions they are meant to describe. If we understand the causes that may produce these conditions, and the treatment necessary under the circumstances, it matters little by what name we call them.

OF THE APOPLECTIC CONDITION, OR THE CONDITION OF CONGESTIVE ASPHYXIA.

In this condition we find the surface swollen, the face red, or bluish, or spotted. The child lies apparently dead; makes no effort at inspiration, makes no movement. The heart may, or may not, pulsate. Should the child die, and a post-mortem examination be made, the vessels of the brain will be found gorged with blood, with, at times, effusions of blood into its substance, or on its surface. The thoracic and abdominal organs will also be found congested; and it is said that, at times, effusions of blood into the peritoneal cavity have been noticed.

The causes which may occasion these phenomena are either asphyxia, or direct compression of the cervical vessels of the child. Asphyxia produces them in the new-born child, just as asphyxia produces similar conditions in the breathing child, or in the adult. The blood is not aërated, congestion of the brain and lungs follows, and paralysis of the cerebral centres results. Anything occasioning asphyxia, either during labor or after delivery, may be considered as a cause of the apoplectic state of the child.

Hence, compression of the cord during labor, twisting of the cord, premature separation of the placenta, etc.,—in other words, anything suspending the fœto-placental circulation, before delivery,—will produce asphyxia, as surely as plugging up the larynx of the breathing animal will produce it. So, too, after birth, any cause suspending respiration, as mucus, or any other material, in the larynx or trachea, may occasion it.

The apoplectic condition may also be produced by any cause giving rise to direct compression of the cervical vessels. Hence we meet with it in face-presentation, and in cases where the cord has been wrapped several times round the neck during labor, etc.

It is not difficult to comprehend the pathology of these cases; indeed, a clear knowledge of this suggests, immediately, the appropriate treatment.

The child's brain is engorged with blood; this engorgement has produced pressure on the cerebral centres, which pressure has paralyzed their action. Hence, when the child is born, its brain fails to respond to those stimuli which Nature has provided to rouse it to the performance of the great function of respiration. The cold air, striking on the warm and wet surface of the child, ordinarily a most powerful stimulus to respiratory action, now is incapable of waking up the oppressed and congested and paralyzed medulla oblongata. But if we cannot awaken the medulla, the custodian of life's essential functions, the child must inevitably perish.

Bearing all this in mind, the treatment is evident. If the cerebral paralysis be the result of mere congestion, in most instances the child, properly treated, will recover. If the paralysis be due to effusion of blood into the substance or on the surface of the brain, it will die.

There are, however, no symptoms that enable us to determine whether the cerebral paralysis is the result of mere cerebral engorgement, and, therefore, curable, or the result of cerebral effusion, and, therefore, almost necessarily fatal. Hence we treat all these cases alike. Remembering that congestion of the brain is the curable cause of the paralysis, we must endeavor to remove it. We bleed the child; that is, we suffer to escape, from the cut cord, one, two, or even three tablespoonfuls of blood; should blood not flow from the cut cord, we may press and squeeze it from its insertion to the cut extremity. Failing in obtaining blood from the cord, an attempt *might* be made to obtain it by opening a vein; though I fear all such forlorn efforts would not succeed.

While the blood is flowing from the cord, sometimes, the blue color disappears; a rosy tint shows itself, first in the lips, then over the face, and, finally, over the body. The medulla acts, respiration is established, the child is saved.

The next remedy is the very hot bath,—a bath of a temperature from 105° to 120° Fahrenheit. This very hot bath acts as a powerful revulsive, tending to relieve the overloaded brain and to equalize the circulation, while at the same time it is a powerful stimulus to the respiratory cerebral centre. After depletion, or without it, a basin or bucket of hot water may be brought to the bed, should the child not yet be separated from the placenta, because depletion from the cut cord has not been practised, and the body of the infant may be plunged in the hot bath; after immersion for from a few seconds to half a minute, the body may be brought to the surface and water, as cold as can be obtained, may be dashed suddenly on the face and anterior surface of the thorax. This expedient is a most powerful stimulus to respiratory action; the first contact of the cold water with the previously very hot skin of the infant is frequently followed instantly by a sudden and full inspiration, and the treatment continued soon secures a satisfactory establishment of the respiratory process.

Should bleeding and the hot bath fail, there may be tried, as a forlorn hope, artificial respiration; though artificial respiration is indicated rather for the next condition I am about to describe, than for the present one of apoplexy or congestive asphyxia.

There are several methods followed in artificial respiration practised on adults, but for the apparently dead new-born child I am confident that there is but *one* way, and that way is to blow directly into the lungs of the child. To do this efficiently, the nostrils must be pressed upon, to prevent the escape of air from the nose; the larynx must be pressed back against the anterior surface of the cervical vertebræ, to guard against the air entering the stomach. The practitioner, applying his mouth to the mouth of the child, blows directly into it; if preferred, some tube may be used, but the mouth is the better expedient. As soon as the lungs are sufficiently inflated to depress the diaphragm and raise the walls of the thorax, the blowing is to be discontinued, and the thorax and abdomen are to be gently pressed, imitating expiration. The blowing is then to be resumed, and the mechanical expiration to be repeated, as long as it is thought desirable.

How long is it desirable to practise artificial respiration in this way on an asphyxiated child? This question is not easily answered, and I shall reply to it by giving the history of a case, all the parties in which subsequently were my patients for many years, and those now living are still under my professional care. Thirty-five years ago, the wife of a young physician was confined with her first child, under the care of a celebrated professor of obstetrics. The labor was complicated and tedious; the patient, during labor, and after delivery, was in great peril, demanding the entire attention of her medical attendant. The child, when born, was apparently dead. The old professor said to the young doctor father (the mother was unconscious, and therefore did not hear), "Doctor, cut the cord, and take the child away: it is dead, and your wife's condition claims my whole care." The father separated the child, carried it into the next room, and placed it upon a bed.

He then went back, and again asked the professor if he were sure the child was dead, receiving again a positive opinion that the child was dead, and that all attempts to revive it would be useless.

The father returned to his dead baby, and, having nothing to do, in a wild, hysterical, utterly hopeless sort of way, began artificial respiration, after the manner I have just described. Half an hour passed, with no results; the agonized father continued his efforts; an hour passed, but the infant seemed as hopelessly dead as it was before artificial respiration was attempted. The man's emotional paroxysm began to subside, and he began to realize that he was literally wasting his breath: still he did not desist. Suddenly he was startled by a slight, apparently spontaneous movement on the part of the child; with renewed energy he continued his labors, and in a short time normal respiration took place, and, to his supreme felicity, and the astonishment of the medical attendant, the child was saved. This happened thirty-five years ago. The great professor is dead; the doctor father (my friend and patient) is also dead; but the child, called back to life by the hysterical blowings of an agonized father, hopelessly practised for the very long period of perhaps an hour and a half,—the child, now a grave, mature man, still lives, the comfort and solace of the mother who that day so nearly died in giving him birth.

Let this most interesting case be my answer to the question, "How long shall artificial respiration be kept up in similar exigencies?"

Electricity and galvanism have been suggested as agents valuable for arousing these torpid nerve-centres, and may, I have no doubt, in some cases prove efficient. They should be employed after the other remedies I have suggested have failed.

SYNCOPE OF THE NEW-BORN CHILD, OR THE CONDITION OF SIMPLE ASPHYXIA.

Another condition of apparent death in the new-born child is syncope, or simple asphyxia.

In simple asphyxia, or syncope, we do not notice the swollen and turgid face, etc., that characterize the apoplectic condition that we have just studied. The child exhibits a mortal pallor, with all the evidences of profound debility. This syncope may be the result of two essentially different causes: it may be due to excessive debility of the child, or to some lesion of its cerebral centres. Hence we meet with it when the infant is diseased, or premature, or has lost blood during labor. Again, it may be the result of a prolonged labor, especially when the head has been subjected to great and long pressure. In such cases the brain is compressed, and the cerebral respiratory centres, in consequence, are paralyzed, and fail to act when the child is born.

Here we have the brain-centres paralyzed, but not from cerebral congestion or apoplectic effusions, as in the paralysis of the apoplexy of the new-born; the paralyzing pressure is from the *outside*, and not from the *inside*, of the head;—there is too little, not too much, blood in the child's brain. The indications for treatment in such cases are, in the first place, to preserve the connection between the child and the placenta as long as the latter performs its respiratory functions; in the next place, to endeavor to arouse the paralyzed cerebral centres to work; in the third place, to stimulate the feeble and fainting child generally and locally. Evidently these syncoptic children do not require bleeding: they have too little, not too much, blood. We direct, therefore, a large basin of very hot water to be brought to the bed, and the child, still attached to the placenta, is plunged into the water; the heat acts generally and locally as a powerful stimulant. Presently, as in the administration of the hot bath already described, the body is to be brought to the surface, and cold or iced water is to be dashed suddenly and forcibly on the face and anterior surface of the thorax. This acts as the most powerful stimulus we have, to arouse the benumbed cerebral respiratory centres to work, and this we keep up, immersing the body in the hot water, and alternating these immersions with the dashing of cold water over the face and anterior surface of the thorax, as I have already directed, for some minutes. Often, the first dash of the cold water will cause an instant response: the child will give a spasmodic gasp, the lungs instantly fill, and the infant's life is saved. If the child can swallow, it will be desirable, as soon as possible, to administer hot spirit-and-water freely.

When all pulsation has ceased in the cord, and we realize that the placenta is of no further use, we may separate the child. The subsequent treatment must be something like that which I have suggested as proper for the asthenic infant; that is, the removal of all obstructions to respiration, and active external and internal stimulation. These are the very cases for artificial respiration, practised as I have already described; for high external and internal temperature; for the use of the galvanic battery.

THE HEAD AS MODIFIED IN SHAPE AND DIMENSIONS BY PROLONGED LABOR.

Children after tedious labors are sometimes born with their heads greatly compressed, and frequently much out of shape. It is not well to interfere in these cases: the proper treatment is to trust to Nature, and not attempt to force or squeeze the head into a good shape and appearance. In a few days the natural elasticity of the structures will bring all the parts into harmonious relationship.

The swelling of the scalp occurring as a sequence of tedious labor (caput succedaneum) is also not to be actively treated. The effused blood in a few days will be absorbed, and the child will experience no subsequent inconvenience.

THE CLOSURE

OF THE

DUCTUS ARTERIOSUS AND OF THE UMBILICAL AND HYPOGASTRIC ARTERIES.

By J. COLLINS WARREN, M.D.

At the period of birth, and before any structural change has taken place in its walls, the ductus arteriosus forms a more or less tortuous canal running obliquely downward from the pulmonary artery to the aorta, into which it opens just below the somewhat sharp curve of the lower border of the arch at the beginning of the descending aorta. The ends of the duct are still open, but in the central portions the walls are approximated, partly from the twisting of the vessel, which now is empty, and partly from a circular and longitudinal contraction of the walls. Water will, however, readily trickle through the canal.

The anatomical structure differs materially from that seen in any other portion of the arterial system.

The inner coat forms one of the most marked peculiarities of the canal. The enormous thickness of the intima is readily apparent, and it also appears to vary considerably in width at different points when seen in longitudinal sections. The inner surface consequently presents many irregularities, and frequently sharp projections. This condition is probably more apparent than real, the inequalities being largely caused by the twists and curves of the canal, which render it impossible to cut sections of the wall which are in all parts parallel to the axis of the duct. The intima rapidly diminishes in thickness as it approaches the opening into the great vessels.

The cells of which the intima is composed lie in a transparent intercellular substance, and are fusiform. They are arranged for the most part longitudinally. By some authors they are supposed to be connective-tissue cells, by others, muscular. The more superficial cells do not, as a rule, present the type of the muscular cell; but very perfect examples of the muscular cells are seen in the deeper portions of this layer.

The boundary-lines of the different coats are exceedingly indistinct. The lamina elastica appears to be wanting at many points; it is more readily made out in cross than in longitudinal sections. The media consists chiefly of bands of longitudinally-arranged muscular cells: these are occasionally separated from one another by circular bands of muscular cells, chiefly at the outer border of the vessel. This coat is almost entirely a cellular one. The few elastic fibres which it contains can be traced into the aorta and the pulmonary artery; they are most abundant in the outer layers of this coat, and occasionally extend throughout its whole length.

As the ductus enters the aorta very obliquely, the angle made by the upper edge of the ductus with the aorta is an acute one, and the wall is here thin in comparison with the lower margin of the opening, where the wall of the aorta forms a nearly continuous straight line with that of the ductus. The tissues of the coats of the aorta and ductus are interwoven at this point, and those of the ductus are spread out in a fan-like shape and are lost in the different layers of the wall of the aorta. The elastic lamina of the aorta does not form here a continuous layer, but is broken into several more or less parallel layers. According to Thoma,[1] that portion of the wall has a muscular reinforcement, which he places in the intima. This confusion of the layers is in reality due to the tendency of the elastic tissue to form a less perfect limiting membrane near the ductus, where all layers are ill defined.

The arrangement of the walls of the pulmonary artery resembles that seen at the aortic opening, but in a reverse order. There the upper wall is the thicker, and it receives the tissues of the upper wall of the ductus, which are freely interlaced with it. The difference between the two margins of the pulmonary opening is not so marked as at the aortic end.

A few weeks after birth a very marked change has taken place in all the tissues of the ductus, which appear to be undergoing hyaline degeneration preparatory to absorption. The outer walls alone remain, and later appear to become greatly strengthened and form a layer of circular muscular fibres which encloses the tissues of the ligament and is continuous with the mediæ of the two great vessels.

A longitudinal section of the ligamentum arteriosum at this period shows well the relation of the degenerating tissue to that which remains.

The central portion of the ligament is composed of a mass of degenerated hyaline tissue, a cleft in which indicates the site of the former duct.

Surrounding this is the wall of muscular tissue, which can be traced into the outer layers of the adjacent mediæ of the great vessels.

At the site of the aortic orifice we find the inner layers of the media aortæ greatly approximated, but still not in contact. The intervening space is occupied by a transparent tissue in which large numbers of spindle-shaped cells are embedded. This new growth springs partly from the intima and partly from the media aortæ. Its centre is pierced by a small vessel, which soon breaks up into capillary branches.

---

[1] Thoma, Arch. f. Path. Anat., 1884.

At eighteen months the ligament is fully formed, and undergoes no essential change in later life. It now consists of a dense bundle of longitudinal fibres composed of fibrous and elastic tissue interspersed with fusiform cells: here and there traces of the hyaline degenerated tissue are found. Surrounding this bundle of fibres is a layer of circular muscular fibres of varying thickness, and enclosing all is the adventitia reflected from the adjacent vessels. In the central axis of the ligament a small vessel is usually found which can be traced either directly or through a few capillaries to the aorta or pulmonary artery; occasionally a cleft or blood-space, lined with endothelium, is seen connecting at either end with a small vessel. Less frequently the bundle of fibres of which the ligament is composed is divided, a mass of loose areolar tissue occupying the space between them: this tissue usually contains a rich capillary net-work.

At the aortic end of the ligament there is still a slight opening in the media aortæ. This coat is slightly everted at this point, and forms a depression in the aorta, quite visible to the naked eye, situated just beneath an overhanging ridge formed by the inferior wall of the aortic arch.

The free edges of the media aortæ at this point are somewhat expanded, and interlace with the tissues of the ligament, which are here much richer in cells. Bands of muscular fibres can be seen running from the ligament into the media and deeper layers of the intima aortæ. In the centre of the depression is an arteriole surrounded by a growth from the superficial layers of the intima aortæ. Occasionally no depression is found, and the central arteriole is then wanting, the edges of the media aortæ being directly united by a musculo-elastic growth from that layer.

The same conditions are observed at the pulmonary extremity of the ligament, but in a less marked degree.

In later life patches of calcification are seen here and there in the ligament, which are probably caused by further degenerative change in islets of unabsorbed fœtal tissues.

In brief, the fœtal tissues at the period of birth undergo a hyaline degeneration and are absorbed. They are replaced by a ligamentous tissue which assumes a more muscular character at either end, where it is attached to the mediæ of the two great vessels and is surrounded by a musculo-elastic layer throughout its whole length.

Occasionally at the period of birth the ductus is occluded by a thrombus; but this is exceptional. The presence of a thrombus in such a position is interesting in the light of recent discussions as to its traumatic or septic origin.

The muscular and elastic nature of the tissue which seals the aortic opening, the overhanging ridge above the depression, and the obliquity with which the ligament is inserted into the aorta, all combine to give great strength to this spot in the aortic wall.[1]

---

[1] The Healing of Arteries after Ligature in Man and Animals, William Wood & Co., 1886.

According to Thoma,[1] a thickening of the intima aortæ is seen, after closure of the fœtal vessels, extending from the ductus arteriosus through the entire length of the descending aorta to the origin of the hypogastric artery, lining nearly the whole tract concerned during fœtal life in the umbilical circulation (*Nabelblutbahn*). It is described by Thoma as a hyaline connective tissue containing at some points large spindle-shaped cells and branching anastomosing cells. This he interprets as a compensatory thickening of the wall adapting the lumen to the new conditions of the circulation.

Thoma regards these cells as connective-tissue cells; but it is not improbable that many of them are muscular in origin.

The fœtal cord encloses three blood-vessels,—the umbilical vein, and the two umbilical arteries, which are continuations of the two hypogastric arteries.

The hypogastric arteries take their origin nominally from the internal iliacs, but in reality form with the upper portion of those vessels the trunks from which the future internal iliacs are given off as branches a short distance below the point of bifurcation with the external iliac artery. They ascend from the pelvis in front of the bladder, occupying the positions of the future superior vesical arteries, to the anterior abdominal wall, and pass through the umbilical ring to become continuous with the umbilical artery.

The umbilical vein originates in the placenta, runs with the cord in spiral convolutions and enters the abdomen at the umbilicus, attaches itself to the loose connective tissue of the anterior aspect of the suspensory ligament, and thus reaches the liver. After giving off a few branches to the left lobe, it divides into two main branches, one of which enters the portal vein and the other the vena cava.

The hypogastric artery presents certain peculiarities which distinguish it from other arteries: its walls are thick and strong and contain a large amount of longitudinal muscular fibre. In this respect it bears a resemblance to the structure of the ductus. It has another peculiarity also in common with that vessel, consisting in the absence of a well-defined outline to the inner wall of the media which a well-formed lamina elastica gives. Elastic tissue is found separating the two coats, but the membranes are thin, not always continuous, and sometimes hard to find. Near the distal end of the vessel and in the umbilical arteries there is little to be seen of any such structure. The tortuous character of the umbilical arteries produces great irregularities on the inner surface. The large amount of muscular fibre favors rapid contraction of the vessels and cessation of the blood-flow although no ligature have been applied. This vigorous contraction, according to Jacobi,[2] is the result of the rigor mortis of the muscular layer and the reflex action produced by the influence of the cooler temperature surrounding the newly-born.

---

[1] Op. cit.

[2] Brooklyn Medical Journal, March, 1888.

The umbilical vein differs but slightly from the arteries, the muscular layer being well developed. There is no true lamina elastica, and, according to Jacobi, no intima. This statement may hold good for the umbilical arteries, as also the assertion that there are no nerves nor vasa vasorum in their walls; but the hypogastric arteries in these respects do not differ essentially from other vessels of their size.

When the placental circulation ceases, a marked contraction takes place throughout the greater part of the vessel, and its most distal portion is filled with a thrombus. While the healing of the umbilical cicatrix is taking place, the distal end of the artery undergoes a hyaline degeneration which pervades its whole thickness. The extremities of the two vessels, in contact just within the umbilicus, are soon reduced to a cord of gelatinous tissue in which all traces of the vessel have disappeared. This change extends for a distance of two centimetres, and is subsequently replaced by a fibrous cord which attaches the superior vesical artery to the umbilicus. In the early days of life but little change is seen in the interior of the vessel: a slight proliferation of the cells of the intima is noticed near the apex of the thrombus, but not elsewhere. By the second month, however, a distinct growth of tissue may be observed throughout the entire length of the inner wall. The coats have contracted, and the inner surface is thrown into deep folds which are bridged over by a growth of young cells lying embedded in a hyaline intercellular substance. This new tissue fills out the irregularities, and in cross-section the lumen may now be seen to present a smooth contour. In the region occupied by the thrombus the growth of new tissue is most active: granulation-like masses intersect the clot and are rapidly obliterating the lumen. Complete obliteration of the vessel does not take place, but in adult life a considerable portion still remains as the superior vesical artery. This vessel has, however, unusually thick walls, and the lumen is greatly diminished in calibre. A microscopical examination of its walls shows that the old hypogastric wall has been greatly contracted, the elastic tissue being thrown into deep folds when seen in cross-section. Within exists a tissue evidently formed after this contraction. The new tissue consists of an endothelium, a well-formed lamina, and a muscular layer. The adult artery has, therefore, a double set of walls, the old walls having apparently contracted to their utmost capacity, and the lumen being further narrowed by a compensatory growth. The newly-formed wall is distinctly muscular in character, and has evidently been developed from the old wall by a growth penetrating through the imperfectly-formed lamina.

Tracing the vesical artery to its extremity, we find the lumen constantly diminishing in size, the newly-formed internal coat becoming less distinct, until the structures examined consist of a cord of fibrous tissue in which a tortuous arteriole finally breaks up into smaller vessels which are not to be distinguished from capillaries. Beyond this point we see only a fibrous cord, in the centre of which is some denser, more opaque tissue, intermingled with traces of an elastic membrane.

The series of changes which has taken place since birth result in a contraction of the vessel, followed by a still greater diminution of its calibre by an obliterating growth in the interior. There is a complete destruction of the distal portion of the vessel, the result partly of hyaline degeneration and partly of an obliterating growth. About one-third of the vessel at its terminal portion is thus replaced by ligamentous tissue. According to Baumgarten,[1] this band of fibres is a cicatricial tissue which has stretched with the growth of the individual, the superior vesical artery being actually the same length that it was at birth. But this view is not in accord with the writer's observations, which show that a considerable portion of the vessel has been destroyed.

---

[1] Centralbl. f. Med. Wiss., No. 41, 1877.

INJURIES OF THE NEW-BORN.

BY THEOPHILUS PARVIN, M.D., LL.D.

THE injuries received by the child during or in connection with labor may be classified as external and internal, the latter of course being, as a rule, the more serious.

But a more convenient classification is given by the part involved; and thus these injuries will here be considered as *of the head,* associating with them those of the neck, *of the trunk, and of the members.*

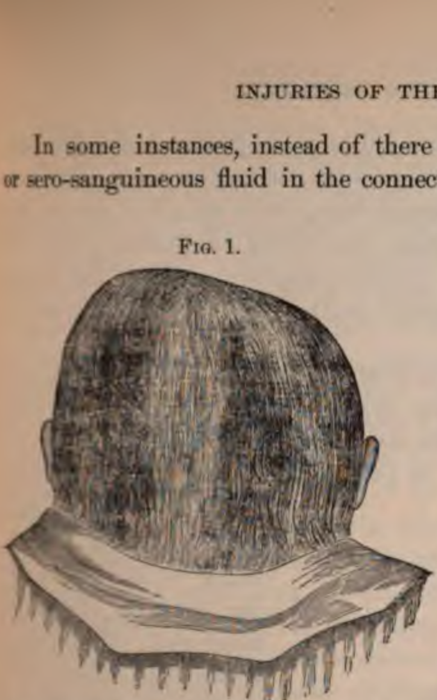
Injuries of the Head and Neck.—From the fact that, in the vast majority of cases, the cephalic pole of the fœtal ovoid descends the birth-canal first, that the propelling force of labor drives this passive mass against resistances, overcoming them, or, on the other hand, moulding that mass, modifying its form, and sometimes even its structure, and from the additional fact that in these cases of cephalic presentation, whether cranial or facial, the part is accessible to digital, manual, or instrumental means for facilitating delivery, it necessarily follows that injuries of the head during labor are much more frequent than those of any other part of the fœtus. The great majority are not serious; they are superficial, and in a few days usually disappear, either with or without the employment of very simple therapeutic means. Some, however, leave permanent disability, or even may be so grave that death results.

Caput Succedaneum, Sero-Sanguineous Infiltration, Kephalohæmatoma spurium.—This is a common, but not a constant, phenomenon; for, if the labor be rapid and the resistance slight, the child may be born without this swelling. Nevertheless, such cases are exceptional, and the occurrence of the caput succedaneum is so common that it might be regarded as a physiological condition.

This swelling may be round, or oval, or in some cases greatly elongated, projecting almost like a pudding-shaped mass. In some instances it may be less than an inch in its longest diameter, supposing it to be oval, but in others two or three inches. The skin which covers it has changed in color, in consequence of the congestion; if the labor has been long, the surface of the tumor may be purplish, or violet-colored. So, too, in case of protracted parturition the surface of the tumor may present phlyctenulæ which when ruptured leave the derm exposed.

In some instances, instead of there being simply an effusion of serum or sero-sanguineous fluid in the connective tissue, rupture of blood-vessels has occurred, permitting hemorrhage, which, breaking this tissue, may be so considerable that a fluctuating tumor results.

FIG. 1.

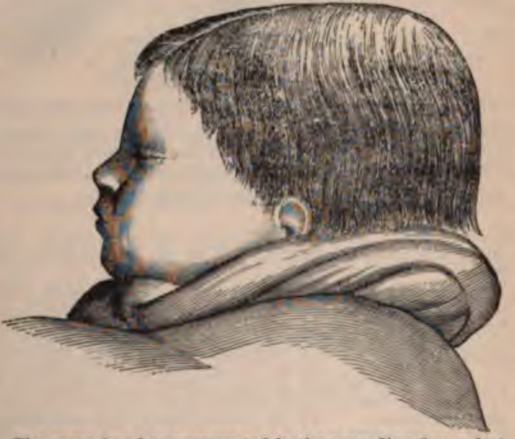


Caput succedaneum, vertex presentation, right occipito-posterior position. (From Depaul.)

The generally received origin of the caput succedaneum is that at that part of the child there is no pressure, while all other parts are uniformly pressed, and hence the former becomes swollen. Mauriceau explained the tumor as resulting from the resisting and partially-dilated os, acting as a cord about the part of the head where the swelling occurs, preventing the return of fluids: this explanation was accepted by Depaul as being in some instances one of the causes, the chief cause being that which has been stated.

The rule is that the swelling does not occur as long as the membranes are unruptured; but, as observed by Tarnier, such rupture is not absolutely necessary, not only according to the statement of Schroeder, but Budin has met with the tumor in some cases where the coverings of the fœtus were intact but extensible: Depaul also refers to the fact that this swelling may be developed prior to escape of the amnial fluid. (Figs. 1 and 2.)

FIG. 2.



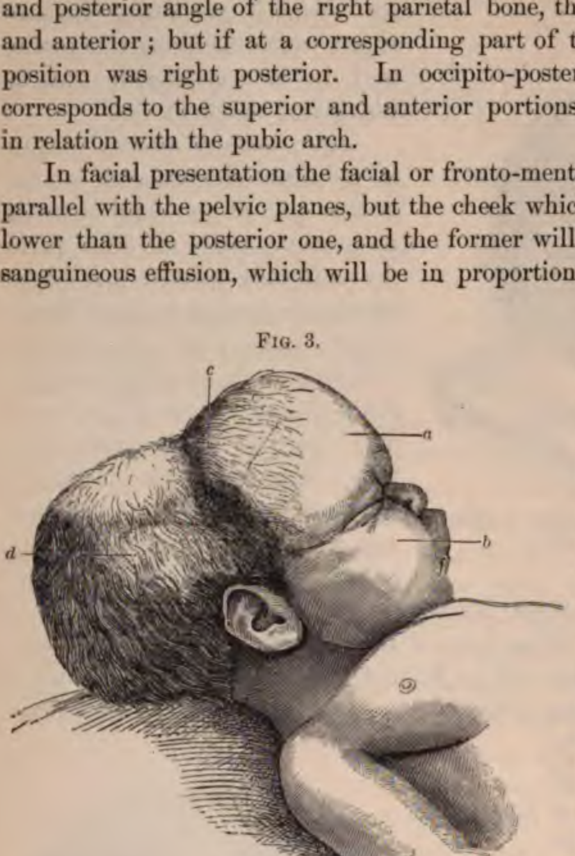
The same head as represented in the preceding figure, but some days after birth. (From Depaul.)

The caput succedaneum is usually formed during the dilatation of the os uteri; but should there be delay subsequently in any part of the birth-canal—such delay being especially frequent at the vulvar orifice—a secondary caput is formed. If the pelvic inlet be narrowed, and the head pressed against the resisting bony ring by active uterine contractions, sero-sanguineous effusion soon occurs, and the tumor often becomes so large as to approach the vulvar opening while the head still remains above the superior strait.

The seat of the caput succedaneum indicates the position which the head occupied in a cranial presentation. Thus, if the tumor be upon the superior and posterior angle of the right parietal bone, the occiput was at the left and anterior; but if at a corresponding part of the left parietal, then the position was right posterior. In occipito-posterior positions, the tumor corresponds to the superior and anterior portions of that parietal which is in relation with the pubic arch.

In facial presentation the facial or fronto-mental circumference is rarely parallel with the pelvic planes, but the cheek which is anterior is somewhat lower than the posterior one, and the former will be the seat of the sero-sanguineous effusion, which will be in proportion to the delay in anterior rotation of the chin; after this rotation, if delay occurs, the swelling occupies the entire facial oval. If extension is rapidly effected, the swelling is found upon the cheek, the mouth, and the chin; but if the process is slow, the deflection remaining for some time incomplete, then the swelling will be upon "that portion of the facial oval which occupies the centre of the basin,—that is, the frontal and ocular region." (Fig. 3.)

FIG. 3.



Caput succedaneum occupying the face and the vertex, *a, b, c, d.* The head was between flexion and extension, the occiput corresponding with the left extremity of the transverse diameter of the pelvis, and the forehead with the right extremity. The pelvis was narrowed, and the labor was long. (From Depaul.)

A single word may be said as to the position of the so-called caput succedaneum in presentation of the pelvis, and in that of the shoulder: the inappropriateness of the term is obvious, nevertheless the tumor designated by it has precisely the same origin and the same essential character as in presentation of the vertex. The swelling in presentation of the pelvis occupies the hip which is the lower, and this is usually, though not invariably, the anterior. "If in some cases the swelling upon the pelvic region is uniform, this is explained either by the slight obliquity of the presenting part, or its early correction, the two hips descending equally. Here, as elsewhere, the skin is of a more or less dark blue, and the tumor formed by the sero-sanguineous effusion variable in prominence and extent." If the child be male, the scrotum may become doubled in size, and black: indeed, instances in which sloughing occurred have been recorded. In presentation of the shoulder the sero-sanguineous tumor occupies the lowest portion, but extends thence anteriorly or posteriorly upon the trunk according as the latter may be inclined in front or behind. In case the elbow or hand descend first, then these become greatly swelled and discolored.

Diagnosis.—It is very rare that a true kephalohæmatoma forms during labor, and therefore mistaking a caput succedaneum for it will be almost impossible; the distinctive marks of the former will be given hereafter. An encephalocele has more of a cylindrical form, the skin covering it is not discolored, the tumor becomes larger when the child cries, the opening in the cranial vault through which the hernia comes may be discovered, and possibly pressure upon the tumor, in an effort to reduce the hernia, produces nervous accidents.

Treatment.—The vast majority of cases of spurious kephalohæmatoma recover without any treatment. Nevertheless, if the skin be broken, and if the swelling be great, or if the effusion be of blood rather than of serum, an erysipelas may arise from the former, or even gangrene ensue, and in the other case phlegmonous inflammation or suppuration may occur. Following a facial presentation, the great swelling of the eyelids and the subconjunctival ecchymoses predispose to conjunctivitis; the lips and the tongue may be so swelled that the child cannot nurse for several days, and it therefore must be fed.

The broken surface resulting from ruptured phlyctenulæ, or possibly from the rude use of the finger-nails, may be dusted with iodoform or with boracic acid; if the swelling is great, compresses dipped in a solution of muriate of ammonia or in a mixture of alcohol and water may be applied; should suppuration be threatened, warm fomentations and the application of a poultice are indicated; while if the distinct formation of pus be recognized, opening the abscess and washing out the cavity with a warm antiseptic solution would be proper.

Kephalohæmatoma, or Thrombus Neonatorum.—By this is meant a soft, fluctuating tumor of the scalp caused by effusion of blood between the periosteum and the bone. It is usually situated upon one of the parietals, upon the right more frequently than upon the left, in some cases upon both, rarely upon the frontal or the occipital, or upon one of the temporals. Authors generally state that the swelling never transgresses a suture; Bouchut, however, asserts that, while usually limited by a suture, it may pass over and involve the adjoining bone; he also quotes the remarkable case of Ducrest, in which the primary thrombus occupying one of the parietals passed over the intervening suture and under the other parietal.

This tumor does not usually appear until from one to three days after birth,—that is, when the caput succedaneum is disappearing; it may be no larger than a pigeon's egg, or may have the size of a small apple; the skin covering it is not discolored, and thus a marked difference exists between this tumor and that previously described; it fluctuates, is not increased in size when the child is crying, and usually presents a distinct bony margin around its base. Hemorrhages, either beneath or above the cranial aponeurosis, have been observed after the application of the forceps, but these are diffuse, have no bony margin defining their extent, and generally are rapidly absorbed.

Thrombus neonatorum occurs, according to Kleinwächter, once in two hundred to two hundred and fifty cases. The swelling disappears in some instances in two weeks, but more frequently it remains for a month or more. Rarely suppuration occurs, and this is liable to be followed by caries of the bone. If there should be also an internal as well as an external effusion of blood, the child perishes with convulsions.

The cause of the affection is by no means clear. Those who, like Earle, Godson, and Descroizilles, accept the opinion that it results from the portion of the head where it is found being constricted by the os uteri, can give no explanation for its occurrence, as has been the case in several instances, in pelvic presentations. Mildner and Hecker held that it resulted from the coats of the blood-vessels being thin, and consequently rupturing; while Langenbeck and Ritter attributed it to defective development of the bone.

Treatment.—Since absorption of the effused blood takes place in the great majority of cases spontaneously, and as the child does not suffer in any wise from the tumor, active interference is not usually indicated. By some the application of a solution of muriate of ammonia, of tincture of iodine, or of mercurial ointment, or compression by means of collodion, or of a thin plate of metal, is advised. Descroizilles remarks that these different applications appear to accelerate the disappearance of the tumor, and cannot cause any irritation or other accidents when prudently made. The employment of setons or of punctures is not advised; nevertheless, should an abscess form, opening it is indicated, and it is possible, too, if the collection of blood remains for some time without change, that aspiration, all antiseptic precautions being used, would be beneficial without in any respect being evil.

Wounds of the Scalp and of the Face.—Contused wounds of the face or of the scalp may be caused by the forceps, the accident depending upon the form of the instrument or upon the mode in which it is used: the prophylaxis belongs to obstetrics, and therefore will not be here considered. Generally such wounds are quite superficial, and disappear in a few days. In their treatment antiseptic powders, ointments, or fomentations may be used. Punctured or incised wounds of the scalp have usually been caused by the obstetrician mistaking the caput succedaneum for the bag of waters: antiseptic applications are indicated. More or less serious injury to the eyes has sometimes been done by the finger of the accoucheur in case of presentation of the face. Such injury, as well as that spontaneously resulting more especially to the eyelids in this presentation, do not require special directions as to treatment. In rare instances dangerous, and even fatal, consequences have followed sloughing of the scalp: this accident has been observed after spontaneous labor, and also has followed delivery with the forceps, one of the blades causing such severe pressure that gangrenous inflammation results.

Facial Paralysis.—This accident, in most instances unilateral, has been observed following spontaneous delivery, but in the majority of cases results from the use of the forceps, and is caused by pressure of one of the blades at the stylo-mastoid foramen, or a little in front of the lobe of the ear. As has been stated, in the infant the complete absence of the mastoid apophysis and the slight development of the auditory canal favor compression of the facial nerve near its point of emergence. In some instances only branches of the facial are compressed, and then the paralysis, instead of involving the entire half of the face, of course affects only the muscles to which those branches are distributed. Facial paralysis from intracranial causes will be referred to hereafter.

The paralysis will not be observed when the infant is sleeping, but when awake and crying, or when attempting to nurse, it is quite apparent. In the majority of cases recovery occurs spontaneously in from ten days to two weeks, and usually there is a notable lessening of the affection within a week. In rare instances the paralysis becomes permanent, remaining after years unchanged; and therefore the practitioner ought to beware of making a positive statement as to the certainty of recovery.

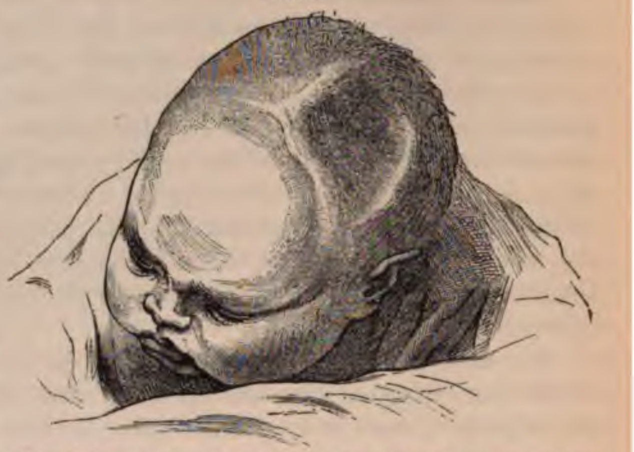
It is generally advised not to employ any treatment until at least a month has passed without any improvement; then electricity may be used, the induction current being first employed, and, if the muscles fail to respond, the continuous current.

Injuries to the Bones of the Head.—*Depressions, Fractures, and Dislocations.*—Depressions and indentations of the cranial bones are most frequently seen when delivery has been effected by the forceps, but they have also been observed after spontaneous expulsion of the child. Still more remarkable was the case reported by Matthews Duncan, in which a persistent impression was made by the finger of the accoucheur upon the right parietal bone in an effort to produce anterior rotation. The first illustration on the following page shows a funnel-shaped depression caused by pressure of the sacral promontory in a narrow pelvis.

While it was formerly believed that a fracture always occurred with depression, this view is no longer held. In one instance a country practitioner informed me that he had successfully used a cupping-glass to relieve a depression of the parietal in a new-born child; but such practice has not been recommended by any professional authority, though it seems rational, and certainly might be tried, as trephining has been proposed by an eminent American surgeon, Dr. Nancrede, in case of such depression, if paralysis be consequent. In most cases, however, these depressions in time disappear, or notably diminish. Indentations, whether made by the forceps or occurring in spontaneous labor, are frequently permanent, but are not usually the cause of any disability.

Fractures of the cranial bones have been observed following spontaneous, and artificial, whether manual or instrumental, delivery. The parietal bones are those most frequently fractured, especially where the fracture occurs in unassisted labor; but the frontal, the occipital, or one of the temporals may suffer this injury. The accident most frequently occurs in case of narrowing of the pelvic inlet, but has also been observed when there was no pelvic deformity, and the child normal in size, and it has been suggested that in such cases the injury may have resulted from the untimely administration of ergot, causing violent and rapid expulsion of the child. The posterior parietal bone is the one usually fractured, when the head is either driven or dragged through the pelvic inlet narrowed in the conjugate diameter, the injury resulting from the resistance of the sacral promontory. (Fig. 4.)

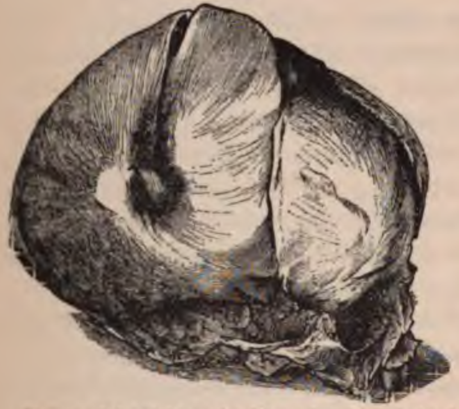
FIG. 4.



Funnel-shaped depression of the anterior half of the left parietal bone from the promontory. (From Winckel's "Lehrbuch der Geburtshülfe.")

Lomer[1] has recently reported twenty-seven cases of fracture of the skull from the use of the forceps. In ten the fracture involved the frontal bone, four of these injuries being over the orbit; five were of one of the parietal bones. The sagittal suture was ruptured six times, the lambdoidal four times, and the occipital bone detached in five cases. (Fig. 5.)

FIG. 5.



Fissure of the right parietal bone from the parietal protuberance to the sagittal suture, the anterior portion pressed under the posterior. (From Winckel.)

If the fracture have associated with it rupture of the longitudinal sinus, a mortal hemorrhage ensues. Even, however, if there be no injury to large blood-vessels, that of smaller ones may give rise to bleeding of consequence, or there may be injury done to the brain with that of the bone, so that these fractures should in no instance be regarded as trivial. Further, such brain-lesion may not always give immediate proof of its presence, but remote, it may be, in imperfect mental development.

---

[1] Zeitschrift für Geburtshülfe und Gynäkologie.

Experiment seems to have pretty conclusively proved that a force approaching one hundred pounds applied in extraction of the child, whatever the method, is very liable to produce fracture of one of the parietal bones. Fractures of the cranial bones are especially liable to occur in irregular applications of the forceps,—that is, when the blades are not applied to the sides of the child's head,—because they do not embrace uniformly so large a surface, and more pressure is required to prevent slipping. A less force will cause fracture under these conditions than in the ordinary method of using the instrument. Fractures of the bones of the face are almost exclusively those of the inferior maxillary, and result from traction made by the obstetrician's fingers in head-last labors. It seems probable, from the investigations of Matthews Duncan, Champetier, and others, that the inferior maxilla may be subjected to a force of about fifty pounds without sustaining injury. In some instances traction upon this bone causes separation of the mental symphysis.

It is well known that among the plastic phenomena of labor is lessening of the occipito-frontal diameter by advance of the squamous portion of the occipital bone beneath the parietals, the movement being permitted by the hinge-like cartilage and fibrous tissue which at this period of development is present, uniting the previously mentioned part with the basilar portion. Should the forceps be applied to the forehead and occiput, this movement may be exaggerated, and injurious pressure upon the brain result. Not only this, but either with or without the forceps the two portions of bone may become separate, and the anterior inferior margin of the squamous part be forced against the medulla.

Little is to be said as to the treatment of these various injuries: some of them are incompatible with life, the child perishing from convulsions, it may be. Yet, on the other hand, an infant may survive some very serious injuries of the head. Thus, Dugès has mentioned an instance in which the child was born with the left eye almost completely outside the orbit, so greatly was the frontal bone depressed; yet the infant did not have convulsions or other grave symptoms.

By gentle and careful manipulation in suitable cases, the normal shape of the head may be restored, fragments of displaced bones being brought in apposition, and pressure upon the brain relieved.

The only injury of the neck which will be referred to is that involving the sterno-cleido-mastoid. Torticollis of obstetric origin has been attributed to injury of this muscle by one of the blades of the forceps. This may explain the condition in some cases, but does not do so in all, for children born head-last have been affected. It seems more probable, however, that, whether the forceps was used or the delivery was by the breech, the labor was difficult, great traction being necessary, this traction causing an injury to the muscle, rupture of some of its fibres, and a consequent hæmatoma. Others have regarded the injury as resulting in inflammation of the muscle. But, whatever the explanation, the characteristic condition present is a tumor situated just above the clavicle and in the muscle. As a rule, this tumor disappears spontaneously, though several weeks elapse before the event, and the function of the muscle is not permanently impaired. Active treatment is not indicated, though after the tumor has lost the sensitiveness it has at first, gentle friction and the application of a weak tincture of iodine may assist its disappearance.

Intracranial Injuries.—These are liable to occur in difficult deliveries, whether those deliveries are spontaneous, or either manual or instrumental. Rupture of the longitudinal sinus has been observed in some cases, and the hemorrhage results in death, though sometimes this may be delayed for one or even for two days after birth. Meningeal hemorrhage is a common cause of the child perishing during labor; according to Cruveilhier, it is the cause of death in one-third of the cases of children dying in this period. Should the child be born alive it may die from asphyxia soon after, but if respiration is fairly established the child may become comatose, have convulsions, usually unilateral, and die: if it escapes these dangers, it is liable to spastic hemiplegia. Sinkler[1] refers to the fact that in the cases of paralysis following difficult labors, spontaneous or artificial, the lesion is often an extravasation of blood over the motor convolutions, and states that if the quantity of effused blood is not great, recovery occurs. Osler[2] found in the records of the Philadelphia Infirmary for Nervous Diseases nine cases of palsy following delivery with the forceps; in some of the subjects there were scars caused by the instrument. McNutt[3] has reported ten cases of intracranial hemorrhage occurring in difficult or instrumental labors: it is remarkable that paralysis occurred in three of these, the delivery being pelvic, while it was absent in the seven others, the presentation being cranial.

Gowers[4] attributes great importance to difficult labor in causing cerebral palsy of the new-born. He states, "Of twenty-six well-marked cases of this affection, of which I have notes, the child was the first born in no less than sixteen, or at least three times as many as would have been without some causal relation to the fact. Of the remaining ten, the head was born last in no less than six. Thus, the labor was 'unnatural' in no less than twenty-two out of twenty-six cases. Of the remaining four, in three it was known to have presented special difficulty: in two, for instance, preceding children had died during birth in consequence of the difficulty." Lovett,[5] on the other hand, concludes from his statistics that the influence of difficult labor in producing cerebral paralysis must have been overestimated, stating that probably accounts of the labors from unprejudiced persons would show a much higher percentage of normal labors.

---

[1] Medical and Surgical Reporter, 1887.

[2] Philadelphia Medical News, 1888.

[3] American Journal of Obstetrics, 1885.

[4] Lancet, April, 1888.

[5] Boston Medical and Surgical Journal, June, 1888.

The essential characteristics of a cerebral paralysis in the new-born caused by labor are that there is no history of disease or injury happening after birth which can explain the condition, and that the paralysis gradually lessens.

There is little to be said as to the treatment of meningeal hemorrhage. Aspiration of the effused blood has been proposed, but it cannot be recommended. If convulsions occur, the potassic bromide and chloral may be given. But when the acute stage has passed—when effused blood, for example, has been absorbed or repair of injured brain-tissue accomplished—there is little to be hoped for from medicines, and, as remarked by Gowers, drugs are useless unless to combat some of the effects of the disease.

If there be associated with facial hemiplegia paralysis of the internal parts of the mouth, an internal injury of the nerve has occurred, and therapeutic means are without value.

Drs. W. J. Little,[1] Langdon Down,[2] and Arthur Mitchell,[3] among others, have apparently established a close connection between difficult labor and the idiocy of many of the children thus born; but the discussion of this subject cannot be presented here.

Injuries of the Trunk.—There will be omitted grave lesions of the spine, such as fractures of vertebræ and injuries of the cord, ruptures of internal organs, whether of chest or of abdomen, and intra-abdominal as well as intra-thoracic hemorrhages: a paraplegia in the new-born in almost all cases is the result of such serious harm that death soon comes: it can neither be averted nor delayed.

Muscles of the trunk may suffer such injury that a hæmatoma, similar to that described as occurring in the sterno-cleido-mastoid, may be present: its treatment is the same as that given for the affection previously mentioned.

In seventy-three cases of injury to the fœtus during delivery, collected by Ruge, the child having presented by the pelvis or podalic version having been performed, there were three instances of rupture of the sacro-iliac joint. It is possible that some cases of ankylosis of the joint result from such injury. Dr. W. H. Parish states that he has seen a tear of the perineum, extending from the vulvar orifice to the rectum, in a new-born, caused by the tip of one of the blades of the forceps which the practitioner had attempted to apply to an unrecognized breech-presentation.

Injuries of the Arms.—In connection with lesions of the superior members, those of the clavicle and scapula, which belong to the arms rather than to the trunk, will be referred to.

Fractures of the humerus are more frequent than all other fractures of the upper extremity and of the clavicle and scapula. The injury generally occurs in an effort to bring down an arm which has ascended in a head-last labor; the ascension is almost invariably the consequence of a hasty effort to extract the child, for if expulsion be left to natural forces the arms will remain folded upon the chest. Separation of the epiphysis of the head of the humerus from the diaphysis is an accident which may be overlooked, or thought to be a luxation, or a paralysis, from an injury to nerves. Küstner,[1] who has especially described this injury, states that its characteristic symptom is that when the infant attempts to move the arm the humerus rotates inward. In its treatment he advises that the epiphysis, now rotating outward, be brought in contact with the diaphysis, and then the arm fixed by a bandage in a position somewhat outward and backward to the thorax. Nancrede advises in the treatment of a fracture of the humerus fixing the whole upper extremity with a moulded splint in a straight position.

---

[1] London Obstetrical Society's Transactions, vol. xviii.

[2] Ibid., vol. iii.

[3] Medical Times, 1862–63.

Paralysis of the arm has been observed in connection with a hæmatoma of the sterno-cleido-mastoid, injury of the deltoid, compression of the axillary nerve, as from the employment of the finger or of the blunt-hook to effect extraction of the body when there is delay after the delivery of the head, and it has followed a shoulder-presentation, the arm protruding, delivery being finally accomplished by podalic version,—the want of power being independent of any cerebro-spinal lesion. Recovery is the rule in these cases. Delore believes that paralysis may result from rupture of a nerve-trunk near the spinal cord; if an upper member only be affected, the palsy, though incurable, does not interfere with the life of the child.

Gowers, in referring to paralysis of the arm, remarks[2] that the nerves of the arms may be damaged in several ways. "The injury may be associated with fracture of the humerus, and is then due either to the displacement of the broken ends of the bone, or to the force that caused the fracture. In such cases the distribution of the palsy is irregular, and varies in each instance. In other cases, however, the injury is higher up to the roots of the nerves as they enter the brachial plexus. This injury is commonly produced by pressure at one spot, in front of the edge of the trapezius. In a few instances the extremity of a much-curved blade of the forceps has pressed deeply here and has effected the injury, leaving at the same time a mark on the skin. In other and more frequent cases the injury is produced by the point of a traction-hook, or the tip of the bent finger, placed above the shoulder for this purpose."

Fracture of the clavicle is usually caused by direct pressure of one or two fingers upon the bone in the effort to deliver the head after pelvic presentation or after podalic version. The injury is treated by fixing the arm, the forearm being flexed, by means of a roller bandage, to the chest, and then properly supporting the member; the child should be as far as possible kept lying upon the back: the fracture is consolidated in six or seven days.

---

[1] Ueber die Verletzungen der Extremitäten des Kindes.

[2] Op. cit.

Separation of the clavicle from the sternum, transverse fracture of the scapula, rupture of the epiphysis of the neck of the bone, and injury of the acromion process are among rare lesions that have been observed in the new-born.

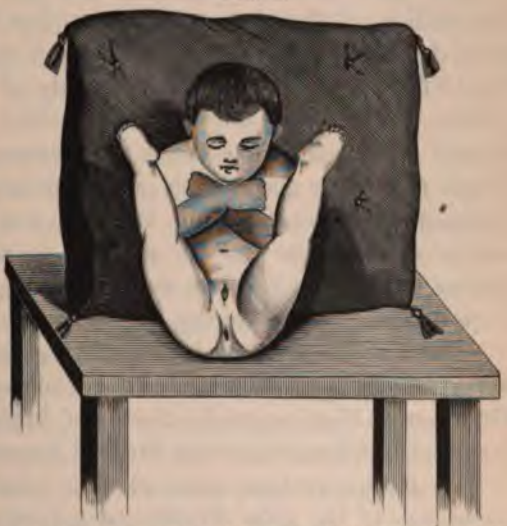
Injuries of the Lower Limbs.—A few instances of fracture of the femur occurring in spontaneous labor have been reported; but most frequently this injury has followed an effort to bring down the thigh in a case of pelvic presentation, where the presenting part was in the mother's pelvis, before pushing up that part so that room for the movement of the thigh could be given, or from traction upon the thigh by means of the fillet or of the blunt-hook.

Dr. Nancrede advises that sheet vulcanite should be used in the treatment of a fractured femur: the material is softened in hot water and accurately moulded to the limb. "An anterior splint should be made which will extend well up over the abdomen, and a posterior splint which will reach from the buttock well below the knee, thus fulfilling the important indication of fixing the joints above and below the fracture. It only requires ten or twelve days for firm union to occur."

Ruge states that dislocation of the hip in obstetric operations is exceedingly rare, he having not found one in three hundred autopsies of the new-born. Küstner, however, mentions that Goschen relates a case in which this injury occurring in birth was successfully treated in a girl thirteen years old by Langenbeck, and that Stromeyer had met with twenty cases. "The only possible way in which this dislocation could occur would be by sudden and violent force drawing down the limb, and then the head of the bone might be thrown upon the ilium."

An unusual position of the lower limbs is observed for several days after labor in that variety of pelvic presentation in which the thighs are flexed upon the abdomen and the legs extended upon the chest, described by French obstetricians as *présentation du siège décomplété, mode des fesses.* The limbs for a time remain in the same attitude which they occupied during pregnancy and in labor, and it is in vain to attempt placing them in any other. (*Fig. 6.*)

FIG. 6.



Position for some days after birth of lower limbs in child born as described in text.

INFANT-FEEDING—WEANING.

By T. M. ROTCH, M.D.

In reviewing the immense amount of literature which has accumulated on the subject of infant-feeding, we find that the superiority of breast-feeding is acknowledged so generally that it may be said to have become a scientific truth.

On the other hand, the opinions expressed regarding artificial feeding are so diverse, and so opposed to one another, that it is evident that much which has for years been taught must be unlearned, or rather admitted to be untrue, before we can expect to make any intelligent advance in this most difficult subject.

The great number of artificial foods, used by physicians according to the fashion of the day, only proves that bottle-feeding has not as yet arrived at that state of perfection where it can compete with breast-feeding. The difficulty in approaching the study of the subject has been in the method, which, with physicians as a class, has been too purely from a clinical stand-point. We know, for instance, how easily we may be misled by the apparently good effects of a medicament where perhaps on further investigation or in the light of some new discovery we learn that the improvement in the case was due not to the drug, but rather to circumstances entirely apart. The same applies equally well to the case of many foods and methods of feeding.

The analyses, the opinions involving expert chemical knowledge, and much valuable general advice, in the following pages, were received from Dr. Charles Harrington, Instructor of Materia Medica and Hygiene and Assistant in Chemistry in the Medical Department of Harvard University.

The feeding-problem is one which is hedged about with many difficulties, on account of the great diversity of individual circumstances and idiosyncrasies.

Certain infants, for instance, may thrive on peculiar mixtures not adapted to infants as a class. Many will not thrive on that food which nature has provided, and the well-being of an infant will depend much upon the circumstances by which it is surrounded, such as affluence or poverty, country or city life.

In those cases where, for one reason or another, human milk is not available, the question of feeding is this: What may be given to take the place of nature's food? In supplying a substitute we should copy in every possible way the physical and chemical characteristics of the food which is universally acknowledged to be the best.

In our endeavor to copy nature we may hope that, as our scientific knowledge advances, more and more light will be thrown upon those points which are now obscured by ignorance.

The recent discoveries in bacteriology, for instance, throw light on the reason for the old and well-deserved popularity of boiled milk in digestive disturbances, and plainly point out to us that had we in the past better understood the significance of the sterility of breast-milk, we should have made more rapid advances in the management of the infant's diet.

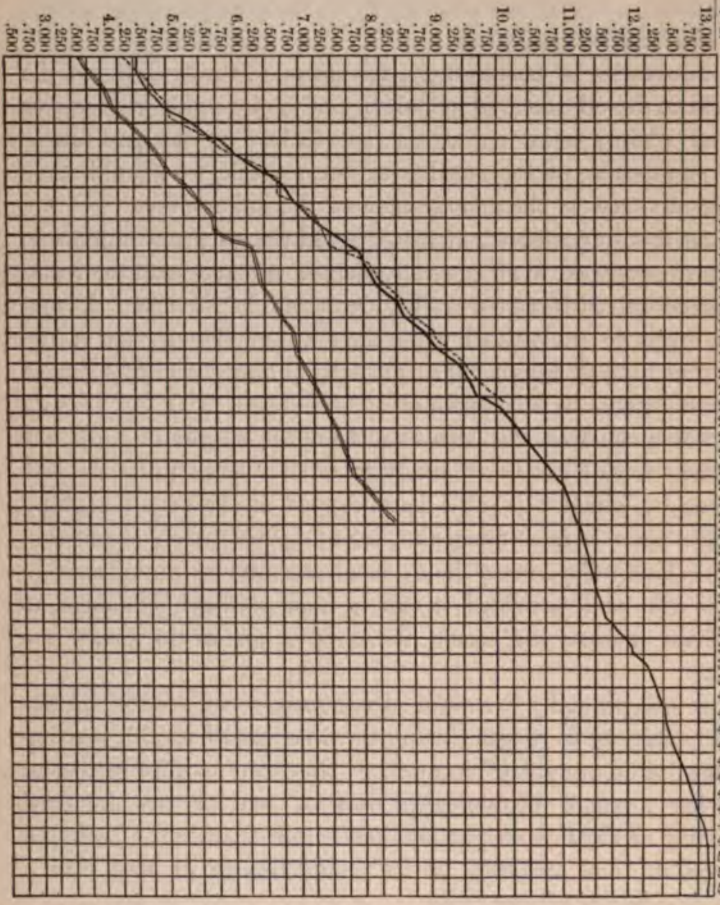
What is of the first importance is that we should recognize our ignorance, and, keeping our eyes opened to all possible scientific advancement, be ready to sweep aside preconceived ideas not resting upon established facts. Young animals at birth begin to receive their nourishment immediately, and a corresponding increase in their weight takes place from almost the first day of life. The human infant, in like manner, should begin with its nursing early, getting what it can from the breast until the full supply of milk has come. In this way it will not be so likely to have a large initial loss of weight to recover, by which it is often handicapped at the very beginning of its career, when there is most danger to be anticipated from a depression of its nutrition. Every day, every hour, is of the utmost importance in the early days of life, and, provided it can be done without detriment to the condition of the mother, the sooner the child is put to the breast the better it will be.

Under rather exceptionally favorable circumstances we see the breast-fed infant steadily gaining in weight during the first year of its life, starting with the average initial weight of from three thousand to four thousand grammes, showing a small physiological loss of one or two hundred grammes[1] according as the first weight is taken before or after the bath and passage of meconium, and attaining at the completion of its first year a weight of from nine to ten kilogrammes.

Instances of continual weekly gains during the first year occasionally come under our notice, and the following chart gives the exact weights of a healthy male infant fed by a wet-nurse for over a year, and will serve as an example of how an infant can thrive on good breast-milk: the analysis of this breast-milk is given on page 284, Analyses VIII., IX., X. The baby was evidently gaining so steadily that the weighing was omitted in certain weeks, which fact is unfortunate, as the weights would probably have shown the same steady gain. A weekly gain is also shown in the chart of a male and a female infant, brother and sister, nursed by their mother. The double line represents the boy's weights in the first twenty-nine weeks of his life, and the dotted line the girl's weights for twenty-one weeks. This continual increase in weight is of great importance in the first year, as it is the chief index by which we note the progress of nutrition and judge concerning the desirability of continuing the food. An average gain of from twenty to thirty grammes a day in the first four or five months and ten to fifteen grammes a day through the rest of the year makes a successful line of nutrition, and may be used as a working basis for the management of the food.

---

[1] C. W. Townsend, Bost. Med. and Surg. Jour., Feb. 17, 1887.



Name, F. S., Male.
Date Birth, June 20, 188
Initial W't, 4500 gram.

Weeks.	Actual Wt.	Date of W'g
1	4,500	June 27.
2	4,612	July 4.
3	4,916	" 11.
4	5,332	" 18.
5	5,684	" 25.
6	6,004	Aug. 1.
7	6,292	" 8.
8	6,644	" 15.
9	6,852	" 22.
10	7,172	" 29.
11	7,476	Sept. 5.
12	7,802	" 12.
13	7,994	" 19.
14	8,170	" 26.
15	8,362	Oct. 3.
16	8,586	" 10.
17	8,912	" 17.
18	9,136	" 24.
19	9,376	" 31.
20		
21	9,968	Nov. 14.
22	10,112	" 21.
23		
24		
25		
26	10,912	Dec. 20.
27		
28		
29		
30		
31		
32		
33		
34		
35	11,680	Feb. 20.
36	11,904	" 27.
37	12,032	March 5.
38		
39		
40		
41		
42	12,544	April 9.
43		
44		
45	12,640	April 30.
46		
47		
48		
49		
50		
51	13,104	June 10.
52	13,376	" 20.

Nature's feeding-apparatus is one which, by collapsing as it is emptied, avoids the formation of a vacuum, with its consequent exhaustion of the infant and prolongation of the nursing-time.

A healthy baby empties the breasts with easy and almost uninterrupted sucking in about fifteen minutes. The quantity ingested is determined by various methods, such as by careful weighing before and after nursing, and by the determination of the actual capacity of the average stomach at different ages and with different weights. These results are of great practical importance, and will be stated later when we come to speak of artificial foods.

The intervals of feeding constitute a very much more important factor in breast-feeding, where the quantity is regulated by the breast itself. It is sufficient for the present to state that the activity of growth in the stomach's capacity, according to Frolowsky,[1] can be represented by the ratio of one for the first week to two and one-half for the fourth week and three and one-fifth for the eighth week, while it is only three and one-third for the twelfth week, three and four-sevenths for the sixteenth week, and three and three-fifths for the twentieth week. The first month being the most critical period for the infant's nutrition, as it is the time when the equilibrium of its metabolism is being established and its chance for life the least, especial value should be attached to the series of careful investigations made at the Children's Hospital in St. Petersburg by Ssnitkin[2] to determine the amount of food which should be given in the first thirty days of life, and from which is deduced the rule, "the greater the weight the greater the gastric capacity." Ssnitkin's general results show also that one one-hundredth of the initial weight should be taken as the figure with which to begin the computation, and to this should be added one gramme for each day of life. The following table represents merely approximate average figures, which are the results of computations made by a number of observers in different parts of the world, and of my own investigations, both clinical and anatomical, during the past ten years.

TABLE I.

The average initial weight of infants is 3000–4000 grammes = about 6.6–8.8 pounds.

The average normal gain per day in the first five months is 20–30 grammes, or about two-thirds to one ounce.

General Rules for Feeding.

AGE.	INTERVALS OF FEEDING.	NUMBER OF FEEDINGS IN TWENTY-FOUR HOURS.	AVERAGE AMOUNT AT EACH FEEDING.	AVERAGE AMOUNT IN TWENTY-FOUR HOURS.
1st week . . .	2 hours.	10	1 ounce.	10 ounces.
1-6 weeks . .	2½ hours.	8	1½ to 2 ounces.	12 to 16 ounces.
6-12 weeks, and possibly to 5th or 6th month	3 hours.	6	3 to 4 ounces.	18 to 24 ounces.
At 6 mos. . .	3 hours.	6	6 ounces.	36 ounces.
At 10 mos. .	3 hours.	5	8 ounces.	40 ounces.

---

[1] Inaugural Diss., St. Petersburg, 1876.

[2] Reitz, Physiologie des Kindesalt., S. 40.

The weight, as well as the age, is necessary to determine the amount for each feeding in the individual infant, the rule being $\frac{1}{100}$ of the initial weight + 1 gramme for each day during the first month.

Illustrations of the above rule to serve as guides for especially difficult cases.

Initial weight.	Each Feeding.		
	Early days.	At 15 days.	At 30 days.
3000 grammes.	30 grammes (about 1 ounce).	30 + 15 = 45 grammes (about 1½ ounces).	30 + 30 = 60 grammes (about 2 ounces).
4500 grammes.	45 grammes (about 1½ ounces).	45 + 15 = 60 grammes (about 2 ounces).	45 + 30 = 75 grammes (about 2½ ounces).
6000 grammes.	60 grammes (about 2 ounces).	60 + 15 = 75 grammes (about 2½ ounces).	60 + 30 = 90 grammes (about 3 ounces).

The only point in the feeding-problem where artificial feeding seems to have the advantage of the breast is in the intervals of nursing. Irregularity in nursing, frequent nursing, and too prolonged intervals often so disturb the quality of the human breast-milk as to transform a perfectly good milk into one entirely unfitted for the infant's powers of digestion; while the element of intervals does not, of course, influence the question of chemical composition in a properly-prepared artificial food. Thus, too frequent nursing lessens the water and increases the total solids in human milk, making it resemble in a certain way condensed milk; while too prolonged intervals result in such a decrease of the total solids as to render an otherwise good milk too watery, and unfit for purposes of nutrition, however well it may be digested. The lesson that may be drawn from these facts is that some general rule for the feeding-intervals should be enforced, such as is represented in Table I., in order that the mother should neither interfere with the infant's digestion by nursing it too frequently and thus giving it a too concentrated food, nor, by neglecting to feed it often enough, interfere with its nutrition by giving it a too largely diluted food. We must recognize two distinct elements in infant-feeding, neither of which can with impunity be interfered with at the expense of the other, namely, digestion and nutrition, it being possible for the milk to be easily digested but non-nutritious, and again to be highly nutritious but difficult to digest and it is the equilibrium of these two elements which makes up a perfect infantile development.

The younger the infant, the greater the metabolic activity, and hence the greater need for frequent feeding; for nutriment is required not only f repair of waste, but also for the rapid proportionate growth; and we th see that the intervals of feeding according to the age as shown in Table become essential in successful feeding.

The next question to be considered is the quality of the food which is provided for the human infant. The later analyses and those upon which most reliance is to be placed are those of J. König, Forster, Meigs, Harrington, and others, and give the following approximate results:

TABLE II.

Human Milk.

Reaction	slightly alkaline
Specific gravity	1028–1034
Water	87–88
Total solids	13–12
Fat	3–4
Albuminoids	1–2
Sugar	7.0
Ash	0.2

Human milk has also been shown to be sterile by Escherich, who experimented with the milk of twenty-five healthy women, and found by keeping it in sterilized tubes that it remained unchanged for some weeks. We have as represented in Table II. a fair knowledge as to the normal composition of human milk, and are at once struck with its simplicity and freedom from a multiplicity of constituents. We must, however, allow that the chemistry of what is put down as albuminoids, which is a general name including casein and an albumen[1] which in its general features agrees with ordinary serum albumen, is too obscure to practically and clinically consider it more minutely. We recognize, however, that this albumen is present, in small and variable quantities, when the mammary gland and its secretion are in a normal condition, except at the time when the glandular function is being established, when it becomes proportionately larger in amount than the casein. It is also wiser, although we know that the ash is made up of a number of different salts, to deal with this constituent as a whole, for the analyses which have been made of the mineral constituents of human milk are so contradictory in their results that collectively they are of no value. On this point we know only that the ash is made up of certain salts amounting to about 0.20 per cent. of the milk; but we do not know the proportion of each compound present.

Reasoning from the strong analogy which must exist between human milk and cow's milk, and being aware of the great variations which occur in the latter, we may assume that human milk is liable to vary in its composition considerably with different milkings on the same day and also with the milking of the same hours on different days, so that we at present are not in a position to state that our knowledge of human milk is sufficiently exact to justify an attempt to formulate a table to show the composition of woman's milk at different ages, however valuable such information may in the future prove to be.

---

[1] Forster's Physiology, p. 563.

It is hardly within the scope of this article to discuss minutely the physiological question of the elimination of various elements by the mammary gland. The fact that such elimination does, however, take place is conceded, and at times becomes of a good deal of importance in the management of the infant's diet.

In the early days of the milk-secretion we find a decided difference in the character of its composition. From our knowledge of the colostrum period of cows, it would seem from analogy that the mammary gland, in the first five or six days, is in part at least an organ by which transudation from the blood can take place; that is, that the colostrum period is one where the mammary gland has not yet reached the perfect development of its function for producing milk from its own cells, and that the milk of this early period is very deficient in casein and proportionately rich in albumen. Under these conditions, and also where, as at times is the case, the milk is abnormal from some defect in the health of the mother, causing the colostrum period to be prolonged or to recur, there seems to be a direct transudation from the blood of such inorganic substances as arsenic, antimony, lead, iodide of potash, mercury, and others, taken by the mother. Well-authenticated cases also come to our notice from time to time where injury has been done to the nursing infant in this way, and where even death has occurred from the elimination by the breast-milk of certain organic substances, such as colchicum and morphine.

The greatest variety of substances have been found in the milk, but no definite rule as to the amount of this elimination has yet been established, so that our knowledge of the existence of this process is valuable as a prophylactic against harm, rather than as a means of direct benefit to the infant in disease, which latter point will not be discussed here, except to draw attention to the fact that the medicinal treatment of infantile disease through the breast-milk is exceedingly inexact.

We must also recognize the clinical fact that it is not only when the milk is in a poor condition that this elimination takes place, but that it may occur at any time during the nursing period in the breasts of women who, so far as we can ascertain, are in a perfectly healthy condition. Thus, every practitioner has at times doubtless observed the laxative effect on the infant of such drugs as the compound liquorice powder given to the mother; and a case has lately come to my notice where a baby vomited for weeks while taking the milk from the breast of its mother, who was unusually strong and well, but who was in the habit of drinking a considerable daily quantity of porter: the vomiting ceased at once and did not return after the porter was omitted.

That both the secretion and the character of the milk are strongly influenced by the nervous system is a matter of common clinical experience, but the exact nervous mechanism which controls it has not yet been fully worked out,—the clinical result, however, being recognized, that emotional mothers do not make good nurses. There are certain other facts known regarding the milk, which it will be well to mention here as having a bearing of more or less practical importance on what remains to be said concerning breast-feeding. Bunge's investigations on the comparison of tissues[1] show that the mammary gland abstracts from the blood just about the amounts of salts found in the tissues.

According to Forster,[2] "milk is the result of the activity of certain protoplasmic cells forming the epithelium of the mammary gland. So far as we know, the fat is formed in the cell through a metabolism of the protoplasm. Microscopically, the fat can be seen to be gathered in the epithelium-cell in the same way as in a fat-cell of the adipose tissue, and to be discharged into the channels of the gland either by a breaking up of the cells or by a contractile extrusion very similar to that which takes place when an amœba ejects its digested food. This observation is thoroughly supported by other facts. Thus, the quantity of fat present in the milk is largely and directly increased by proteid food, but not increased, on the contrary diminished, by fatty food. In fact, proteid food increases, and fatty food diminishes, the metabolism of the body. A bitch fed on meat for a given period gave off more fat in her milk than she could possibly have taken in her food, and that, too, while she was gaining in weight, so that she could not have supplied the mammary gland with fat at the expense of fat previously existing in her body. We also have evidence that the casein is, like the fat, formed in the gland itself. When milk is kept at 35° C. outside of the body, the casein is increased at the expense of the albumen. When the action of the cell is imperfect, as at the beginning and end of lactation, the albumen is in excess of the casein; but so long as the cell possesses its proper activity, the formation of casein becomes prominent. That the milk-sugar also is formed in and by the protoplasm of the cell is indicated by the fact that the sugar is not dependent on a carbohydrate food, and is maintained in abundance in the milk of carnivora, when these are fed exclusively on meat, as free as possible from any kind of sugar or glycogen. We thus have evidence in the mammary gland of the formation, by the direct metabolic activity of the secreting cell, of the representatives of the three great classes of food-stuffs, proteids, fats, and carbohydrates, out of the comprehensive substance protoplasm."

With the aid of such facts as have been stated above, we can now judge more intelligently as to the various questions which arise in connection with the subject of infant-feeding. The general rule deduced from these facts is, manifestly, that a healthy woman should nurse her child. The younger the infant the more important the breast-nursing, the gastro-intestinal canal being in a more active state of development and certain of its functions being still unprepared for use in the early months of life. It is much more difficult to adapt an artificial food to the sensitive growing infantile digestive apparatus at this early age, thus accounting in a measure for the rule, that the younger the child the greater the mortality. There is no doubt, however, that the mother's milk in a considerable number of cases met with in the practice of physicians among civilized nations appears to be entirely unfit for her offspring, and it at times becomes a question of considerable judgment as to whether the infant shall be withdrawn from its mother's breast either temporarily or entirely. It is here, in my opinion, that in the future the careful and repeated analysis of the milk will play a great *rôle* in aiding us to determine wisely this question.

---

[1] Archiv für Physiologie, 1886, 539.

[2] Physiology, p. 564.

I am fully convinced that a large number of infants are deprived of their natural food and placed on artificial foods on insufficient grounds. We thus assist to keep up the resulting high mortality figures; and I believe that these figures will be sensibly reduced when, in consequence of our taking a more enlightened view of the subject, we shall increase the number of infants who are fed from the breast during the first three or four months of life.

A particular reason, among many, for waiting at least four or five months before beginning with artificial feeding is presented by the fact that the stomach after a rapid growth has become by the fifth or sixth month a more perfect receptacle both as to size and as to function.

Among numerous instances of the same kind which have come to my notice, I might cite, by way of a simple illustration of weaning for insufficient reason, the case of an infant three months old, which recently was brought to me from a neighboring town to have its artificial food regulated. The history of this case was that its mother, a healthy primipara, about twenty-two years old, had nursed the infant for six weeks, during which time the infant was fretful, suffered much from colic, and never seemed satisfied. For these reasons, although there was a gain in weight and the napkins showed a fairly good digestion, it was by the advice of the attending physician weaned at once. On careful inquiry, I found that this infant had been nursed almost continuously night and day with intervals usually of only one hour, and it was evident that the frequent nursing had resulted in producing a concentrated milk, which the infant's gastro-intestinal canal was rebelling against, and the infant at six weeks of age was deprived of its supply of breast-milk in July, and placed upon an artificial food containing seventy-eight per cent. of starch, simply because the important factor of intervals had not been thought of as a means of improving the milk and relieving the pain and the apparent hunger.

On the other hand, the general health of the mother should be carefully investigated, as women suffering from constitutional syphilis or chronic consumption are manifestly unfit for nursing; and at the same time we should be careful, unless decided symptoms of disease are present, not to set aside the milk of a delicate-looking woman until it has been analyzed. The rapid progress which is being made in the detection of the bacillus tuberculosus not only in the sputum but also in the milk and in other secretions, may in the future be of much practical importance in the determination of the question whether a woman should nurse an infant; but the present state of our knowledge is only sufficiently advanced to allow us to state that this bacillus has been detected in the secretion of the mammary gland.

A case of considerable interest came to me in consultation early in July [1888], which points to the possibility of our being at times too hasty in our decision to deprive an infant of its mother's milk. The mother, a rather delicate primipara, twenty-five years of age, was delivered, July 3, of a boy seven pounds in weight. Within four hours puerperal convulsions set in, from which she recovered, but was left with albuminuria (0.25 per cent.) and casts. The latter disappeared in a few days, but the albumen, though somewhat diminished, continued; and the patient, though naturally of a calm disposition, was in a highly nervous condition, fearing that she could not nurse her baby, but decidedly opposed to having a wet-nurse. The milk appeared in considerable quantity on the fifth day, but the baby did not thrive, and, although it gained somewhat in weight, was very fretful, slept very little, and looked badly, so that the attending physician became alarmed, and, after treating it for its dyspepsia without much success until it was five weeks old, and finding that there was still about 0.25 per cent. of albumen in the mother's urine, decided with me that the breast-milk should be withheld until we could determine the cause of the trouble, and an analysis was accordingly made, with the following result:

ANALYSIS I.

Fat	1.62
Sugar	6.10
Ash	0.17
Albuminoids	3.54
Total solids	11.43
Water	88.57
	100.00

This analysis revealing the probability that the large amount of albuminoids was causing the disturbance of digestion, and that the small amount of fat was not sufficient for nutrition, the attending physician was very anxious to procure a wet-nurse; but, while we were endeavoring to get a proper one, we decided to empty the mother's breasts with the breast-pump[1] every day, thus relieving her from the worry of attempting to nurse her baby and of seeing it fail to gain, and thus also giving her undisturbed nights and a great deal of out-door life. The infant was in the mean time placed on an artificial diet, which was digested very well, and, as it ceased to cry, the mother's mind became tranquil, and the albumen in her urine in a few days was reduced to a trace. This treatment was carried out for a week, the milk continuing to flow freely, and an analysis was then made of the mother's milk and also of that of a healthy wet-nurse, whose own baby was thriving on her milk; the following results were obtained:

---

[1] During the past three months of June, July, and August [1888] I have had under my care a baby seven months old, who was dying of starvation, as I had been unable to prepare for it an artificial food which it could digest and thrive on. This infant also was totally unable to nurse from the breast; but the breast-milk of a wet-nurse that I procured agreed with it perfectly, and this nurse has pumped the milk from the breasts and fed the baby with it from a bottle for over three months with the greatest success, the infant thriving and now being in such a healthy condition that it is about to be weaned. This case shows the perhaps exceptional, but at times very great, value of the breast-pump.

	ANALYSIS II. *Mother.*	ANALYSIS III. *Wet-nurse.*
Fat	3.20	3.04
Sugar	6.40	6.60
Ash	0.18	0.12
Albuminoids	2.52	2.32
Total solids	12.30	12.08
Water	87.70	87.92
	100.00	100.00

The two milks being equally good, it was then decided to allow the infant to begin to take one nursing daily from its mother, although the albuminoids were still about one per cent. higher than the infant seemed likely to digest: it was consequently given to its mother, nursed well, seemed satisfied, digested its meal without trouble, and at six months is still being nursed.

However great may be the variation in the composition of animals' milk, and from our extended knowledge of the chemistry of cow's milk we know that this variation is a marked one, we must bear in mind that a far greater variation probably occurs in human milk. The physiological influence of the emotions on the nervous system, with its resulting changes in the mammary secretion, necessarily has a much wider range in the woman, subjected as she is to the worries and vicissitudes of civilized life, than in the animal carefully stabled and pastured. This variation in a mother's milk must be seriously considered, and the individual milk, rather than the general superiority of mother's milk, investigated in each case, if our feeding is to be successful. Instances not infrequently arise where such continual shocks are brought to bear upon the mother in her daily life, or where her own temperament is such an undisciplined one, that her milk, ordinarily good, becomes totally unfitted for her infant, and at times acts as a direct poison, with most disastrous results, so that the welfare of the infant in such cases unquestionably demands the change to a wet-nurse.

A nursing mother should be made to understand that these variations are liable to arise however good her general health may be, and that, while she is simply fulfilling a duty demanded by nature from those who bear children, her duty when once she has undertaken to nurse is to avoid as much as possible these variations, by regulating her life to a normal standard and avoiding excitement. Both of these requisites of a normal lactation come within the province of the physician to explain as he would any other branch of rational medicine, for many a mother by her course of life renders her milk unfit for the proper alimentation of her infant through ignorance of what seem to the physician but the simple dictates of common sense, and she will be only too thankful for advice on this subject. Instances of this arise where, as observed by Zukowski,[1] seasons of fasting, with their accompanying excitement of the emotions, have induced such an influence on the milk, the fat especially being decreased to as low as 0.88, that many of the nursing infants became sick and gave evidence of imperfect nutrition.

We must next consider the question of the variation in the milk which takes place from natural causes, such as the return of menstruation. Does such a return necessarily contra-indicate the continuation of nursing? As in all questions of this kind, we cannot adopt and follow an inflexible rule, but must be guided by what seems best for the individual case. Infants are at times affected so seriously by the alteration in the constituents of the milk which occurs once in four weeks, that their nutrition is markedly interfered with, and a change to a more stable food is indicated. Again, the only disturbance which may arise is a temporary and slight digestive attack for a day or two, which apparently does not materially affect the infant, and makes us hesitate to run the risk of depriving the infant of a food on which it thrives during twenty-six days out of twenty-eight. We must also not be too hasty in concluding from the bad symptoms in the infant that we should at once withdraw it permanently from the breast, for the catamenia may appear once and then not again for a number of months, the infant's powers of digestion in the mean time becoming so much more fully developed that they are unaffected by the catamenial milk. Even where the catamenia recur regularly, the disturbance which may have been marked at one period may for many reasons fail to recur at the next: so that the question is reduced to whether the composition of the milk shows a recovery of the equilibrium of its constituents within a few days, or remains affected to such a degree as to endanger the integrity of the infant's nutrition.

My own experience, so far as it goes, is in favor of allowing the infant to continue with the breast, unless it is decidedly contra-indicated by circumstances such as have just been mentioned.

I have seldom met with cases which could not without permanent injury be tided over the small amount of temporary digestive disturbance which is usually met with. Within a few days I have seen a case where the catamenia returning produced no effect whatever on the infant; and this is only an instance of what in all probability often occurs where mother and infant are at the time in an otherwise normal condition. There have, as yet, been too few analyses made during the catamenial period to justify us in drawing any definite conclusions as to the chemical status of the question; but the probability is that the milk will be found to be deficient in fat and to have its albuminoids increased, following the general rule of disturbed mammary secretion, and consequently in a condition to interfere temporarily with both digestion and nutrition.

---

[1] Jacobi, Intestinal Diseases, p. 4.

It may be of interest, from what has been said above concerning the variations in the milk which may arise from emotional causes and menstruation, to report the analyses of the milk of a mother and a wet-nurse where these influences appeared to produce certain chemical changes. The mother, a healthy but rather delicate primipara, the period of whose pregnancy had been supervised by me with the greatest care, and whose temperament was subject to extremes of despondency and excitement, was delivered in March, after a short and easy labor, of a healthy boy. She was exceedingly anxious to nurse her infant, but within a few hours after its birth she was seized with an uncontrollable fear that she would be unable to do so. In spite of all the assurances which could be given her to the contrary, and of the plentiful supply of milk which in due time came in the breasts, she remained in a very nervous, despondent condition. As the infant began to show decided signs of indigestion, I thought it best, before proceeding further, to investigate the composition of the milk. This resulted as follows, and plainly showed the necessity of not persisting further, as it was evidently much altered from unavoidable nervous conditions which seemed likely to recur through the whole of her lactation:

ANALYSIS IV.

Mother's Milk.

Fat	0.62
Sugar	5.80
Albuminoids	4.21
Ash	0.20
Total solids	10.83
Water	89.17
	100.00

Under these circumstances, although there was an abundant supply of milk, a healthy wet-nurse, whose baby was strong and thriving, was procured, and the infant immediately began to gain in weight and ceased to show any digestive disturbance. After a month, however, the infant was found not to have made its weekly gain, to be unusually restless, and to be having rather more frequent fæcal discharges than usual. It was then discovered that the wet-nurse was menstruating, and an analysis of her milk was made on the second day, resulting as follows:

ANALYSIS V.

Wet-Nurse.

CATAMENIAL MILK, SECOND DAY.

Fat	1.37
Sugar	6.10
Albuminoids	2.78
Ash	0.15
Total solids	10.40
Water	89.60
	100.00

The catamenia lasted only about four days, and did not return for some months. The infant after the first twenty-four hours showed no disturbance whatever, soon began to gain again, and was not affected by the subsequent recurrence of the catamenia. An analysis made one week after the catamenia had ceased showed a decided change for the better,—that is, increased fat, decreased albuminoids; and forty days later a still greater improvement, as was anticipated from the blooming condition of the infant.

ANALYSES.

Wet-Nurse.

	VI. SEVEN DAYS AFTER CATAMENIA.	VII. FORTY DAYS AFTER CATAMENIA.
Fat	2.02	2.74
Sugar	6.55	6.35
Albuminoids	2.12	0.98
Ash	0.15	0.14
Total solids	10.84	10.21
Water	89.16	89.79
	100.00	100.00

A much more serious state of affairs arises when the nursing mother becomes pregnant; for here the almost universal clinical experience is that the infant, for various reasons, cannot continue to be fed by its mother, it being unusual for a woman to have sufficient vitality to nourish properly her living child and growing fœtus. The danger of reflex miscarriage from the continual irritation of the mammary gland by nursing, I personally have had no experience with, but this is mentioned as one of the dangers contra-indicating the continuation of nursing by a pregnant woman. We must, however, here also not judge hastily, but take all the circumstances of the case into consideration before deciding on a measure of such vital importance to both child and fœtus. If the mother remains strong and vigorous and the analysis of her milk shows no deterioration, while the infant is a delicate one just beginning to thrive on its rightful supply of natural food, or if it is during a hot period of the year, and especially where a wet-nurse cannot be procured, it will often be wisest to take some risks and continue the nursing for a certain time, perhaps six or eight weeks, and then according to circumstances gradually substitute an artificial food. Almost every case will differ, in the questions to be decided, and must be judged on its own indications and contra-indications, always, however, recognizing the generally accepted rule that lactation and pregnancy are incompatible.

The food of the nursing woman is closely connected with the food which she provides for her infant. We have already spoken of the possibility of the elimination of various substances by the mammary gland, and we should impress upon nursing women the importance of a more carefully arranged regimen than when they are not nursing, and of a limited use of drugs.

Saline cathartics may at times not only act unfavorably on the infant, but very decidedly lessen the flow of milk, or even stop it altogether. Certain vegetables and fish will in some individuals cause discomfort to their infants. A plain mixed diet, with a moderate excess of fluid and albuminoids over what they are normally accustomed to, will, as a rule, give the best results.

According to what we have already said concerning the physiology of lactation, we should, in cases where the milk is found to be poor in fat, reduce slightly the amount of fatty food taken by the mother and increase the proteid elements. We should also be exceedingly careful in suddenly changing the customary diet of a healthy nursing woman on purely theoretical grounds. The mistake was made for many years of keeping women on too low a diet in the early period of lactation, with a consequent delay in the establishment of a sufficiently nutritious milk-supply, and a corresponding increased initial loss of weight in their infants. Where, however, we are especially likely to err is in permitting a healthy hard-working wet-nurse, accustomed to a somewhat coarse but nutritious diet, on entering a refined home to adopt totally different habits of exercise and an unaccustomed diet, rather than endeavoring to have her continue in her natural mode of life. This sudden change of life frequently results in ill health to the nurse, with its accompanying deterioration in the quality of her milk, or at least in so changing its quality as to make it an unfit food for her foster-child.

A notable instance of too radical a change of diet was brought to my notice about a year ago, by Dr. J. B. Swift, of Boston. A wet-nurse was procured for an infant seven days old, and her milk was digested well during two or three weeks, while she was fed on an abundance of good food and rich milk. The infant then began to vomit thick curds, identical in appearance and toughness with the curds of cow's milk; and an analysis, as shown by the following figures, presented the amount of total solids increased to a most marked degree, the percentage of albuminoids corresponding far more nearly to cow's milk than to woman's milk. The nurse was put on plainer food and skimmed milk, and the infant ceased to vomit. The infant and nurse then continued well and strong during the whole year, the infant making a weekly gain. The third set of figures gives the analysis of this milk in the twelfth month.

ANALYSES.

Wet-Nurse.

	VIII.	IX.	X.
	TWO DAYS BEFORE CHANGE OF FOOD.	RICH FOOD FOR A MONTH.	FOOD REGULATED AND MILK AGREEING WITH INFANT.
Fat	0.72	5.44	5.50
Sugar	6.75	6.25	6.60
Albuminoids	2.53	4.61	2.90
Ash	0.22	0.20	0.14
Total solids	10.22	16.50	15.14
Water	89.78	83.50	84.86
	100.00	100.00	100.00

This case will be of considerable interest later when we come to speak of albuminoids in the preparation of artificial foods.

I have little confidence in galactagogues, beyond proper food, exercise, and general hygiene, for their number betrays their inefficiency.

It not infrequently happens, especially among women of the upper classes and nursing women of all classes, when their general health is not in a perfectly normal condition, that the supply of milk is not sufficient to satisfy the infant, and the question arises whether the mother's milk shall be entirely given up or whether it shall be supplemented by some other food. My own individual experience, both in private and in hospital practice,—and, so far as I can ascertain, this experience agrees with that of other observers,—is in favor of assisting the mother to nurse her infant during the earlier months of its life. I have found that where the artificial food is carefully regulated by frequent analysis until the infant is making decided progress in its weight and general condition, this method of rearing infants is far superior to withdrawing the mother's milk and feeding the child exclusively upon artificial food.

We have, on the one hand, a much wider range for regulating the mother's milk, by increasing or diminishing the number of the artificial feedings, and on the other hand, if the mother's milk agrees with her infant, an excellent opportunity for making our artificial food correspond to what nature has provided. We are far better equipped to cope intelligently with the feeding-problem by this method than by any other which is known, excepting a continual change of wet-nurses until one is found whose milk both chemically and clinically fulfils the wants of the individual infant,—a proceeding which can rarely be carried out in the present status of parental prejudice, though it has in my hands at times proved to be eminently successful.

In regulating the administration of this mixed feeding we should allow ourselves to be guided as much as possible by known physiological laws; never, however, persistently following out these laws where the clinical result does not correspond to them, for there may be other physiological factors in the problem which as yet are beyond our knowledge.

It is a fact pretty widely acknowledged that the mother's milk, as a rule, is more likely to be suited to her infant's digestion than the milk of another woman; but here again we have as yet too few cases where direct investigation by means of chemical analysis of the two milks has been made, to lay down actually as a fact what we can merely grant as a supposition, that an idiosyncrasy in the mother's milk will find an analogue in her infant's digestive powers. The reverse of this proposition has also been held to be true, that at times some idiosyncrasy in the mother's milk will make it radically unfit for her infant. The probability, however, is either that analyses will show that these milks are poor ones or that we shall find that the infants have unusually weak digestive powers.

From what has already been said, we need hardly state that if for any reason a mother cannot nurse her child, the food which will of all others be most likely to give satisfactory results is that of a wet-nurse. The question as to whether a wet-nurse shall be employed is, however, one of serious import, and must in each individual instance be decided by giving full weight to all the many circumstances which are involved in the case. It is the duty of the physician fully to explain that a good nurse is far superior to any artificial method of feeding, while the reverse of this statement must always be kept in view, that a poor nurse, whether from temperament, or age, or general health, or quality of her milk, had better be set aside where the conditions are favorable for a successful artificial feeding. It is perhaps better that the nurse's milk should correspond in age somewhat nearly to that of the infant she is to suckle, but a difference of some months in age need not necessarily be a contra-indication, as we are not yet in a position to say definitely that the milk differs sufficiently in different months to be of vital importance in choosing a nurse. A feeble child will nurse more easily and probably have better care from a multipara than from a primipara. The preferable age of the nurse is between twenty and thirty years. Her other requisites are a condition of good health and a quiet temperament. It will save much trouble, and will often obviate the frequent necessity for changing, if we have before her engagement a chemical analysis of her milk; in fact, all the points which have been above referred to for a successful maternal nursing are of equal significance in the wet-nurse.

Quite a number of nursing women, especially those in the higher classes, find that at variable periods in the course of their year's lactation their milk begins to fail, and they are forced first to lessen the number of their nursings and then to wean entirely. The time, then, when the infant should be weaned almost always settles itself, without our intervention, at varying periods. The period of lactation, however, and the one which might be called physiologically normal, can, when the breast-milk remains of good quality and quantity, be carried through the first year with benefit. We have certain guides which aid us in determining the proper time for beginning to wean. Physiologically, we are told that certain functions, such as that which converts starch into glucose, are but slightly developed in the early months of life, and that they exist and are gradually being established during the first year, not, as a rule, being perfected and in a condition upon which we can call upon them with impunity until the last two or three months of the year. Another sign which aids us somewhat as an index by which we can judge of the progress of this functional development is the appearance of the teeth, calling our attention to the fact that nature is preparing a means for the infant to digest and assimilate a different form of food from that which it has so far received by sucking, the presence of six or eight incisors usually in the normally-developed infant corresponding to the full development of the pancreatic secretion.

Again, a most valuable index, which assures us that we need not be anxious to change the infant's food during the first year, is the continuous increase of weight, which, with the general blooming condition of the infant, represents a normal lactation. As in the case of all physiological rules, however, we must admit of certain variations which in the especial case are as important for the infant's welfare as the rule itself,—namely, the curtailing or lengthening of the period of lactation by a month or two, according to the season of the year, the irruption of the teeth, or the condition of the child (as in recovery from an illness), it being wiser to feed the infant from the breast during the heated portions of the year, and to wean in cool weather, either before or after the hot season, according to the individual circumstances of the case.

An interdental period also is preferable to a dental period, on account of the possible disturbances which may arise in the latter and interfere with the proper action of the new functions which are being called upon to perform their duties. Where there is any uncertainty as to the character of the milk which the infant is taking, especially in the latter months of lactation, a chemical analysis should be made at once, and repeated, with an interval of some days, several times; for the latter months, though not so difficult to manage intelligently as the early period of the infant's life, are much more likely to need careful supervision than the middle period, which, from its usually uninterrupted tranquillity, has been called the period of normal nutrition. Where the infant has, through an insufficient supply of milk in the mother, become for some time accustomed to several meals of artificial food daily, the matter of weaning becomes a very simple one, for we know that we have a food which will agree with it; but where we have to begin to wean directly and to adapt a food to the infant's digestive capabilities, as in cases of sudden failure of the milk or sickness in the mother, this procedure becomes much more intricate, and is at times fraught with considerable danger. It is in these cases that an analysis of the milk made when the mother was in good condition often proves to be of great assistance, for it is not a very difficult matter to make an artificial food which shall correspond to this analysis in its percentage of fat, albuminoids, sugar, total ash, and water.

Unless under very exceptional circumstances, sudden weaning is to be deprecated, though of course we must allow that it is often done with impunity. The safest method, so long as we can never judge beforehand what infants will be likely to be unfavorably affected by sudden weaning, is to take plenty of time, and gradually ascertain, perhaps by frequent changes, which form of food is best adapted to the case. We then gradually accustom it to this food, omitting one by one the breast-feedings, until finally we are sure that we have an artificial food on which the infant will thrive, with the proportion of starch, the new element which may now usually be introduced into the dietary, carefully adapted to its amylolytic function, which has but lately arrived at its full development, and which varies in different infants. When this change has been accomplished, the breast can with safety be entirely withdrawn.

The danger of injudicious weaning was strongly impressed upon me some years ago in a case which I watched for several days, through the courtesy of Dr. Sinclair, of Boston, and which it seems well to put on record. A rather delicate nursing infant, fourteen months old and backward in its development, having cut only four teeth and being in the process of cutting four more, was, without the advice of the physician, suddenly deprived of the plentiful supply of breast-milk of its healthy mother, in the latter part of November, and fed upon oatmeal gruel. Vomiting and prostration immediately began, and continued until the oatmeal was omitted and the breast resumed, when the infant again began to thrive. Three weeks later, the mother, through ignorance of the cause of the first attack, again weaned her infant suddenly, and again, without any preparation, fed it on oatmeal gruel. On the following two days the infant vomited incessantly and was much prostrated. The oatmeal was then changed to barley, and this again, as the vomiting continued, to Mellin's Food. The symptoms, however, grew worse, and the now thoroughly terrified mother again put the baby to her breast, with, however, this time a disastrous result, as her milk from nervous influences was so changed in its quality that it acted like a poison on the infant, which fell into a condition of collapse. Dr. Sinclair was sent for, and a few hours later I was consulting with him. A wet-nurse with a healthy four-months baby was immediately procured, and after several days of complete prostration the baby began to revive, and somewhat later was gradually weaned without trouble. It may be well to add, for the encouragement of those who may in their practice be so unfortunate as to have cases of this kind to deal with, that after the mother's milk had poisoned the infant, and when I first saw it, the skin was gray and cold, the fontanel sunken, and the eyes fixed, and yet recovery took place.

It would here seem not inappropriate, before entering upon the subject of artificial feeding, to speak somewhat more fully of the value of the chemical examination of the milk. From what has already been said, it will be seen that although such analyses enable us to work more intelligently, yet the conclusions which we can draw from them are far from being precise, owing to the extreme variations which take place at different times, and to the insufficient number of reliable analyses which have so far been made. We should, therefore, be extremely guarded in drawing conclusions for the present, merely looking upon our figures as approximate. On the other hand, it is of great importance that when reliable analyses are made they should be published, and thus, as our material increases, enable us in the future to arrive at what cannot but be important facts to aid us in the regulation of infant-feeding. Thus, we already are led to expect to find in the poor milks, which do not agree with the infant, an excess of albuminoids and a diminution of fat beyond what we have so far been able to determine as the normal average percentages of these two elements. Again, where a variation takes place in the milk, it is more likely to be found in the fat and albuminoids than in the sugar and total ash. We should also advise a number of analyses, rather than one, in order that the error of an especial and temporary variation may be corrected. The importance of and assistance which can be gained from these analyses are in my opinion very great, and many more analyses should be made than we are now in the habit of deeming necessary.

The question of expense should not for a moment be considered by those who can afford to have analyses made, for not only will real benefit come to their own children through money spent in this way, but these analyses, by being published and collated, will prove of great value for the proper regulation of the feeding of infants in all classes of society.

The mere microscopic examination of milk, beyond the determination of the presence or absence of colostrum-corpuscles, is too uncertain and misleading to be in any way depended on, the chemical analysis being the only practical method which can be recommended.

It was only lately that a physician skilled in the use of the microscope, in a neighboring town, sent me a specimen of woman's milk which he stated was rich in fat, but which Dr. Harrington's analysis showed to have only a little over one and one-half per cent. of this ingredient. The presence of an undue amount of yellow coloring-matter is also at times very misleading.

An error which, however, we must always allow may interfere with the true analysis of the milk which the infant has actually received into its stomach at the end of the nursing, and which must necessarily invalidate the reasoning from our analyses, is what I have already referred to in speaking of the changes which from slight causes may arise and influence the especial specimen which is being analyzed. Thus, we should recognize that the milk varies considerably in its percentage of fat and total solids in the different periods of a milking, and that the composition of the milk which the infant has in its stomach may differ very widely from the composition of a specimen taken directly before or after the nursing.

Harrington's analysis of the three portions of a milking will illustrate the meaning of what has just been said.[1]

TABLE III.

	FAT.	TOTAL SOLIDS.	WATER.	ASH.
"Fore milk"	3.88	13.34	86.66	0.85
"Middle milk"	6.74	15.40	84.60	0.81
"Strippings"	8.12	17.13	82.87	0.82

The experiments and analyses of J. Reiset and Peligot[2] also are of considerable interest, as showing not only the increase of solids at the end of a nursing, but also that this increase is mostly of the fat, and to a lesser degree of the albuminoids, and also, as I have already stated, that a short interval of nursing increases the solid constituents in proportion to the water, the reverse of this being found to be true where the intervals are long.

---

[1] Harrington, 8th Annual Report Mass. State Board of Health, 1884, p. 189.

[2] Hermann, Handbuch der Physiologie, Bd. V., Theil I. S. 404.

Heidenhain explains this physiological phenomenon by saying that his investigations point towards the fact that during the pauses between the milkings the cells of the glands are growing and a proportionately small amount of solids and a proportionately large amount of water are secreted, while the irritation of milking causes increased activity of the milk-cells, with a corresponding increase in the solid secretion and lessening of the water. Thus, Peligot's table giving the analysis of an ass's milk in three different portions shows the relations of the solids both to the water and to one another.

TABLE IV.

Ass's Milk (*Peligot*).

	1st Portion.	2d Portion.	3d Portion.
Butter	0.96	1.02	1.52
Milk-sugar	6.50	6.48	6.50
Casein	1.76	1.95	2.95

His second table shows the changes of proportion according to the intervals of the nursing.

TABLE V.

Ass's Milk (*Peligot*).

	Milking-Intervals.		
	1½ Hours.	6 Hours.	24 Hours.
Butter	1.55	1.40	1.23
Sugar	6.65	6.40	6.33
Casein	3.46	1.55	1.01

The next table is also interesting, and should be recorded.

TABLE VI.

Cow's Milk (*Reiset*).

Time since Last Milking.	Percentage of Solids at	
	Beginning.	End.
12 hours	9.33	16.04
6 hours	12.80	16.06
2½ hours	12.84	13.08

Harrington's analyses of woman's milk showing the "strippings" of a two-hours interval and the "fore-milk" of a twelve-hours interval are also of considerable interest.

TABLE VII.

	"Strippings," 2 Hours' Interval.	"Fore-Milk," 12 Hours' Interval.
Total solids	15.32	10.14
Water	84.68	89.86
	100.00	100.00

With these chemical and physiological facts before us, we are forced to acknowledge that we must be very circumspect in the conclusions which we deduce from such analyses of human milk as up to the present time have been made. An error in these conclusions, where a correct chemical analysis has been made, is less likely to occur from the sugar and ash than from the albuminoids and fat, and is most likely of all in both frequency and degree in the latter.

In the preceding pages ample recognition has been given to, and great stress has been laid upon, the importance of feeding infants during the early months of life by means of human milk. We know, however, that in civilized communities the necessity of supplying the infant with food not from the human breast will often arise, and will in all probability be a demand which will increase rather than decrease as our civilization advances. With this prospect before us, and appreciating the difficulties which in a large number of cases are liable to arise when we attempt to adapt an artificial food to the wants of an infant, it manifestly becomes a duty to endeavor to reduce the high mortality figures induced by artificial feeding. With this purpose in view, we should carefully investigate the different methods of feeding and adopt some more uniform plan for starting human beings in life; for diversity and not uniformity is now the rule. With the exception of the very small proportionate percentage of inherited diseases which occur at birth, this diversity of method in feeding is the most prolific source of disease in early infancy. The group of symptoms which for want of a better name is represented by dyspepsia, difficult digestion, occurs most frequently in the three periods when the infant's digestion is likely to be tampered with,—namely, in the early weeks of life, when experiments are being made to determine what food will be best to start with; next, when, in addition to the irritation arising from the beginning of dentition, new articles of diet are added to the original food; and, thirdly, at the time of weaning, when there is often a sudden and entire change in the character of the food. The proper management of the first of these periods is of the greatest comparative importance, because it is the time when, as before stated, the stomach is in its most active period of growth, and when the function of digestion is being established, and, following the rule of functional establishment, is in a state of unstable equilibrium. This demands the most careful regulation of the bulk of the food given to make it correspond to the rapid increase in the gastric capacity. We thus avoid the danger of overtaxing this capacity by too great volume in the beginning of nutrition, at the same time providing the sensitive developing function with the proper materials for nutrition, and thus avoiding by prophylaxis the dyspepsia of the later periods of infancy and childhood, the seeds of which are continually being sown in this early transitional period. We therefore have not only the question of infantile digestion but also that of infantile development to deal with. We should recognize the fact that the problem of artificial feeding is not a simple one, and that we cannot too often reiterate that the question which but too commonly is supposed to be a simple one, and the one which in the great majority of cases is alone considered, namely, "Which food shall we give to the infant?" is a misleading and insufficient one. The problem is a combination of factors of which the kind of food is only one, and I personally have long been convinced that the neglect to investigate thoroughly and carry out in detail the combination of these by no means insignificant general factors has had much to do with our failures with artificial feeding in the past. If this fact be more uniformly insisted on in the future, it will prove to be of great value in the reduction of the mortality figures in the first two years of life. It would seem also that the present is a most opportune time for raising a note of warning against allowing our enthusiasm over any one especial theory in the feeding-problem to warp our better judgment. There will surely in the future be a reaction which will relegate to its proper place every theory which, because of being lately advanced, happens to enjoy an undue amount of credit, and at the same time is actually doing harm by keeping in the background other theories which, each in its own sphere as a significant part of a complete whole, may be of very great importance in the successful solution of the general problem. An error of oversight of one-eighth in a mathematical problem is not so great as one of one-fourth, but nevertheless the attention to, and correcting of, the greater error, will not prevent the neglect of the smaller from completely destroying a correct result. Until lately it has been the quality of the food which has been monopolizing, in my opinion to comparatively too great a degree, the attention and brains of the medical profession; to-day it is sterilization which in feeding has become prominent, representing a faint picture of the great advances in rational medicine which it is hoped are to follow from the undoubtedly brilliant discoveries which have been made in bacteriology. Already one of the latest German writers[1] on artificial feeding has stated that the physiology and pathology of infantile digestion depend not on the chemical but on the biological character of the food, and, if we are not on our guard, this tendency to exaggeration will spread, and by its influence will blind us to much good work which in other directions has already been done, and which we cannot afford to ignore. Not that I would for a moment be understood to underrate the value of sterilizing an infant's food, for it has for years proved of very great benefit in my practice and that of others to have the food thoroughly boiled before giving to the infant, but I predict that just so much as we enhance the value of this one important part of the whole at the expense of others which possibly may be proved to be of less individual importance, just so much further shall we be from an intelligent comprehension of the subject.

To feed an infant one month old with six ounces of acid cow's milk every four hours, no matter how thoroughly such a mixture has been sterilized, would be a radical offence against well-known anatomical and physiological laws. It therefore seems to me that time will be well spent in the discussion of the subject of artificial feeding, if we investigate and endeavor to copy each in its turn the various devices which nature makes use of, for we must admit that we are not in a position to try to improve on nature's method.

---

[1] Hochstetter, Allgemein. Wien. Med. Zeit., No. 15, 1888.

It is certainly wiser and more economical not to spare expense and trouble in arranging the infant's diet, for, as has been explained above, the period of active growth of an organ is the time when its function is readily weakened, and when once weakened the digestive function is a prolific source of annoyance and expense in childhood and adolescence. Cheap foods and cheap methods of feeding, unless they are the best that can be procured, should not be tolerated more, and in fact not nearly so much, in the early feeding of infants than in adult life; we often, however, see a food recommended for a young infant because it is cheap and easily prepared, and yet when its well-known lack of nutritive ingredients would with adults at once stamp it as unfit for use.

In discussing the treatment of disease we advocate what is best, without reference to what it costs, and then, in the especial case where expense is an element which has to be taken into consideration, we endeavor to adapt our treatment to these considerations, but always approaching as nearly as possible to our first standard. In like manner I believe that we are doing wrong to the public if we allow ourselves to be handicapped in such a difficult question as infant-feeding by the cry of expense. Infant-feeding is an expense which is vital to the welfare of the human race, and we can, without being accused of extravagance, safely relegate to the province of the manufacturers of patent foods the recommending to the public of foods which if judged by the amount that is offered in bulk are cheap, but which when judged by their nutritive properties are extremely expensive.

Our scientific knowledge and clinical investigations have not yet enabled us to follow nature exactly, and we therefore have not yet obtained an ideal method of artificial feeding. We must, nevertheless, go as far as the present state of our knowledge will allow, thus gaining a little ground every year; and we must be especially careful not to be led astray by the fictitiously brilliant results which are reported from time to time in favor of certain foods. Instances are continually occurring where one food will fail and another when substituted for it succeed; and yet these successes are merely temporary, and the mortality always remains far above that from human breast-milk.

In nature's method of feeding we have first a receptacle, the human breast, which provides a fresh supply of food at proper intervals, absolutely prevents fermentation of the food before it enters the infant's mouth, incites to action the necessary digestive fluids, avoids a vacuum by collapsing as it is gradually emptied, thus allowing the food to flow continuously, and finally is practically self-regulating as to the amount of daily food according to the infant's age. Secondly, the food itself is adapted to the infant's digestive function, and for its development, by its temperature, 98° to 100° F., its alkaline reaction, and its chemical constituents. Given these factors, how nearly can we approach them artificially? Human ingenuity has not yet been able to devise anything which approaches the perfection of nature's receptacle, and the best that we can do to offset this complex mechanism is to adopt that which is exactly the reverse,—namely, a receptacle of absolute simplicity,—and thus combat the tendency to fermentation by preventing, through perfect cleanliness, the receptacle from becoming a source of fermentation.

To illustrate to my class of medical students my ideas as to a proper receptacle for the sterilization and administration of an artificial food, I had made in the spring of 1887 what are practically enlarged test-tubes, which, being open-mouthed beyond what is usually provided in the ordinary nursing-bottle, and having no angles, are readily cleaned. I also devised at the same time, and for the same purpose, a simple sterilizer for household use, which can be made at any tinsmith's at small expense. The rubber nipple takes the place of that of the breast, and a small hole near the end of the feeding-tube prevents a vacuum being formed and regulates the rapidity of the flow, while it allows it to be continuous; this is done by rolling up the edge of the rubber nipple from the hole with the finger, or letting it cover the hole, according to the demand shown by the infant. The artificial receptacle is not self-regulating, and hence we must determine anatomically the amount of food in bulk which nature provides for the average infant at different ages, and from these average figures deduce the proper amount for the especial infant. The feeding-tubes are graduated for the more important periods of growth, for the purpose of continually impressing upon the mother and nurse what the physician often only has the opportunity of telling them at the beginning of the nursing period,—namely, that the error is in giving too much food rather than too little, an error also which naturally results when, as is commonly the case, the usual eight-ounce nursing-bottle is provided as the receptacle at the very beginning of infantile life.

FIG. 1.

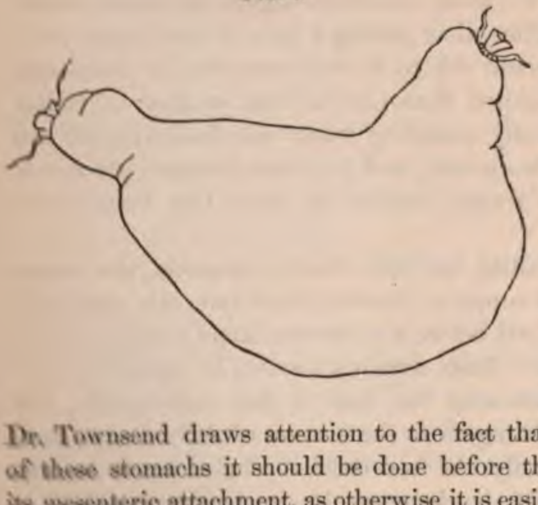
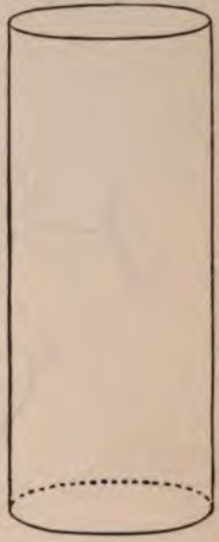


Fig. 1 represents the stomach of an infant five days old, in life-size, which was prepared for me by Dr. C. W. Townsend, of Boston. It was found to hold twenty-five cubic centimetres, and Dr. Townsend draws attention to the fact that in measuring the capacity of these stomachs it should be done before the stomach is separated from its mesenteric attachment, as otherwise it is easily stretched by the introduced fluid, so as to show a greater capacity than would be possible during life.

Fig. 2 represents the actual size of the tube, which is sufficiently large for each feeding during the first week; and when we consider the space which would be needed to represent the full-sized nursing-bottle, these two diagrams express better than can be explained by words the disproportion between the size of the infant's stomach and the amount which the mother supposes it should hold to keep her child from being starved.

FIG. 2.



Referring again to Frolowsky's investigations, already mentioned on page 273, we see that there is a very rapid increase in the gastric capacity in the first two months of life, while in the third, fourth, and fifth months the increase is slight. Guided by these data, which we find correspond closely with the results of clinical investigations bearing on this point, we should rapidly increase the quantity of the food in the first six or eight weeks, and then give the same quantity up to the fifth or sixth month, unless the infant's appetite evidently demands more, when of course a gradual increase should be made. A considerable increase in the quantity needed, also, usually takes place between the sixth and tenth months.

Of the different causes which regulate the gastric capacity, the weight of the infant has the greatest influence, and it is perfectly possible for a poorly-developed infant of small weight to have a gastric capacity no greater than a normally-developed infant of half the age. This possibility must be taken into account when we attempt to regulate the bulk of an artificial food to the age of the infant. An infant of six weeks has been brought to me, whose general development and weight corresponded so closely to those of the normal average infant of twelve weeks that it was self-evident that the two ounces of food which would ordinarily have been the proper allowance, so far as its age was concerned, was not sufficient, and that its weight indicated a gastric capacity for an allowance of four ounces, which in fact it took and digested with the greatest ease, while with any amount less than the four ounces it was never satisfied.

Fig. 3 represents, in life size, the stomach of a female infant twelve months old. This infant's weight was 4289 grammes, which corresponded to about the weight of the average normal infant at two months. The gastric capacity, 120 cc. (4 ounces), corresponded to the weight rather than to the age.

Another very important influence on the gastric capacity is the kind of nourishment which the infant has received.[1] The breast-fed infant in the early months of life has a uniformly-developed stomach, and, as a rule, of smaller capacity than the stomach of the artificially fed,—the muscular fibres of the fundus in the latter stomach being weak and its form abnormal.

---

[1] Fleischmann, Die Ernährung des Saüglingsalters, p. 17.

It is common in the artificially fed, where the quality of the food is poor and the quantity too large for the age and development, and where rachitis has been a consequence, to find the stomach dilated to a capacity entirely out of proportion to the infant's age and weight.

Fig. 3.

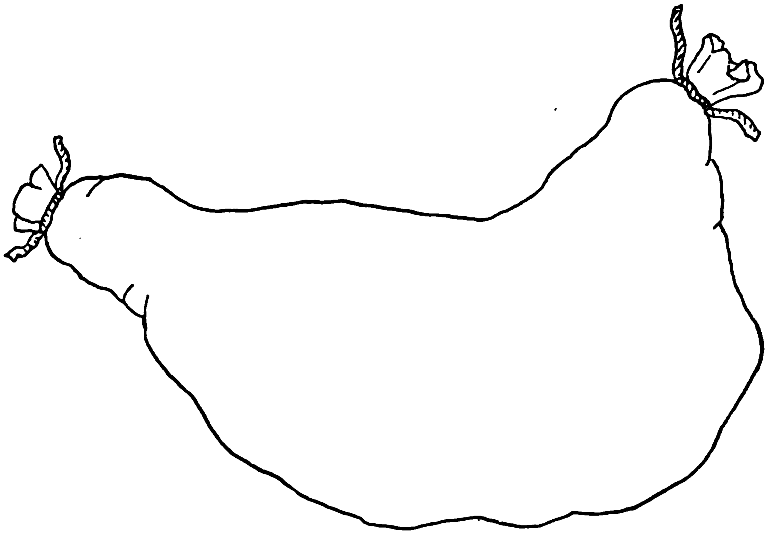


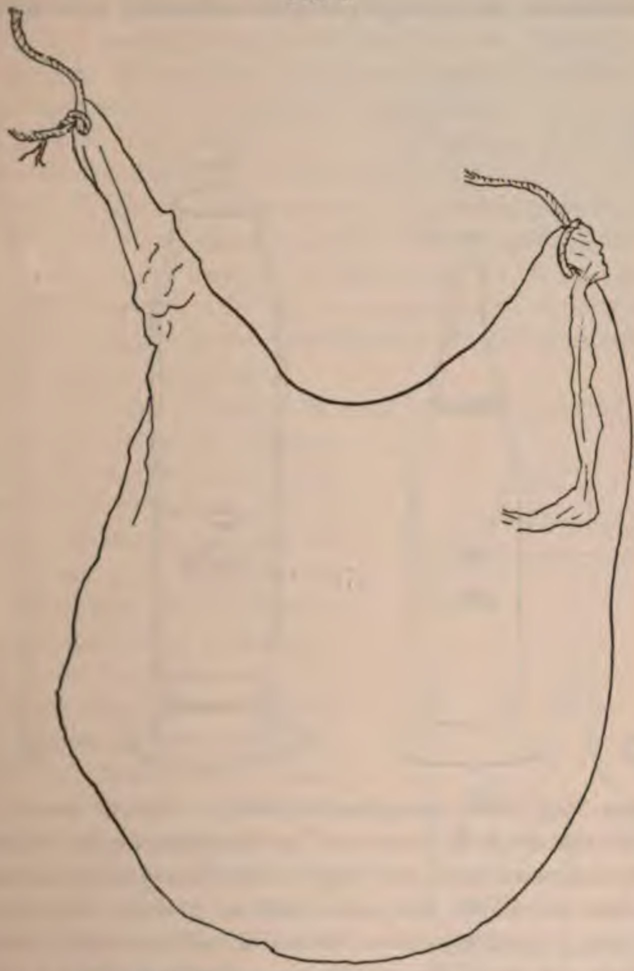
Fig. 4 represents, in life-size, the dilated stomach of an artificially fed, rachitic infant seven months old. The gastric capacity in this case was 300 cc. (10 ounces), corresponding to the average infant of twelve months, and the shape of the dilated stomach, with its very much increased greater curvature and its lessened smaller curvature, is very significant.

Allowing, then, that owing to the distensibility of the stomach these measurements of the gastric capacity are only approximate, we can nevertheless see from the above anatomical diagrams, which have been prepared with great care, that the figures in Table I. provide us with a fair working basis by which we can determine the amount of food to be given at different ages so as to correspond to the marked periods of the stomach's growth. Figs. 5, 6, 7, and 8 represent feeding-tubes drawn on a scale of about one-third, and have the proper capacity for the amount of food which should be given to the infant during these periods.

Fig. 5 is a tube of small calibre, graduated to hold two ounces, and intended to be used for feeding during the first six weeks of life and later as a measure for the larger tubes in preparing the artificial food in its varying proportions.

The smaller tube, Fig. 6, holds four ounces, has a calibre of one and five-eighths inches, and a height of six inches; it is to be used from the sixth week to the fifth or sixth month, and is intended to correspond to the above-described rapid growth of the stomach in the first two months and its insignificant further increase in size up to the fifth or sixth month;

FIG. 4.



it is represented in the diagram with the nipple adjusted for use. The large tube, Fig. 8, has a calibre of one and six-eighths inches, a height of eight and three-fourths inches, and corresponds to the common half-pint nursing-bottle; it is represented in the figure without the nipple, and shows the air-hole, which, together with the mouth of the tube, is stopped with cotton. Another medium-sized tube has been made to go with the set, and this has a calibre of one and six-eighths inches, a height of seven and three-fourths inches, and holds six ounces. It of course is not a necessity, but is intended to be used between the sixth and tenth months, merely to enunciate the importance of careful supervision of quantity throughout the first year, as, where a food qualitatively correct is being used, the error, as a rule, is in giving too great an amount. It is represented in Fig. 7.

A few words regarding the process of sterilization and the connection of bacteriology with the feeding-problem will here be necessary, as explanatory of what will be said later about especial artificial foods. The practical utility of destroying the developed bacteria in the milk in certain intestinal disorders has long been recognized clinically. Jacobi many years ago recommended that the milk to be used for the infant during the day should be boiled as soon as received, and kept in tightly-stoppered, inverted bottles on ice. Lister has shown that cow's milk as it comes from the udder is sterile, and that it quickly becomes infected in various ways, as by the hands of the milkers, the air of the stables, etc. Professor Soxhlet, of Munich, found that calves one week old, when taken from the udder and fed with their mother's milk from a trough, were affected with diarrhœa, which disappeared on their being fed again directly from the udder. Soxhlet's experiments also showed that under the same conditions of temperature the milk of three cows as ordinarily milked turned sour in about half the time that the same milk did when the udders and milkers' hands were carefully washed and other precautions for cleanliness were taken before the milking.

FIG. 5.

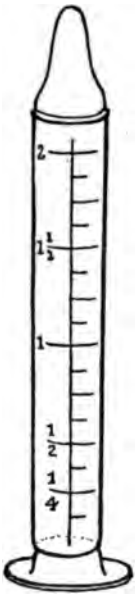


FIG. 6.

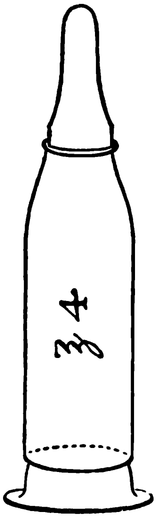


FIG. 7.

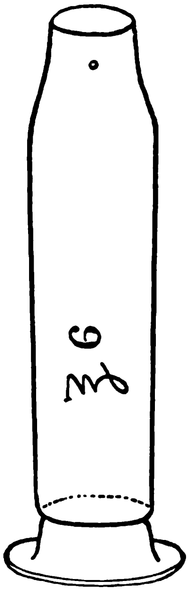
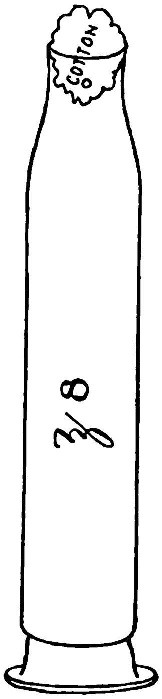


FIG. 8.

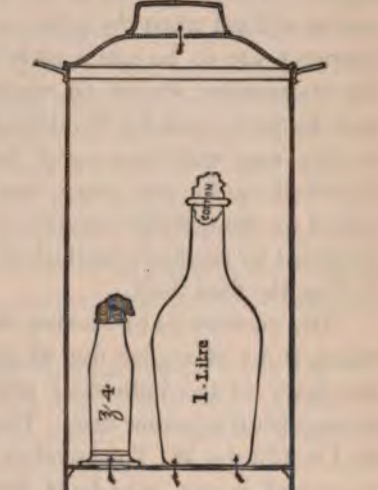


After the true significance of sterilization had been explained by various bacteriologists, Professor Soxhlet undertook a series of experiments to determine the length of time milk would remain sweet after sterilization. His method was to immerse the milk, contained in stoppered bottles, in boiling water; and he found that thirty to forty minutes of this immersion practically sterilized the milk and enabled it to be kept sweet for varying intervals of time according to the variations in the process of the sterilization. Soxhlet also devised an apparatus for sterilizing and for feeding, and this apparatus is already being used by a large number of families in Munich. His feeding-arrangements are, however, clinically imperfect and unsatisfactory, his use of the long rubber feeding-tube being especially objectionable, and much of his apparatus unnecessary.

Soxhlet published the results of his investigations in the *Münchener Medicinische Wochenschrift*, Nos. 15–16, April, 1886.

Dr. Harold C. Ernst, Demonstrator of Bacteriology in the Harvard Medical School, had already been successfully feeding sick infants on sterilized food as early as the summer of 1884, when in the spring of 1887, attention having been drawn by him to the subject of sterilization, I was led to devise the feeding-tubes for sterilization already described, and the steamer represented in section below.

FIG. 9.



A paper which embodied my work on this subject was read by me at the May, 1887, meeting of the Obstetrical Society of Boston, and was published in the *Archives of Pediatrics*, August, 1887, Soxhlet's article at that time not having been heard of by me. My idea was that if sterilization was necessary or advisable we should adopt as simple a method as possible for family use. This was in a measure accomplished by sterilizing in a receptacle from which the infant could be directly fed, without pouring from one vessel to another and running the risk of fresh infection.

Following Dr. Ernst's advice, I sterilized by exposure to steam for twenty minutes, and thus destroyed the developed bacteria. A single steaming, however, may not destroy the spores, which may be developed later unless the process is several times repeated.

For purposes of feeding, the first sterilization or killing of the germs is in all probability all that is practically necessary.

Fig. 9 represents the sterilizer, with the water boiling under the litre flask, which is stoppered with cotton, and the four-ounce feeding-tube, with its mouth, its nipple, and its air-hole tightly enclosed in the rubber cot, as described on the following page. A gas-flame is preferable to that of an alcohol lamp. This steamer answers very well for sterilization such as is necessary in infant-feeding. It is simply a tin pail eight or nine inches in diameter and nineteen to twenty inches deep, raised on three legs sufficiently high to allow a Bunsen burner to stand under it. Four inches from the bottom of the cylinder is a perforated tin diaphragm, on which the feeding-tubes stand while being sterilized. There is a small vent for the escape of the steam in the cover. Water is placed in the bottom of the steamer to the depth of about an inch, and in about ten minutes after lighting the gas-jet the water begins to boil. The food is then poured into one of the feeding-tubes, and an ordinary rubber nipple adjusted as on any nursing-bottle. Over the nipple, as an extra precaution for the exclusion of contamination, a non-perforated rubber cot is drawn tightly down on the tube. As soon as the water has been boiling for a minute or two, the tube is placed in the steamer, the cover applied, and the steaming continued for twenty minutes. The tube can then be removed, allowed to cool until of a proper temperature, 98° to 100° F., and on removing the rubber cot and putting the nipple in the infant's mouth the food is received as sterile as from the human breast, so far as the developed bacteria are concerned. Food sterilized in this way can be kept for a number of days, and can be utilized when the infant is to be taken on a journey. Where longer journeys are to be taken, such as an ocean voyage or across the continent, the sterilization should be repeated several times with intervals of a day, and the flasks used for the sterilization hermetically sealed. Food prepared in this way will keep sweet for many weeks. Where the steamer just described can for any reason not be obtained, a simple colander with a lid, placed on the teakettle, makes an effective sterilizer. The food can also be sterilized by Soxhlet's method of immersion in boiling water, or by directly boiling the food itself.

The question as to whether the milk should be boiled or steamed is one which is not of a great deal of significance, and can be settled according to the fancy of the individual practitioner, the object of sterilization being accomplished in either case. The remarks on this subject, however, made by Dr. Charles W. Townsend at the June, 1887, meeting of the Obstetrical Society of Boston, may be of interest to those who are using boiled milk as a food for infants. Dr. Townsend said,—

"The process of boiling milk of course sterilizes it, destroying not only the putrefactive bacteria and disease-germs which may be present, but, according to Newton and Wallace,[1] changing and driving off tyrotoxicon, one of the poisonous ptomaines produced by these bacteria. This takes place at a temperature of 180° F. Boiling, however, brings about other changes than those caused by sterilization by steam. The odor and taste of boiled milk are decidedly different from the odor and taste of raw milk. Boiling expels about three per cent. of gases, CO_2, N, and O. As the boiled milk cools in contact with the air, a scum forms, which is the albumen coagulable by heat, entangling in its meshes a certain amount of fat. This coagulable albumen, according to Winter Blythe, equals one-fifth of the casein in amount.

---

[1] Phila. Med. News, Sept. 25, 1886.

"Boiled milk does not become sour as quickly as raw milk, due, of course, largely to the fact of its sterilization.

"As to the effect boiling has on the digestibility of milk, there is a difference of opinion. There is a general impression that boiled milk is more constipating than new milk, which is probably derived from the fact that milk sterilized by boiling is less likely than raw milk to cause diarrhœa.

"According to Schreiner[1] and to Randolph,[2] boiled milk is more quickly coagulated by acids than raw milk, while the reverse is the case with regard to the action of rennet. Thus, raw milk at the body-temperature coagulates firmly almost immediately on the addition of a neutral rennet solution, whereas boiled milk under the same conditions does not clot for a far longer period, and the coagula are not so firm.

"The investigations concerning the relative digestibility of raw milk and boiled milk, found by siphoning out the contents of the stomach, are so contrary in their results that they need not be mentioned here. Randolph[2] and Reichman[3] came to exactly opposite conclusions, and Dujardin-Beaumetz[4] disagrees with both of these writers, saying that there is no difference. He, however, refers to Pinard as saying 'that prolonged boiling peptonizes a part of the albuminoid substances contained in the milk.'"

My own experiments comparing steamed with boiled milk show that the odor and taste of boiled milk are present when milk is steamed, but to a much less degree than with boiled milk; also that while a thick scum is formed on milk boiled for twenty minutes, which is tenacious and does not disappear on shaking, only a very thin scum forms on milk steamed for twenty minutes, and this is not tenacious, and almost entirely disappears on shaking.

Soxhlet did not make any direct bacteriological study of the subject, but simply took the curdling of the milk as a sign of infection. Measured by this standard, his milk kept about three weeks. According, however, to Jeffries, milk becomes full of bacteria and seriously altered before curdling occurs, and certain species of bacteria do not curdle milk.

Dr. J. Amory Jeffries, of Boston, published in the *American Journal of the Medical Sciences* for May, 1888, a paper entitled "The Sterilization of Milk and Food for Infants." Dr. Jeffries very aptly remarks "that it is a curious fact that, while older people are chiefly fed on sterilized food,—that is, cooked food,—infants are fed on food peculiarly adapted by its composition and fluid state to offer a home for bacteria." Jeffries's work is more distinctly bacteriological than Soxhlet's; agar-agar cultures were made

---

[1] Loc. cit.

[2] Phila. Med. News, June 21, 1884.

[3] Zeitschr. für Klin. Med., 1885, ix. 565.

[4] Boston Med. and Surg. Journ., Aug. 5, 1886.

[illegible] different fluids were sterilized, and the colonies of [illegible] results, however, coincide with those of previous [illegible] that steaming for fifteen minutes is sufficient to kill [illegible] while a second steaming is necessary for complete [illegible] and twenty lots of milk steamed but once, [illegible] distinct signs of change within a month, while [illegible] steamed twice did not change at all.

[illegible] also show that spores develop slowly and indeed [illegible] as he says, is an excellent medium for growth, [illegible] bacteria, like seeding among higher plants, is [illegible] growth. He also explains the preservation of [illegible] but once by the absence of any enduring spores [illegible] article of very great interest and value to the prac- [illegible] Bacteria of the Alimentary Canal" in the *Boston* [illegible], September 6, 1888, Jeffries has reviewed the [illegible] bacteriologists up to the present time, and, as [illegible] a direct bearing on the subject of feeding, I have [illegible] extensively from his article, and acknowledge [illegible] for the assistance which I have received from [illegible]

[illegible] and Escherich have shown that living bacteria are [illegible] stomachs of man and animals, and the former author [illegible] bacteria can pass through the stomach into the [illegible] a considerable time." Jeffries goes on to say, "Of [illegible] of the forms found in the stomach, little is [illegible] one, and the species have not been sufficiently [illegible] to recognize them with certainty. Miller has [illegible] give off carbonic dioxide and hydrogen gas, lactic, [illegible] being formed." "Of the flora of the intestines [illegible] of that of the stomach. The researches of Brie- [illegible] Escherich[4] have now proved that a large number [illegible] the faeces. Brieger isolated two new kinds, one a [illegible] grape- or cane-sugar into ethylalcohol, with a [illegible] other the well-known Brieger's bacillus. This spe- [illegible] in vast numbers, ferments sugars, and decomposes [illegible] ten species from the faeces, six of these also [illegible]. Of these, some produced acid fermentations [illegible], they were not sufficiently studied to show their [illegible] Escherich studied especially the faeces of infants, [illegible] of kinds of bacilli,—among them a small bacillus

---

[illegible] Chem., 1884, p. 306.

[illegible], 1884, p. 492.

[illegible] Congress f. innere Medicin. 1884.

[illegible] Darmbacterien des Säuglings und ihre Beziehungen zur Physiologie [illegible] Verdauung. 1886.

capable of converting milk-sugar into lactic acid, carbonic dioxide and hydrogen gas being evolved, either in the presence or absence of air,—a facultative anaërobic species, his *Bacillus lactis aërogenes*. . . . Escherich established, by the examination of a large series of cases, the fact that the kinds occurring in the fæces vary with the food, that is, the intestinal contents." Jeffries again says, "Starting at birth with the sterile meconium, consisting of mucus, epithelium, and the like, infection by the mouth and rectum quickly occurs, and in a short time almost any form may be found, but chiefly such putrefying forms as *Proteus vulgaris*.

"With the suckling of the infant and the substitution of the refuse of the milk and secretion of the digestive tract for the meconium, a sharp transition occurs. Instead of the generally distributed forms causing decomposition, only two kinds are regularly found,—*Bacillus lactis aërogenes* and Brieger's bacillus, the first chiefly in the upper parts of the intestine, the second in the lower parts. Passing on to the period of mixed diet, quite a number of forms appear, among them the *Streptococcus coli gracilis*, the putrefying green fluorescing, a tetrad coccus, and several kinds of yeast. This brings us to the pith of the subject: why are the flora so limited in the milk-eating infants and so diverse in others? What drives the forms found in the meconium out? That they can live there is clear, as shown by their presence the day before. Again, what prevents forms so common with meat diet from gaining a footing? It is not the milk alone, for milk is an almost universal food for bacteria, and all the kinds found in the intestines thrive in it.

"Escherich answers as follows: the *Bacillus lactis aërogenes* and the milk diet keep out the other forms.

"Formerly, even before the nature of ferments and putrefactive processes were clearly understood, the significance of this question was seen. The chyme is a mass admirably adapted for putrefaction or fermentation, yet ordinarily but little of either occurs. It is an alkaline or, as in the milk-fed, acid mixture rich in albumens, fats, and the starch group, amply provided with water and warmth. Such a mixture outside the body at an equal temperature would quickly decompose. It was generally held that some preservative action was exerted by the digestive juices: Bidder's and Schmidt's dogs with biliary fistulæ were held to explain the whole. These dogs, deprived of their bile, became emaciated, and suffered from diarrhœa and decomposition of the intestinal contents. Thus it seemed clear that in the absence of the bile decomposition occurred; that is, that the bile was a powerful germicide or germ-inhibitor. During the last few years, however, different results have been obtained in cases of biliary fistula. Röhmann's[1] dogs did not suffer from diarrhœa or putrefaction in the intestines: hence it is clear that the bile is not the cause of prevention. The diarrhœa, if present, is due to the large amount of fat passed on to the lower intestines.

---

[1] Röhmann, Beobachtungen an Hunden mit Gallenfistel, Breslau, 1882.

"Maly[1] and Emich[2] ascribed value to the bile acids, especially the taurocholic, basing their results on crude methods; and Lindenberger,[3] really leaving the subject, attributed the action to the organic acids in combination with the bile.

"All this argument and belief in the decided germicidal action of the bile occurred in the face of the well-known fact that bile itself will decompose.

"From a bacteriological stand-point, Miller has shown that a ten-per-cent. solution of bile, if anything, favors growth. Macfadyen has studied bile, bile salts, and bile acids in varying strengths. The only positive results were got with the acids, these arresting development of bacteria if sufficiently strong, especially taurocholic acid. Neither acid had much effect, and least of all on the forms causing putrefaction. *Proteus vulgaris* was only arrested by a strength of from one to two per cent. The pathogenic forms were arrested by a much smaller quantity, from one to one-half per mille.

"It is thus clear that other causes must be sought for. One of these is to be found in the lack of oxygen in the intestines, as pointed out by Escherich and strangely forgotten by others. There is certainly very little free oxygen in the chyme, if any; not only is it scarce in the food at the start, but is taken up by the chemical changes during digestion, and also by the intestines. This clearly must be a potent factor, for the majority of bacteria require a fair supply. Accordingly, many bacteria are found in the fæces which will not grow in the air, as shortly stated by Macfadyen, and the mass of those isolated in the air are able to grow without it.

"This apparent contradiction, the absence of oxygen in the intestines, and the presence of both aërobic and anaërobic bacteria, is probably explained by the ability of the aërobic kinds to draw oxygen from oxyhæmoglobin. They thus breathe through the intestines, as it were, when in close contact with the walls, while the anaërobic kinds live in the mass of the chyme, and do not, so far as we know, reduce oxyhæmoglobin.

"Escherich, though he points out the absence of oxygen, does not seem to give it full value, or rather forgets the subject in treating of the action of his *lactic acid bacillus*. As before stated, this form is regularly found in great numbers in the upper part of the intestines of milk-fed children. Here it converts a considerable part of the milk-sugar into lactic acid (Baginsky[4] says acetic acid, but has given no proof), and thus prevents the other forms from growing, most forms being susceptible to an acid reaction, and especially to the organic acids. The action of salicylic acid is known to all, and recent experiments, of which Macfadyen's (the last) are the best, show acetic, butyric, and lactic acids to be efficient germ-inhibitors in strengths of from one to one-half per mille according to the species.

---

[1] Maly, [illegible] logie, v. 184.

[2] Er[illegible] Akad. d. Wissenschaft, Wien, 1882.

[3] [illegible] llingar, 1884.

[4] [illegible] aschrift, 1888, Nos. 20 and 21.

"In milk-fed infants another point is the comparative inability of bacteria to attack casein, so that the bacteria are literally starved.

"To sum up, we may conclude that the bile acids, lack of oxygen, lack of suitable albumens, and the presence of organic acids are the causes of immunity from the putrefying and fermenting kinds of bacteria to which we are exposed. Certain forms are probably limited by the lack of water, that is, fluid state, doing poorly if unable to swim freely about. It must not, however, be supposed that bacteria are scarce in the intestines; on the contrary, they form a large part of the dry substance of the fæces.

"The ferments act by the production of various acids, chiefly from the milk-sugar. In small amounts, as in the case of *Bacillus lactis aërogenes*, the acid seems to be of benefit, and certainly does no harm, as it regularly occurs in healthy breast-fed infants. In large amounts, however, it must tend to over-acidify the contents of the intestines and interfere with the action of the digestive fluids."

The remaining factor of the feeding-problem—formulated on page 293, and which has not yet been considered—is the chemical constituents of the artificial food: what shall they be? Taking the average human breast-milk as the safest standard, we are impressed with the fact that the helpless young of all animals and of human beings are carnivorous; also, that although a vegetable diet would often seem far the easiest method of procuring nourishment for young infants, yet nature has persisted in providing an animal diet. We should therefore be very cautious about endeavoring to introduce into our artificial diet a vegetable element, which, as judged by our standard, must be a foreign element. Milk is the food which our reason tells us should be given to the young infant, and a milk which will approach as nearly as possible to the average human milk. That of various animals has from time to time been recommended as the best substitute for human milk, the recommendation being based on their analyses approaching more or less nearly the composition of human milk. The milk, however, of all animals has to be modified to correspond to human milk; and when we begin to modify, it is as easy to change the proportions of the different constituents to a great degree as to a small. The fact that one animal's milk approaches in its analysis more nearly to human milk than another is not of much significance, other considerations being far more important; and it is most important of all that we should use one which can be obtained everywhere all over the world by the people at large. This at once settles the question that it is the milk of the cow to which we must turn our attention. Cow's milk may differ in its composition from human milk to a greater degree than does the milk of the ass or the mare, whose milk approaches, so far as is shown by analyses, most nearly of all animals to that of woman; but this in all probability is for the very reason that cow's milk is so universally used as a food for human beings of all ages.

If the ass and the mare should become domesticated as a food-supply to the same extent that the cow has been, there is every reason to suppose that their milk might change in its composition and their comparatively undeveloped mammary glands increase in size, just as has been the case with the cow, an animal which for thousands of years has been used for the production of milk, and which probably did not in the beginning give such an over-production of the mammary secretion as is the case now. In fact, in Egypt,[1] where formerly there was either no trade in milk or very little, we find represented on the monuments cows with only slightly-developed udders, while the generative organs of the male animals are clearly depicted,—a fact of some significance when we remember the well-known tendency of the Egyptians to realistic representations. It is, then, from the public demand and by breeding that cows have been made to produce so much more milk than is necessary for the support of their young. Not only quantitative but qualitative differences exist in animals according to the development of their mammary glands; and, as Martiny[2] has shown in his collection of statistics on this subject, the condition which determines the quantity and quality of the milk depends on the development of the organ which produces it. The question of artificial feeding, then, is practically reduced to some modification of cow's milk, for this is the milk which is most easily procured everywhere, and, as the milk of all animals must be modified for the human infant, it is as easy to deal with cow's milk as with any other.

A further exemplification that cow's milk is practically the universal source of the artificial food-supply for infants in most civilized communities, is the fact that the various foods, patent or not, all depend for their basis on cow's milk, and that without this addition of milk they would show but an insignificant percentage of many of the most important ingredients of the food: so that logically we should not speak of the various foods as such, but merely as adjuvants to cow's milk, for if this is thoroughly understood it will, in many minds, do away with much misapprehension regarding the apparently successful results of innumerable foods which, in reality, when given to the infant, are merely a means of modifying the almost universal representative of the artificial foods, cow's milk. Cow's milk, therefore, should be carefully compared with the standard human milk, in order that we should know how nearly it resembles it; and Table VIII. is a comparison of the average human milk and the average cow's milk, the figures representing the later and more reliable analyses.

TABLE VIII.

	WOMAN'S MILK, DIRECTLY FROM THE BREAST.	COW'S MILK AS ORDINARILY RECEIVED, ABOUT TWENTY-FOUR HOURS OLD.
Reaction	Slightly alkaline	Slightly acid.
Coagulable albuminoids	Small proportionately	Large proportionately.

---

[1] Stumpf, Deutsches Archiv für Klinisch. Medicin, Jan. 18, 1882.

[2] B. Martiny, Die Milch, ihr Wesen und ihre Verwerthung, Danzig, 1872.

TABLE VIII.—*Continued.*

	WOMAN'S MILK DIRECTLY FROM THE BREAST.	COW'S MILK AS ORDINARILY RECEIVED, ABOUT TWENTY-FOUR HOURS OLD.
Coagulation by acids	Not perceptible in test-tube	Marked in test-tube; greatest with pure milk; less with milk diluted with water, and when 1 to 5 is not perceptible.
Water	87–88	86–87
Total solids	12–13	13–14
Fat	4	4
Albuminoids	1	4
Milk-sugar	7	4.5
Ash	0.2	0.7
Bacteria	Not present	Present.

We must, however, recognize that infants in general, as represented by those who live in cities and large towns, do not receive their supply of milk at once from the cow's udder, but that the milk, as a rule, is about twenty-four hours old, and it is, therefore, cow's milk twenty-four hours old that, until further improvement is made in delivering milk, we must compare with fresh human milk and modify to correspond to it.

From this comparison we at once see that human milk and cow's milk differ as markedly from each other in their chemistry as they do in their clinical results as foods; and, as practically we must use cow's milk in artificial feeding, our wisest course is to modify it until we have approached the chemistry of human milk as closely as possible. There is no doubt, however, that cow's milk unmodified has at times proved to be clinically successful in the rearing of infants. In parts of France, notably in Brittany, the infants among the peasantry are put directly to the cow's udder, and with apparently good results.

I know of a strong, healthy woman, twenty-four years old, who is one of a family of eight, all of whom in their infancy never tasted any milk but that which they received from their parents' cow, sucking it directly from the udder. The great weight of evidence, however, is that the average infant in the early months of its life does not digest unmodified cow's milk when introduced by the usual methods.

The exceptional instances where it is tolerated have their counterparts in the success of many other foods diverse in their composition, and only serve to prove that human digestion can at times be tampered with without much apparent immediate injury, and to emphasize the general rule, that the chemistry of the food which will produce the best average result should be the chemistry of human milk.

I am aware that Escherich[1] has announced that he has successfully fed a baby ten weeks old on unmodified cow's milk one quart per day, sterilized; but it is only necessary to refer to Analyses VIII., IX., and X. on page 284, to show that sterilization alone is not sufficient for success, which statement indeed is merely an every-day experience of those who clinically have much to do with the management of the infant's food. The milk in the above-mentioned Analyses VIII., IX., and X., being directly from the breast, was certainly sterile. It will be noticed that the chief difference in IX. and X. is in the albuminoids. The infant was doing perfectly well with the albuminoids at 2.53. The nurse was fed on a rich diet, and the infant vomited curds which, so far as could be judged, were identical in size and toughness with the thick curd of undigested cow's milk. The analysis then showed the albuminoids 4.61, corresponding to the percentage of cow's rather than woman's milk. The vomiting continued until the woman was put on a less nutritious diet, when the vomiting ceased, and the infant continued to thrive on the milk with its albuminoids reduced to 2.90.

---

[1] Jahrb. für Kinderheilk., Oct. 1887.

Before speaking of the various modifications of cow's milk which are necessary to make it correspond to human milk, it will be well to say a few words about its properties, as represented in Table VIII.

The reaction is stated to be slightly acid; and this is the case whether it has stood twenty-four hours with ordinary care or whether it is tested directly from the udder. This I have determined by direct experiment: so that practically the same amount of modification will be correct for the first twenty-four or thirty-six hours, so far as the reaction is concerned.

Of the total nitrogenous constituents of the milk which are classed under the general term of albuminoids, and of which the casein and the albumen are parts, the coagulable albuminoids are proportionately larger in amount in cow's milk than in human milk, so that under the same conditions a larger curd will be formed with the former than with the latter.

In conjunction with Dr. Harrington and Dr. Townsend, I have recently made some careful experiments as to the relative coagulability by acids of woman's milk, cow's milk, and cow's milk diluted with lime-water and barley-water in various proportions. The coagulation by rennet was not found to be a satisfactory or reliable test.

Table IX. gives the results of these experiments, which may prove to be of considerable value.

TABLE IX.

Equal volumes of fluid in test-tubes. Ten drops of acetic acid added to each test-tube. Each test-tube inverted slowly three times, so as to insure thorough, equal, and uniform mixing in all.

1.	Woman's milk		No curd perceptible to the eye.
2.	Cow's milk raw		Large curds.
3.	Cow's milk boiled		Same as 2.
4.	Cow's milk sterilized by steam		Same as 2.
5.	Cow's milk	2 parts	Finer than 2.
	Water	1 part	
6.	Cow's milk	2 parts	Same as 5.
	Lime-water	1 part	
7.	Cow's milk	2 parts	Slightly finer than 5 and 6.
	Water	1⅛ parts	

TABLE IX.—*Continued.*

8.	Cow's milk	2 parts	Same as 7.
	Barley-water	1 part	
9.	Cow's milk	1 part	Finer than 7 and 8.
	Water	4 parts	
10.	Cow's milk	1 part	A very fine curd; finer than 9.
	Cream	2 parts	
	Solution of milk-sugar, 18 drachms to the pint	3 parts	
	Lime-water, 1 Water, 3	2 parts	
11.	Cow's milk	1 part	Same as 1; no curd perceptible to the eye.
	Water	5 parts	

When a few drops of mercuric nitrate solution were added to woman's milk and to cow's milk diluted 1 to 5, a fine coagulum was produced in the woman's milk and a still finer one in the cow's milk.

There was found to be practically no difference as to the rapidity of the coagulation of the different fluids.

Cow's milk taken directly from the udder was found to coagulate in just as large curds as when twenty-four hours old.

It is thus seen that there is no difference in the coagulation of raw, boiled, or steamed milk; also that practically the size of the curd depends on the dilution of the albuminoids, rather than on any especial property belonging to the substance with which the dilution is made. With lime-water the result is the same as with water in equal amount, and barley-water only shows a fractional difference from the results with plain water.

The fat, so far as we know, is both in amount and in quality the same in both milks.

The albuminoids, as shown in the table, are four times as great in amount in cow's milk as in woman's, while the milk-sugar holds the relation of 7 in woman's milk to 4.5 in cow's milk. The ash, on the contrary, is in woman's milk only 0.2, while in cow's milk it is 0.7.

In cow's milk as commonly used for food we must recognize the presence of bacteria.

The question is now reduced to the different methods employed in modifying cow's milk. This may be done by diluting it with water, by concentrating it and then diluting it when used, or it may be modified by the various patent foods or by any other adjuvant, such as barley-water, lime-water, or cream.

In order to ascertain the correctness of the statement so often made that "attenuants act mechanically by getting as it were between the particles of the coagulum during coagulation and thus preventing their running together and forming a large compact mass," I have lately experimented as follows with various substances.

In each of six test-tubes of equal calibre, and containing 5 c c. of hot water, 10 c c. of milk were placed. In test-tubes II., III., IV., V., and

VI. were added equal portions respectively of Mellin's Food, Robinson's Barley, Imperial Granum, cracker-crumbs, and bread-crumbs. The albuminoids were then coagulated as before with acetic acid, and the following results were obtained:

Test-tube I. hot water and milk,—finest curd of all.
" II. " " " " and Mellin's Food,—not so fine as I.
" III. " " " " " Robinson's Barley,—about like II.
" IV. " " " " " Imperial Granum,—not so fine as II. and III.
" V. " " " " " cracker-crumbs,—not so fine as IV.
" VI. " " " " " bread-crumbs,—not so fine as V.

There is no doubt that where no attenuant was added the curd looked decidedly finer, while where attenuants were used there was not a great deal of difference between the substances employed, except the possibly rather larger curd according as the attenuant contained a larger percentage of starch.

We may conclude, then, until something more definite is known concerning this rather theoretical method of treating the curd, that dilution with plain water is the most practical and efficient means at our command.

Table X. has been prepared to show the analysis of the different modifications as they are given to the infant, and serve as a reference table to the physician, who by this means can readily see how near to or far from the standard human milk he is getting when he decides to use one of these modifications in feeding.

TABLE X.

Comparison of Woman's Milk with Cow's Milk and Cow's Milk modified.

(The figures are approximate and represent general averages.)

	REACTION.	STARCH.	WATER.	TOTAL SOLIDS.	FAT.	ALBUMINOIDS.	SUGAR.	ASH.
Woman's milk	Slightly alkaline.	0	88	12	3–4	1–2	7	0.1–2
Cow's milk	Slightly acid.	0	86.8	13.2	4	4	4.5	0.7
Cow's milk, 2 parts . . Water, 1 part	Slightly acid.	0	91.20	8.80	2.67	2.67	3	0.46
Cow's milk, 1 part . . Water, 1 part	Slightly acid.	0	93.40	6.60	2	2	2.25	0.35
Cow's milk, 1 part . . Water, 2 parts	Slightly acid.	0	95.60	4.40	1.33	1.33	1.50	0.23
Cow's milk, 1 part . . Water, 4 parts	Slightly acid.	0	97.36	2.64	0.8	0.8	0.9	0.14
Condensed milk	Neutral.	0	28	72	10	10	50	2.0
Condensed milk, 1 part[1] Water, 9 parts	Neutral.	0	90.31	9.69	1.35	1.35	6.73	0.26
Condensed milk, 1 part[1] Water, 15 parts . . .	Neutral.	0	93.92	6.08	0.83	0.83	4.35	0.17
Loefland's sterilized milk	Acid.	0	62.87	37.13	10.85	10.27	13.78	2.23
Loefland's sterilized milk, 1 part[1] Water, 6 parts	Acid.	0	94.02	5.98	1.75	1.65	2.22	0.36

---

[1] By volume.

TABLE X.—*Continued.*

	REACTION.	STARCH.	WATER.	TOTAL SOLIDS.	FAT.	ALBUMINOIDS.	SUGAR.	Ash.
Nestlé's Food. Fat . . . . 1.91 Albuminoids 8.23 Sugar . . . 38.92 Ash . . . . 1.59 Starch . . . 40.10 } 1 Water 10 11	Neutral.	8.65	91.75	8.25	0.17	0.75	8.54	0.14
Imperial Granum. Fat . . . . 1.01 Albuminoids 10.51 Sugar . . . trace Ash . . . . 1.16 Starch . . . 78.93 } 3 Milk . . . . . 32 Water 64 99	Slightly acid.	2.36	92.88	7.12	0.08 1.88 1.36	0.31 1.38 1.64	Trace. 1.5 1.5	0.03 0.23 0.26
Mellin's Food. Fat . . . . 0.15 Albuminoids 5.95 Sugar . . . 48.20 Ash . . . . 1.89 Starch . . present. } 3 Milk . . . . . 48 Water 48 99	Slightly acid.	Present.	91.74	8.26	0.004 2.0 2.004	0.17 2.00 2.17	1.44 2.25 3.69	0.05 0.35 0.4
Carnrick's Soluble Food. Fat . . . . 5.00 Albuminoids 18.22 Dextrin . . 67.74 Water . . . 6.14 Ash . . . . 2.99 } 3 Water 96 99	Neutral.	Unchanged starch is present to a very considerable extent. (Dextrin 2 p. c. claimed by the makers.)	97.22	2.78	0.15	0.54	?	0.09
Barley-Water. As usually made with Robinson's Barley, contains *starch* 1.4. } 1 Milk 2 3	Acid.	0.47	90.75	9.25	2.66	2.66	3.0	0.46
Biedert's Cream Mixture. (for infant of 3 months). Cream . . . ℥ 1 Milk . . . ℥ 1 Water . . . ℥ 8 Milk-sugar . ʒ 1 } ℥ 5	Acid.	0	91.56	8.44	2.7	1.8	3.8	0.14

TABLE X.—*Continued.*

	REACTION.	STARCH.	WATER.	TOTAL SOLIDS.	FAT.	ALBUMINOIDS.	SUGAR.	ASH.
Meigs Mixture. Cream (14 to 16 per cent. fat) . ℥ 2 Milk . . . . . . ℥ 1 Lime-water . . . ℥ 2 Sugar-water: Milk-sugar, ʒ 17¾. Water, 1 pint. ℥ 3 ℥ 8	Strongly alkaline.	0	88.35	11.62	3.50	1.21	6.66	0.25
Mixture recommended.[1] Cream (centrifugal cream, 20 per cent. fat diluted ¼ or ⅓). ℥ 2 Milk . . . . . . ℥ 1 Lime-water (diluted ¾). ℥ 2 Milk-sugar ʒ 3⅜ Water . . ℥ 3 ℥ 3 ℥ 8	Slightly alkaline.	0	88.42	11.58	4	1.11	6.26	0.21

---

[1] The figures in this case were obtained by actual analysis of a mixture as made by one of my patients from the ordinary milk and cream supply.

NOTE.—To prepare 1 pint of food for use in 24 hours: take milk and cream (20 per cent.) as soon as it comes in the morning, and mix as follows:

Milk, ℥ 2;
Cream, ℥ 3;
Water, ℥ 10;
Milk-sugar, 2 measures.

Place in flask in steamer and steam for twenty minutes; then remove the flask from the steamer and when still slightly warm add lime-water ℥ 1; place on ice, and give the proper amount at the proper feeding-times.

In considering the preparation of the various foods with reference to making them correspond in their analyses as nearly as possible to human milk, the question is somewhat simplified if we recognize the fact that although the percentages of the ingredients of human milk vary under certain circumstances, yet, as has already been explained in an earlier part of this paper, so far as the age is concerned, in the early months there is so little difference that a variation is as likely to occur between different milks of the same age as in the same milk at different ages, so that we probably are doing wisely not to change the percentage of the ingredients, but as the infant grows older give a food qualitatively uniform, but of varying quantity.

It will at once be seen by referring to Table X. that no matter how cow's milk is diluted it cannot be made by dilution alone to correspond to human milk. It is well, however, to remember that clinical experience has shown that infants seem, even in the early days of life, to digest the albuminoids well enough, provided that they are sufficiently diluted,—that is, about four times, which reduces them to one per cent.; and this will be of significance when we come to prepare a food which shall correspond to human milk. If, however, we reduce cow's milk so that the percentage of albuminoids is one, the fat and sugar fall so far below the standard that, although the ash has the proper percentage, yet we have an acid food markedly deficient in its nutritive quality and with its total solids represented by 3.25 instead of 12.

There is a very large number of patent foods, but they all claim about the same advantages, and closely resemble one another in their constituents, and in their endeavor to make cow's milk easily digestible, and also to make their resulting analysis agree as closely as possible with that of human milk. There are, however, certain differences by which we can divide them into classes, and we can speak of individual foods as representing their class, and thus illustrate the composition of all foods which have so far been devised for infant-feeding.

My own opinion in regard to patent foods, as a whole, is that they must necessarily be unreliable; they are thrown on the market in such numbers that the competition is extreme, and when once they have made a reputation I cannot but feel that irregularities and changes, slight, perhaps, in the eyes of the makers, may unintentionally creep in and carry their composition still further from that of the standard human milk.

Analyses show that there is a lack of uniformity from year to year, and that original claims are apparently forgotten or are allowed to give way to cheaper production. In fact, as my experience in the feeding of infants increases, and as I examine year by year the different foods, old and new, as they are actually given by myself and others, I am strongly impressed with the belief that, with our present physiological, chemical, and clinical knowledge, all the patent foods are entirely unnecessary. Their claims are not supported by intelligent and unprejudiced investigation. Their manufacturers are not in a position to judge correctly concerning them. The merit of their, at times, apparent success does not belong to them, but to other accompanying circumstances. They do great harm by impressing upon the public that a cheap, easily-prepared food is being manufactured for the good of the infant and is better than anything that can be procured elsewhere. They vary too greatly in their analyses to keep even within the acknowledged varying limits of human milk.

It is high time for us, as physicians, to appreciate exactly how inefficient in themselves and how misleading in their claims are these artificial foods, and also in what a false position, as protectors of and advisers to the public, we are placed in doing anything but ignoring them. They have a place in this article simply because there is no doubt that they are kept in the market by the physician rather than by the manufacturer. The latter is only doing what any capitalist interested in a business venture would do. The former, it seems to me, is acting somewhat blindly, and is unintentionally aiding the business interests of others at the expense of his own future reputation as a scientist, and of his ability for adapting the truths presented to him by physiology and chemistry to his clinical every-day practice among infants. It makes but little difference to us as physicians as to what these foods are claimed to contain when put on the market. It makes a great deal of difference to us what the mixture contains when given by the mother to the infant according to the directions on the label. For instance, a food may show by its analysis a fair percentage of fat or sugar, and the analyst may state this in an article on the results of his investigations or in a report to the manufacturer, and yet this same food when diluted for the infant's feeding may have these constituents reduced far below the reasonable limits of nutrition.

I repeat that I am about to mention certain representatives of the different classes of patent foods, merely to furnish the practising physician with a ready means of seeing at a glance exactly what he is giving when he uses these foods, and not because I consider that their mention will be of any direct benefit to the subject of infant-feeding.

I have already published in the *Archives of Pediatrics* (August, 1887) part of Table X., and I would here state that in the figures for diluted condensed milk in that article a correction should in justice be added to each calculation, on account of the greater specific gravity of condensed milk (the mixture being made by volume), while the other foods on account of a lower specific gravity have their percentage rated somewhat higher than they really deserve.

An examination of Table X. will at once show how all of these foods, when prepared for and given to the infant, widely differ from the standard food, woman's milk, which is represented in the upper line of the table; it will also explain how difficult it is to make the artificial foods correspond to human milk by the methods which are usually employed, and also the errors in percentage which result from these methods; it is, in fact, a series of figures which represent the element of nutrition rather than of digestion, and the merits of every food should be determined in this way before submitting it to the test of clinical experience, for our common sense must certainly be better satisfied if we know not only that the infant is digesting the food, but also that the food itself is similar (or as nearly so as we can make it) in its proportions, ingredients, and reaction to the standard which, in its results, shows the lowest rate of mortality.

The patent foods can practically be divided into—first, those which are manufactured from cow's milk modified by cereals, and, secondly, those which are not. The first class contains the starch of the cereal unchanged, or converted either into dextrin or glucose.

We can take as examples of the different classes of patent foods especial foods which will represent large numbers of other foods so far as illustrating their good or bad qualities is concerned. We have, then,—

I. Condensed milk with the addition of about fifty per cent. of cane-sugar; represented by "Anglo-Swiss Condensed Milk."

II. Condensed milk without anything added to it, simply cow's milk evaporated to one-fourth of its volume and then sterilized; represented by "Loefland's Sterilized Milk."

III. Peptonized milk.

IV. Condensed milk mixed with a cereal and its starch unchanged; represented by "Nestlé's Food."

V. A cereal food with its starch converted into glucose; represented by "Mellin's Food."

VI. Equal parts of powdered milk and wheat, the milk partially peptonized and the starch converted into soluble starch and dextrin; represented by "Carnrick's Food."

In making this division of the foods, and in discussing their analyses, I do not here enter into the question of whether these especial analyses are correct. They are taken directly from the advertisements of the foods themselves, and the directions for preparing each food are taken from its own printed labels and circulars.

Condensed milk represents, in its production, its chemistry, and its clinical results, a very fair illustration of what has been said on the subject of patent foods. It has strong advocates and strong opponents, but a simple consideration of its vital properties will easily explain its successes and its failures. It is a preparation which varies greatly in its composition, at times being a mixture which has evidently been made from skimmed milk. Condensed milk is not a sterile food, which might be supposed from the process of its manufacture to be the case, cultures having been made directly from the can by both Gautier[1] and Jeffries.[2] Any food, however, which is mixed with water would at any rate have to be resterilized when mixed for the infant's use. In Table X. the percentage of the ingredients of condensed milk—when diluted, as it commonly is, nine times—is given, and we at once see why it is easily digested but non-nutritious, for the albuminoids, ash, and sugar have been reduced to the proper amount, but the fat is far from attaining the proper percentage.

When again, as seen also in the table, and as is very often done by physicians, the dilution is made fifteen, all the ingredients excepting the ash are so far reduced below a reasonably nutritious food that we can well understand the bad results which have so often been reported concerning condensed-milk-fed infants.

The large amount of cane-sugar used in preserving condensed milk is a point rather against than in favor of this preparation, and will be referred to later, in discussing the proper kind of sugar which should be put into an artificial food.

---

[1] Semaine Médicale, 1887, 20.

[2] Amer. Jour. of the Med. Sciences, May, 1888.

Clinically, then, condensed milk represents a food easily digested but not sufficiently nutritious: the former explained by its low percentage of albuminoids and ash, its neutral reaction, its antiferment properties, and its proper percentage of sugar; the latter, by its great lack of fat. Among the poorer classes and in infant asylums it is a favorite food for the physician to prescribe, because the infants digest it so easily; but the testimony of those clinical observers who look beyond the temporary digestion to the subsequent nutrition of the child supports the view that condensed milk, even if we set aside the objections to which all patent foods are subject, must be modified by more than the addition of water before it can safely be given as a continuous food to the average infant. For preparing the way for other more nutritious foods in cases of difficult digestion, for convenience in travelling, and where for any reason the intelligence or the proper desire to take trouble about the food is lacking in the parents, condensed milk, from its simplicity in preparation as well as from its other attributes mentioned above, is a valuable addition to other more rational methods of artificial feeding. The commonly accepted opinion that condensed milk contains too much sugar is an error, for by referring to the table it will be seen that, as usually given, the sugar in the mixture is below the proper percentage, and we have merely the fat to deal with, and the reaction, which should be made alkaline. We must, then, modify this condensed-milk mixture; and not only is the fat an important part of this modification, but the proper amount of fat; for, although it is admitted that a large percentage of surplus fat is frequently found in the fæces of infants whose digestion and nutrition are normal and whose food is breast-milk, yet we have no more right to conclude from this that a small percentage of fat is sufficient for nutrition, or that a large percentage will be taken care of by this outlet, than we have to assume that there is too much oxygen in the blood because we find that a certain surplus of oxygen is found in the arterial blood and returned to the lungs in the pulmonary veins. In fact, it is far more probable that nature introduces a certain percentage of fat into human milk with a purpose which can only be accomplished by that percentage, and that it is an error to vary this percentage beyond the variation which commonly occurs in average human milk.

The production of animal heat is so very important a part of the young infant's well-being that it is not surprising that we should find so large a percentage of fat and sugar in the food which is provided for it. We should remember also that, while the sugar is the more digestible of the two, the fat contains more potential energy (that is, heat-producing power), in a given weight, than the sugar, and that its presence in the milk is not only for the purpose of nutrition, but also as a means for the maintenance of the bodily heat. This function of the fat cannot with impunity be trifled with, and is essential for that active metabolism spoken of in an earlier part of this paper, with its corresponding rapid increase in growth, so well exemplified in the very organ, the stomach, which receives this heat-producing food. A proper amount of fat is probably of great aid in the regulation of the fæcal discharges. From what has been said above, we should naturally expect that unless the standard percentage of fat was attained, or at least a near approach to it, trouble would be likely to arise; and this corresponds to my individual experience in the cases where the especial ingredient which was disturbing the success of the food was the fat. I have found clinically that under the proper percentage of fat the nutrition suffers, the digestion is not so good, and there is a tendency to constipation, while where the fat percentage is decidedly above the standard the digestion is very much affected, there is a tendency to diarrhœa, and in consequence a resulting poor nutrition.

Unless, then, it is impossible to be more exact in arranging the percentage of fat in condensed milk, as is often the case among the poorer classes, where cod-liver oil is used as a cheap expedient for rectifying this source of error, the addition of indefinite amounts of fat to a food is to be deprecated, just as it is unwise to add indefinite amounts of sugar, and we should seek for a better combination than is offered to us in condensed milk.

It is, however, practically a very simple matter to increase the percentage of fat in a mixture such as condensed milk and water, by means of the proper amount of cream of a given fat percentage, such as will be explained later. Thus, by referring in Table X. to condensed milk diluted nine times, we see that in one hundred parts of such a mixture there is only 1.35 per cent. of fat, and to raise this percentage to the proper 4 per cent. we must add sufficient 20-per-cent. cream to make up 2.65 per cent. of fat in the condensed-milk mixture: this is easily done by the rule of three:

$$100 \text{ parts cream } (20 \text{ p. c.}) : 20 :: x : 2.65.$$

$20\,x = 265$. $x = 13\frac{1}{4}$ c.c. of cream to be added to each 100 c c. of condensed milk diluted nine times: this, practically, is about one drachm to every ounce.

The second kind of condensed milk, represented in the table under the name of "Loefland's Sterilized Milk," must be diluted ten times in order that the albuminoids should be brought within proper limits for digestion. This reduces the total solids, fat, and sugar to such a minimum percentage that for purposes of nutrition a considerable amount of modification would be needed, and quite as much as for fresh cow's milk.

Peptonized milk is cow's milk with its albuminoids partially or entirely predigested by means of the extract of pancreas and soda. Now, there is no doubt that the albuminoids of cow's milk have been a source of trouble to the infant's digestive apparatus, and under certain circumstances can with great benefit to the infant's digestion be treated by predigesting them for a time and thus allowing a stomach which otherwise digests well to rest and recover itself. It is of use, also, where a decided idiosyncrasy of the individual precludes the digestion of these ingredients of the milk; but, besides that, the indigestion is often attributed to the lack of power to digest albuminoids at all, while in fact the stomach is simply rebelling against an amount of albuminoids above the standard percentage, or against some other ingredient. It would seem that, for the average infant digestion, this predigesting of the albuminoids or any other constituent of the milk is contrary to nature's teaching. There are certain natural functions which should be allowed to act as they do on human milk, and it seems irrational and contrary to the laws of physiology not to encourage all the functions to act naturally, each in its own province, instead of forestalling their action and allowing them to fall into disuse and thus be weakened. The baby's stomach is intended to digest albuminoids, and not to have the albuminoids digested for it. Clinically, also, the use of peptonized milk supports this view, for, so far as I know, no very brilliant results have been obtained from its use. Peptonized milk, then, is a food consisting of too large an amount of digested albuminoids, too little sugar, and a very large over-proportion of ash.

Any food which introduces an element foreign to the ingredients of human milk is to be looked upon with suspicion, as it is not likely that we can improve on nature's method of adapting the food to the infant's digestive functions: we should therefore consider carefully before recommending the various classes of food which contain starch, which by referring to Table X. will be seen to be the foreign element which enters into the representative patent cow's-milk modifications,—Nestlé's Food, Imperial Granum, and Carnrick's Food. As has already been said, it is not merely necessary to know the percentages of the different ingredients as they occur in the printed analysis of the especial food; to us the important question is, what, as shown in the table, are the sums total of cow's-milk percentages and the patent modification percentages, these sums total being what the infant receives. Judged by this standard, Nestlé's Food provides a larger amount of starch for the infant's digestion than the other foods, almost one-third less total solids than woman's milk, practically no fat, too little albuminoids, one-half too little sugar, and a fair percentage of ash. A mere glance at the figures in the table then tells us whether we have an easily digestible and nutritious food to deal with. For instance, the success of Imperial Granum is evidently in its correct percentage of albuminoids and ash making it easily digestible, but its failures are readily explained by its reaction, its foreign ingredient, and its very low percentage of fat and sugar.

Reference has been made above to the capability shown by even very young infants to digest the albuminoids of cow's milk when it is reduced to one or two per cent.; and this is a factor which probably enters to a greater degree than is usually recognized into the easy digestion of these foods, and possibly too much credit has been given to the starch as a means of making the albuminoids more digestible. At any rate, it is a question worth consideration, for it certainly is more rational not to introduce a foreign ingredient like starch into the food if we can make it digestible in some other way. Examining this question of albuminoid percentage in the three large classes of patent foods represented in Table X., we find Imperial

Granum 1.64, Mellin's Food 2.17, and Nestlé's Food 0.74. Now, all these correspond very closely to the albuminoid percentage of human milk, that of Mellin's Food perhaps being rather high, and all these foods are found to be easily digestible; so also where barley-water is mixed in the usual way with milk, one to two, the albuminoids of the resulting mixture are notably diminished in amount and naturally are more easily digested than when they stand at a higher percentage, as in cow's milk undiluted. This has already been shown in Table IX., where the question of the beneficial effect of the starch itself on the albuminoids is seen to be somewhat problematical. This brings us at once to the consideration of whether starch should be made a part of the infant's food. Physiologically, we know that during the first ten or twelve months of life the function of converting starch into sugar is in the process of development. It is also known that a partial conversion of the starch can be performed at quite an early age, and by exceptional cases to a much greater extent than by the average infant. But, besides the well-known fact that the presence of a function does not necessarily mean that it must be used, it is also rational to suppose that when a function is being developed it should not be taxed with a trial of the use which will later be demanded of it. That is, a function develops more perfectly if its power is not too early exerted to its utmost. With these facts before us, we judge, as indeed we could also do without them by simply referring to the best known food for infants, woman's milk, that starch should not form a part of the infant's artificial food. Although it may perhaps not be of a great deal of significance in connection with this discussion of starch, I might mention that I have had a number of infants in the early months of life fed for twenty-four hours entirely on the preparation of barley-water represented in Table X., and in every case, except one, starch was found in the fæces; and this will be of considerable interest when we consider that the very low percentage (0.47) of starch in the mixture does not provide much material for conversion into sugar.

The especial merit which is claimed for the next representative patent modification of cow's milk on page 315, No. V. (Mellin's Food), is that it contains almost fifty per cent. of converted starch: the manufacturers, recognizing that starch is not a proper ingredient physiologically for an infant's food, have had this starch converted into glucose. It is difficult to understand why, except perhaps for financial reasons, expensive machinery should be made for the working of a material which should not exist in an infant's food, and for the purpose of chemically converting this into sugar, which might as such be directly added to the food in the beginning. In addition to this, the resulting sugar is glucose, which is not the sugar of woman's milk, but the final product of the milk-sugar digestion; and thus again the natural function of converting milk-sugar into glucose is allowed to fall into disuse, which is objectionable, just as in peptonized milk such disuse of a function is unwise.

Referring to the table, we next see that the percentages in the mixtures which are derived from Mellin's Food itself are exceedingly insignificant, the fat being inappreciable, the albuminoids so low as to be of little value for nutrition, the ash only half the amount of the smaller figure representing woman's ash, and even the sugar being only about one-fifth as large in amount as in woman's milk. The resulting percentages where the cow's milk has been added give us a mixture containing one-half too little fat, a somewhat too high amount of albuminoids, almost one-half too little sugar, and an ash just double the highest figure representing the ash percentage in woman's milk. It will be noticed that starch is entered in the table as present, though it is claimed to be entirely converted into sugar. This starch, as found by Dr. Harrington in a number of different specimens, though perfectly appreciable was not sufficient in amount to be of any great importance. Cow's milk being acid, we should naturally expect to find the resulting mixture with any of these foods acid; and this, as seen in the table, is the case.

The next patent modification of cow's milk, Carnrick's Food, is not assisted in its nutritive properties by additional cow's milk, and consequently presents by its own analysis a mixture which practically amounts to a two-per-cent. solution of dextrin and water. Fifty per cent. of a cereal has been introduced in the food, and it is claimed that its starch is immediately converted into dextrin. By this procedure the infant's undeveloped function is called upon to exercise its power many months before nature has intended it to be used. The reverse of this, the allowing a developed function to fall into disuse by having its work done for it, might be said to occur in the predigesting of the albuminoids; but the table shows us that there is only about one-half per cent. of these albuminoids left by the dilution to digest. The manufacturers claim in their circular that in the process used by them the starch is first converted into soluble starch and then into dextrin; but careful examination by Dr. Harrington has shown that a very great proportion of the starch is unchanged and insoluble, and that the great bulk of the food is not capable of solution. The name, therefore, of "Soluble Food" is a misnomer.

The total solids, when prepared according to direction, outside of the "dextrin" percentage, are represented by 0.77; the fat is scarcely appreciable, the ash slightly under the lowest figure representing woman's ash. Where woman's milk, then, contains certain ingredients amounting in all to twelve per cent., the same ingredients in Carnrick's Food when mixed according to the directions on the label are represented by 0.77.

From what has already been shown in Tables IX. and X. it will be understood that the dilution of cow's milk with barley-water will be but very little more satisfactory than with plain water, and, as the resulting mixture with barley-water is acid, the addition of lime-water in sufficient amount to produce the proper degree of alkalinity will be preferable, the barley-water supplying a percentage of starch of no nutritive value to the mixture, and its assistance in digesting the albuminoids being much over-rated, if, indeed, it exists at all. Lime-water, on the other hand, is the most simple adjuvant which we can use for making cow's milk alkaline, for so small is the amount of lime contained that its addition in even considerable quantity to cow's milk does not materially alter the amount of the total ash, while it will render cow's milk alkaline when added in such small amount as one-sixteenth part, so that simply for this purpose alone (making an acid milk correspond in its reaction to woman's milk) it is very valuable, for it apparently does not produce any other changes in the milk. The question next arises as to whether cow's milk can be modified without the use of patent foods or foreign ingredients and made to correspond to the percentage given in the upper line of Table X. This has been accomplished, with more or less success, by the addition of milk-sugar, of fat in the form of cream, and of lime-water.

Biedert, having come to the conclusion clinically that the young infant could digest easily a mixture containing one per cent. of albuminoids, devised what he called his cream mixture, which, as seen in the table, contains the proper amount of albuminoids and ash, but entirely too little fat and sugar. This mixture also, though a step in the right direction, is far from exact in the method of its preparation, either as originally recommended by Biedert or in the condensed patent "Cream Preserve" which under his name has appeared in the market, and is open to all the criticisms which have been made on patent foods in general.

Dr. J. F. Meigs, of Philadelphia, having found in a very extensive practice among infants that certain proportions of milk, lime-water, cream, and milk-sugar appeared to suit the average infant's digestion and nutrition better than anything else that he had experimented with, advised his son, Dr. A. V. Meigs, to determine chemically how near this mixture approached to the analysis of average human milk. The younger Meigs, having already determined chemically that the average woman's milk contains but one per cent. of albuminoids, found in the analysis of his father's mixture that it also presented one per cent. of albuminoids. He then perfected the mixture still further by using a definite amount of milk-sugar, seventeen and three-quarters drachms to a pint of water, and a cream of about sixteen per cent. fat. With these ingredients in the proportion of one part milk, two parts cream, two parts lime-water, and three parts sugar-water, he found the almost unvarying analysis to be an alkaline mixture containing about 88.35 water, 3.50 fat, 1.21 albuminoids, 6.66 sugar, 0.25 ash. This mixture at once established the possibility of a simple method of making the composition of a mixture correspond to that of woman's milk, and gives us as definite a chemical basis to work with as we are justified with our present chemical and physiological knowledge to expect. The details of making the mixture, however, form a very important part of its practical success, and will be referred to later, for unless these details are precisely carried out the mixture is no better than those which have already been mentioned.

A great deal has been said and written about "cream mixtures." These have met with many alleged successes and failures. So many of the successes and so many of the failures, however, have, in all probability, had so little to do with the mixtures as "cream mixtures," that it cannot be said that our clinical knowledge has so far accumulated many scientific or practical facts concerning them. It would indeed seem that the theory of the "cream mixture" has not been thoroughly understood by the general practitioner or practically carried out by him, although he, as a rule, is the one who has the greatest opportunity for doing so. It is better not to use the term "cream mixture." We merely add the cream so as to supply a fat which, so far as we know, corresponds to the fat of woman's milk, and to make the percentage of this especial ingredient of the mixture agree with its percentage in woman's milk. We should not attach so much importance to this or to any of the ingredients of the artificial foods as to name the food from it, but should give all the ingredients an equal share of importance, or, as I have stated above, we inevitably fall into the error of neglecting some of them. The intention of adding cream is simply to add fat: we therefore should not speak of cream or fat mixture, any more than we should of sugar mixture, as it conveys the false idea to the average practitioner that it is the cream which is of especial importance.[1] An infant's artificial food must, as I have already said (until our knowledge is much more extended than at present), consist of cow's milk modified. Cream is one of the modifying elements, and may be counted simply as fat. The fat is an important part of the whole, but only so far as it is in the proper amount of three to four per cent., for in excess it is a disturbing element.

The "Meigs Mixture," even when carefully prepared, has not given nearly so good results as those obtained with woman's milk. We have, however, gone as far with it as the chemistry of to-day will permit, so far as the actual percentages are concerned. Much, however, can be added to this factor of correct percentages by improvement in the exact application of the principles involved; and this is to be accomplished by a more extended examination of this mixture. For some years I have made a careful study of the Meigs mixture, both clinically and in the laboratory, and the following are the results of my investigations. So many analyses of this mixture have been made for me by Dr. Harrington with uniformly satisfactory results that I have assumed that so far as the question of percentage is concerned I can safely accept the proportions of one part ordinary mixed herd milk, two parts cream twelve to sixteen per cent., two parts lime-water, and three parts sugar-water.

The taste of the lime-water is very perceptible in the Meigs mixture, and destroys the striking similarity in taste to woman's milk which is attained by using a smaller amount of lime-water. The Meigs mixture is strongly alkaline, while woman's milk is very slightly alkaline and often neutral. The infant at times does not like the taste of the lime-water, and the parents are, therefore, often opposed to its administration in such large amount. It is wiser to try and make an artificial mixture approach as closely as possible to woman's milk in both taste and reaction; and this can easily be done by reducing the amount of the lime-water, for, as has been explained, it is useful only for the purpose of making the acid mixture alkaline.

---

[1] I am continually meeting with physicians who speak about their success or failure with cream mixtures, and who are feeding the infants simply on cream and water,—that is, diluted fat, just as Reed & Carnrick's Food is—even if we accept the manufacturers' figures—diluted dextrin.

Dr. Harrington has made an estimate by actual experiments of the amount of lime-water which was needed to produce an alkalinity in the mixture which would correspond to the alkalinity of human milk. His results were as follows:

MEIGS MIXTURE WITH	REACTION.
25 per cent. lime-water	Strongly alkaline.
12.5 per cent. lime-water	Still strongly alkaline.
6.25 per cent. lime-water	Slightly but distinctly alkaline, and corresponding to woman's milk.

It is thus seen that there is at least four times too much lime-water in the Meigs mixture; and we accomplish our desired result of making the reaction of the mixture correspond to that of woman's milk by adding, in place of one-fourth lime-water, three-sixteenths plain water and one-sixteenth lime-water.

Cream varies much in composition, owing to the different methods employed in the process of skimming. In cities or towns where the cream can be obtained from a centrifugal machine the question of the fat is much simplified, and I have practically found that it has been much easier to regulate this factor in the food for infants in and near Boston than for those who live at a distance in the country, although the latter would naturally be provided with fresher milk and cream than could be procured in the city. In the country, however, the milk has at any rate to stand some hours for the cream to rise, while the cream from the twenty-four-hours-old city milk is removed by the machine in a few minutes, so that the question of time is somewhat obviated, while the opportunity of obtaining an unvarying percentage of fat is far greater in city cream than in country. In large cities, such as London and New York, where the need for exact infant-feeding is felt to a greater extent than in the smaller cities and towns, on account of the density of the population, a cream of almost unvarying percentage in fat can easily be provided for the people at large, if physicians will but take the trouble to attend to it. I myself, in Boston, have had but little difficulty in arranging this question of cream with one of our large milk-dealers, who uses centrifugal machines. This dealer has two grades of cream, a very thick cream which he sells at sixty cents a quart, and a thinner cream which he calls his ordinary cream and sells at thirty cents. On inquiry, I found that his machines are run at an almost unvarying rate of seven thousand rotations of the wheel to the minute, the resulting cream being thin or thick according as the stopcock supplying the milk to the machine is turned on to a greater or less extent. I found that the ordinary cream keeps sweet longer than the thick cream. I next had a careful analysis made of the ordinary cream every day for two weeks, and found that the average and almost unvarying percentage for the fat was twenty. This ordinary cream, then, which is about as thin as the dealer's machine will make it, is really of very good quality, and we can count on its containing about twenty per cent. of fat.

When a cream of sixteen per cent. fat has been used in the Meigs mixture, I have usually found that the resulting percentage of fat was rather above than under four, so that by diluting the ordinary centrifugal cream with one-quarter part water, and then calling this "cream," I have, by adding this resulting fifteen per cent. cream in the proportion of one-quarter to the mixture, usually obtained a satisfactory resulting fat percentage of from three to four. Where centrifugal cream cannot be obtained, what Dr. Meigs calls ordinary cream will often give a very fair resulting fat percentage; but if the patients cannot afford to have an analysis made the physician should at least ask to see a specimen of the cream which is being used, since people show the greatest lack of intelligence in judging what is good ordinary cream, thinking that if it comes from their own cow in the country it must be good, while really it may be a very poor cream so far as evidence is given to the eye. It is well, however, to advise people not to use the rich cream of fancy cattle, but the cream from mixed common herd milk, and also to have always the same person skim the milk, and to have the milk stand for the same time and in the same temperature, for the percentage varies considerably with different skimmers and according as it has stood for a longer or shorter time and in a cool or a warm place.

We have already spoken so fully concerning the albuminoids and ash that it is hardly necessary to refer to them here again, except so far as is requisite to emphasize the importance of reducing the percentage of the albuminoids in the mixture to one, and the ash to 0.1–0.2.

The proper percentage of sugar to be given in an infant's food has been stated to be from six to seven; and the method of obtaining this percentage has been shown in the chemistry of the Meigs mixture.

Regarding the kind of sugar which should be used in making up an artificial food, we have certain questions to consider which would seem to be not altogether unimportant. Cane-sugar has been in the past and is still a favorite form with which to regulate this part of the solid constituents of the food, and the reasons given for using it have been its preservative qualities, as seen, for instance, in the manufacture of condensed milk, and the theory that it is not liable to set up excessive so-called lactic acid fermentation, with its consequent disturbance of digestion, as is supposed to be the case with milk-sugar. Cane-sugar in a concentrated form as it is found in condensed milk seems to act as a preservative; when, however, it is diluted, as in its administration to the infant, the cane-sugar ferments very readily, and under these circumstances is no better than milk-sugar. Reasoning from analogy, we should say that milk-sugar being the form which is always found in the milk of all mammals, it would be natural to suppose that this form of sugar has been put there for some good purpose, and that it is needed for the accomplishment of some process which takes place after the food has been swallowed by the infant. Both cane-sugar and milk-sugar are converted into glucose in the intestine. There seems, however, to be some difference in the degree to which they can be used for purposes of nutrition before they are converted into glucose. Cane-sugar, so far as is known, is merely a reserve, and cannot be directly used for nutrition; in fact, this holds true in whatever it exists, whether in plants or in animals. Milk-sugar, on the other hand, is probably not merely a reserve, but may possibly be utilized as such in the economy. Thus, Bernard[1] has shown that seven grains of milk-sugar dissolved in an ounce of water could be injected under the skin of a rabbit without the subsequent appearance of sugar in the urine, while under the same conditions and in the same amount cane-sugar was found to be eliminated as foreign matter by the kidneys.

Milk-sugar undergoes no direct alcoholic fermentation, but readily undergoes a change to lactic (possibly acetic) acid in the presence of nitrogenous ferments, while cane-sugar easily undergoes alcoholic fermentation, but changes to lactic acid less readily than milk-sugar: cane-sugar, however, takes on the butyric acid fermentation more readily than milk-sugar. On page 304 I have already referred to the *Bacillus lactis aërogenes* (Escherich) as being present in normal digestion and for the purpose of acting on the milk-sugar to produce an organic[2] acid which will drive out the more noxious forms of bacteria, which by their presence would interfere with normal digestion. When also milk-sugar is converted into glucose, we physiologically have a gradual conversion into lactic acid, which may aid in the digestion of the albuminoids, thus giving us a very valuable addition to the means at our command of rendering modified cow's milk digestible.

Dr. Jeffries writes to me concerning this question as follows:

"In reference to the question as to the difference of the various kinds of sugar in the digestive tract, the following seems to be of interest. Starch, dextrin, inulin, cane-sugar, and dextrose afford material for the butyric acid[3] fermentation; milk-sugar first after completed hydration.

"Again, Escherich,[4] in speaking of Brieger's bacillus, says, 'Milk is coagulated with sour reaction first after several days (eight to ten) at the body-temperature. With exclusion of air this bacillus cannot grow either in milk or milk-sugar solution, but will in grape-sugar.'

---

[1] Flint, Physiology, 1879, p. 467.

[2] According to Escherich, this organic acid is lactic; while Baginsky holds that the *Bacillus lactis aërogenes* turns milk-sugar largely into acetic acid, producing only minimal amounts of lactic acid, and he suggests the name of *Bacillus aceticum* for this form of bacterium.

[3] Flügge, Mikroorganismen, 1886, p. 484.

[4] Darmbacterien des Saúglings, p. 67.

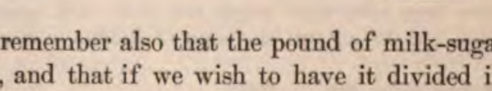
"We thus see that the milk-sugar offers less danger of the butyric acid ferment, which we know makes much trouble at times in the body, and under the conditions of the intestine should be exempt from the assaults of Brieger's bacillus."

When, in connection with what has been said above, we consider that by means of sterilization we can practically put an end to the lactic acid fermentation which may have begun to act on the milk before it enters the stomach, it would seem that we are justified on both physiological and bacteriological grounds in using the same animal sugar in our artificial mixtures which is found in the infant's natural food, instead of introducing a vegetable sugar, which in any milk is a foreign element.

The dangers from lactic acid are, at any rate, much exaggerated by writers on this subject, and there are many other questions which if more carefully attended to would render the supposed evil results of the lactic-acid bugbear much less noticeable.

We can now discuss the best method of preparing the food for household use. We will suppose, by way of illustration, that we are using a centrifugal cream of twenty per cent. fat. We dilute this cream one-quarter, and make this diluted cream, containing fifteen per cent. of fat, one-quarter part of the whole mixture. It was found by Meigs that, as already stated, the proper percentage of sugar in the mixture was obtained from a solution of milk-sugar seventeen and three-fourths drachms to one pint of water. In the analyses of the mixture I have found that the sugar percentage was, if anything, usually somewhat under seven per cent.: so that, to simplify the figures, and without running any risk of appreciably changing the percentage from seven, I have added eighteen drachms of milk-sugar to the pint of water. In the same proportion we find that in every three ounces of water there should be three and three-eighths drachms of milk-sugar, and that this three and three-eighths drachms should be the amount for every half-pint of the mixture. I then had a tin measure made to hold three and three-eighths drachms of milk-sugar. This obviates the expense of having the milk-sugar put up in packages by the apothecary, and is sufficiently exact not to alter the sugar percentage in the mixture. One of the leading apothecaries in Boston sells a pound of the highest grade of milk-sugar for fifty cents and gives with it one of these measures, which is represented in Fig. 10.

FIG. 10.



It is well to remember also that the pound of milk-sugar contains seven thousand grains, and that if we wish to have it divided into packages of three and three-eighths drachms and to pay about one dollar and a quarter instead of using a measure and paying fifty cents, we can order thirty-five packages to be made from the pound and we shall still have the resulting percentage in the mixture substantially correct. We must also remember that the proportion of lime-water should be one-sixteenth part of the whole mixture,—that is, one-half ounce for the half-pint. Before describing the exact manner of preparing the food from these materials, it will be well for me to state the result of my experiments with the sterilized Meigs mixture, as it may save other investigators the trouble of repeating them.

On steaming a mixture of cream, milk-sugar, water, and lime-water in the usual way for twenty minutes, it was found that the liquid had become of a light-brown color. Dr. Harrington found that the color was due to certain brown products formed by the action of the lime-water on the milk-sugar at a high temperature. This color in itself does not alter the value of the mixture; but Dr. Harrington also found that, while at the beginning of the steaming the reaction of the mixture was strongly alkaline, this reaction as the steaming was continued grew gradually less and at the end the reaction might be neutral. This change in the reaction Dr. Harrington supposed to be due partly to the formation of a compound of lime and sugar and partly to the fact that on heating lime-water much of the lime is thrown down, so that, as the object of the lime-water is to render the acid mixture alkaline, this object is defeated when the mixture is sterilized. The lime-water should therefore not be added until after the mixture has been steamed and partially cooled.

On page 300 I have described my method of sterilizing a single feeding for the infant, and my four-ounce tube with its rubber cot is represented in Fig. 9 undergoing sterilization. Sterilizing each meal, however, is considered a great deal of trouble by many mothers; and when we consider that at any rate the milk and cream should be sterilized as soon as they are received in the morning, we can well see that some other method may be preferable in these cases.

To meet this demand, I have lately been in the habit of having the nurse prepare the whole quantity of food which is to be used by the infant in twenty-four hours, by mixing it as soon as the milk and cream come in the morning, pouring it into the litre bottle represented in Fig. 9, sterilizing in this way the whole twenty-four hours' food at once, and keeping it in the ice-chest to be used when necessary. Giving the proportions of the various ingredients to make up a half-pint of the mixture is sufficient explanation for preparing larger quantities, such as a pint or a quart. The directions to be given for preparing a half-pint of the mixture by this method are very simple, and can be carried out by individuals possessed of a very small amount of intelligence.

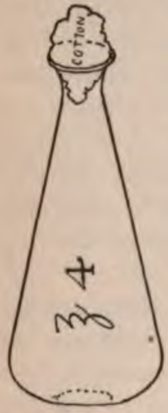
Mix, as soon as received in the morning,—

Cream (20 per cent. fat), ℥1½;
Milk, ℥1;
Water, ℥5;
Milk-sugar, 1 measure.

Steam the mixture in the bottle for twenty minutes, the mixture being introduced by means of a funnel, in order that the neck of the bottle shall be kept dry. The bottle is to be stopped tightly with a cotton plug. After steaming, remove the bottle immediately and allow it to cool partially; then add half an ounce of lime-water, and keep on ice. This is the simplest way of preparing the food, and will probably prove to be the most practical and the most popular; but of course it is open to the objection that every time the infant is fed the cotton has to be removed from the bottle, with the resulting danger of contamination of the remaining fluid, which indeed is but slight. Where, however, as in very hot weather, this objection is found to be a valid one, small bottles for each feeding should be used.

I have arranged this latter method as follows. The mixture is made in the morning as before, and is then poured into the number of Erlmeyer flasks corresponding to the number of feedings. The flasks are to hold four or eight ounces, according to the amount to be given at each feeding. Eight or ten flasks are usually needed, and they are to be stoppered with cotton and have their mouths carefully dried, as was directed for the large litre bottle. In this way the food can be prepared by one steaming for twenty-four hours; and, as the cotton is not removed until the feeding-time, the mixture will keep indefinitely and need not be put on ice. Fig. 11 represents a four-ounce Erlmeyer flask. When this method of preparation is used, the proper amount of lime-water is to be added at each feeding.

FIG. 11.



So much has been said about the expense of preparing a food with cream and milk-sugar that it will be interesting to examine into the actual expense incurred in using this mixture.

The cost of feeding an infant three or four months old will represent approximately the cost for the most important part of the feeding-period and the one which is most difficult to manage.

This cost amounts to about twelve cents a day; and there are very few parents who are unable to pay this for their infant during the early months of life. The expense of feeding in this way cannot be said to be great or beyond the means of the people at large, so that, although the food and its method of preparation are the results of the scientific investigation of what is best without regard to cost, the actual daily expense happens to compare well with what we can reasonably demand as the price which the poor should be expected to pay for the nourishment of their offspring.

In conclusion, we can fairly say that it is possible in artificial feeding to approach the standard human breast-milk much more nearly than is usually attempted, and there is no reason why clinical results should not be greatly improved, if physicians will only take additional time and trouble to follow more uniformly nature's teaching. In all classes of life a much greater amount of time, expense, and thought is given proportionately to the preparation of food for the adults of the family than for the infants. This is a mistake both from a humanitarian and from an economical point of view, for the infant is much more susceptible to irregularities of diet, with their resulting suffering, than the adult, and when once the train of symptoms usually called dyspeptic is established, infinitely more trouble and expense are entailed than if more exact methods of feeding had been adopted before the digestion was disturbed. In the early weeks of lactation, after the mammary function has been fully established it is well to have a number of analyses made of the mother's milk, and to keep the results as a control record to act as a guide for the preparation of an artificial food in case, as so frequently happens, something should occur to end the nursing at an early period. It is highly probable that the digestive function of the individual infant may have certain idiosyncrasies which correspond to some idiosyncrasy in the percentages of its mother's milk; and in cases of difficult digestion where the artificial food, which has been made to correspond to the analysis of average woman's milk, fails to agree, reference to this control record may accomplish the solution of the problem sooner than if we have to ascertain experimentally, by changing in turn the percentages of the different ingredients, in which particular ingredient the idiosyncrasy of this especial infant is to be found. The assistance of the skilled chemist is too little sought after in determining these questions of infantile digestion and nutrition, and in the future must necessarily be made use of if there is to be any advance for the better in the subject of artificial feeding.

Where an infant, then, is to be fed with artificial food, give precise directions as to the times of feeding, the amount at each feeding, and the feeding-apparatus which is to be used. See that the analysis of the food corresponds as closely as possible to that of human milk; give instructions as to the proper temperature of the food; see that the reaction is slightly alkaline; and then, if there is any difficulty with the digestion, sterilize the food. If this is not successful, refer to the control record, and adapt the food to any maternal idiosyncrasy shown by this record. If no control record has been kept, experimentally try to discover the especial idiosyncrasy of the individual infant by changing the percentage of the fat, sugar, albuminoids, or ash.

WET-NURSES.

By WILLIAM H. PARISH, M.D.

THE difficulties attending the selection of a wet-nurse are of such a character that the physician must bring into play his professional knowledge and must exercise the greatest care and shrewdness. No other one should assume the responsibility. The risks to the infant are so serious, and it is so difficult to avoid them fully, that some experienced practitioners disapprove entirely of the employment of a wet-nurse. Not only must the milk be nutritious and adapted to the infant, but the risk of the infant's contracting some serious and it may be loathsome disease must be avoided. In this country the class of women from among whom wet-nurses are chiefly attainable consists largely of the ignorant poor and immoral, those who are specially liable to be diseased and to practise deception. Unfortunately, too, the number of available women from among whom a choice must be made is always small, and, moreover, the demand for a wet-nurse may be so urgent on account of the child's failing condition that delay is deemed impossible.

Moral Fitness, etc.—The moral character of the woman must be considered. While most probably her milk cannot influence the future moral organization of the growing child, yet her close association with the infant possibly may make a permanent impress on its pliant brain. Moreover, the woman will bear a close and peculiar relation to the family in which she is introduced, and she may become a cause of no little unhappiness if she is dissolute or of bad temper. She soon learns or believes that her services cannot be dispensed with, and she becomes an unbearable tyrant. If of intemperate habits, she, when in a state of intoxication, may injure the infant by accident or by design, and at that time will furnish milk of an injurious character. If of violent temper, she will furnish during her exhibitions of temper milk unfit for the child; for authenticated cases have been reported of even convulsions occurring because of milk altered by such mental disturbance. A woman of bad temper, or one without due sense of her responsibility, may leave suddenly, possibly when the child cannot bear the consequent abrupt change in diet. The wet-nurse should be cheerful, active, good-natured, temperate, moral, and of average mental capacity. If dissolute, she is liable to contract syphilis or gonorrhœa, and the child may thus become infected. When the woman is an inmate of a hospital, it is not only necessary to learn of her conduct while in the hospital, but her habits of life when out of the institution should be ascertained from her acquaintances. By preference she should be married; but in this country married women do not often undertake wet-nursing. If her child is illegitimate, it is best that it should be her first child. Repetition of illegitimate pregnancy is indicative of a degree of moral depravity that should render her fitness more than doubtful. To say, however, that only mothers of legitimate children should be accepted would be almost equivalent to rendering the attainment of a wet-nurse an impossibility in the United States.

In Europe it is not unusual to employ a legitimate mother to suckle the child several times during the twenty-four hours, she coming to the house for that purpose, while at the same time she suckles her own child at her own home. This plan has its drawbacks; for naturally the woman gives the preference to her own child, and her diet and general hygiene cannot be so closely overlooked as when living with the family employing her. There is some danger, too, that she may expose herself to the contagion of such diseases as measles, scarlatina, etc., and convey the poison to the child she wet-nurses. There are circumstances, however, under which such employment of a wet-nurse would seem judicious, provided, of course, the woman was shown to be suitable. Generally, in America the woman is entirely separated from her own offspring, and the latter, if living, either is placed in some home for infants, or is given into the care of some woman to be fed artificially and usually to die. A proper appreciation of the moral obligation involved would induce the parents of the favored child to make due efforts to secure the proper care of the infant deprived of its natural rights. It is also in the interest of their child to exercise this humane act, for a knowledge on the part of the wet-nurse that her child is receiving kind attention will go far towards securing that mental equanimity which is necessary to the furnishing of a proper amount of suitable milk.

General Physical Condition.—A good wet-nurse should be robust and strong, but not very fat. Not only should she be in apparent good health at the time of employment, but she should also be free from evidences of serious past dyscrasia.

A scrofulous woman cannot furnish good milk. The applicant must be questioned as to the occurrence, especially during her childhood, of the symptoms of scrofulosis, and evidences of the disease must be looked for, such as cicatrices in the region of the cervical glands or enlargement of those glands. The presence or absence of scars about the joints must be ascertained. Existing tuberculosis, or the tuberculous taint as indicated by the family history, should exclude her as a wet-nurse. The possibility of the transference of tuberculosis to the infant, it seems to me, cannot be questioned. Giving suck to a child tends to develop a latent tuberculosis in the woman, and thus the woman giving no evidences of tuberculosis when engaged may become distinctly tuberculous during lactation, if she is of a diathesis in which tuberculosis readily develops.

A woman who has suffered with rachitis in her childhood should be rejected. The evidences of rickets are to be looked for in the altered condition of such bones as the clavicles, the tibiæ, those of the forearm, the ribs, and the vertebræ. General dwarfing of the osseous system may alone indicate rachitis. Neither the tuberculous nor the rachitic woman will furnish nutritious milk, nor can she continue to suckle the child for the usual period of twelve or fourteen months. It is well to remember also that rachitic women are liable to lose their offspring during labor and consequently seek not infrequently the position of wet-nurse.

Marked anæmia may indicate some serious dyscrasia, though moderate anæmia may disappear under generous diet and proper medication.

The most important constitutional disease to exclude is syphilis. It must be remembered that the woman may be ignorant of ever having had a chancre, or if she is cognizant of the fact she will probably deny that it ever existed. She must be cross-questioned as to the multiform manifestations of the disease. An inspection of a large part of her skin-surface is necessary to determine the existence or absence of a syphilide or of characteristic or suggestive cicatrices. The mouth, throat, and nasal passages also must be examined. Should the symptoms raise a doubt in the examiner, and he be unable to decide the point to his full satisfaction, the opinion of an expert specialist must be secured.

A syphilitic woman cannot give milk duly nutritious; and there is almost a certainty that the child will become infected through some syphilitic lesion, it may be of the nipple or the breast, or of some other part of the person, as of the lips, the tongue, etc.

As it would be criminal to furnish a syphilitic wet-nurse for an infant, I agree with Parvin that it would be also criminal to secure a wet-nurse for a syphilitic infant. In Prussia the latter is punishable by a special law.

An important aid in determining the presence of syphilis is the examination of the infant.

It must be remembered, however, that at birth the offspring may present no evidences of syphilis, but, on the other hand, may at that time be robust though syphilitic. Should, however, the child be three or four months old, and there have been in it at no time any evidence of syphilis, it may be decided that the child probably is not infected with that disease.

Yet a healthy infant does not furnish proof of a non-syphilitic mother; for she may have acquired syphilis during the latter part of her pregnancy, in which case the child may escape infection; or she of course may become syphilitic after her delivery.

A syphilitic offspring should certainly lead to the rejection of one presenting herself as a wet-nurse, without considering the unestablished theory that a healthy mother may give birth to a syphilitic child and herself remain healthy.

A healthy offspring merely furnishes no evidence against the mother as to her infection with syphilis; and we must still scrutinize the woman with the greatest care.

Acute or chronic non-syphilitic exanthemata are contra-indications as to the fitness of the wet-nurse in proportion to their severity and character. One who has epilepsy or who has been epileptic must not be accepted. There is evident danger that during an epileptic seizure the child may be injured, and it is also not improbable that the milk furnished by such a woman is not only insufficiently nutritious but also that it may impart a nervous diathesis to the child. One with a family history of insanity should not be employed, nor one who has been insane. It is well known that the insanity of lactation is especially apt to develop in those having a predisposition through inheritance, and it is possible that the liability to insanity may be transferred through the milk to the child wet-nursed.

The hypochondriacal woman should also be rejected. She cannot furnish the best milk, and hypochondria may eventuate in insanity under the strain of lactation and of separation from her own child, or in case of its death.

All acute diseases, unless trivial in character, and whether contagious or not, render the woman unsuitable.

Pregnancy, of whatever duration, renders the woman unfit, because very frequently the consequent alteration in the character and the diminution in quantity of the milk render it decidedly insufficient and deleterious.

If she menstruates, the milk is usually so altered at the period as to disagree; and a menstruating woman should not be engaged unless it is known that her milk remains good during the period or the demand for a wet-nurse is exceedingly urgent. I have repeatedly seen a nursing child made ill by the milk of its mother taken during the menstrual flow. In fact, in several instances it has occurred under my observation that a child, that had thriven admirably on its mother's milk prior to the return of menstruation, became fretful and subject to attacks of indigestion, and lost in weight, both during the menstrual and the intermenstrual period, indicating not only that the milk of the menstrual period had materially altered, but also that the intermenstrual milk no longer agreed with the child.

Nature has not intended that pregnancy and lactation or menstruation and lactation should coexist.

Abnormal Conditions of the Genitals.—The applicant for the position of wet-nurse may deny the existence of any symptom of genital disease. Yet a skilful questioning may secure the needed information. It will be safer, however, to insist upon an examination. One must look for the scar of chancre, although this not infrequently will elude observation. Chancroids and vegetations are positive contra-indications.

A microscopic examination may reveal the gonococcus, if gonorrhœa exists. Gonorrhœal tubal disease is a decided contra-indication, even though evidences of existing vaginal or urethral gonorrhœa cannot be ascertained.

There is in such a patient an actual risk that the infant may become infected. Even a latent gonorrhœal septic or catarrhal salpingitis may become at any time an active inflammation and render the woman bed-ridden.

Of course the discovery of hydro-, hæmato-, or pyosalpinx, or of an ovarian cyst, or of fibro-myomata, or of sarcoma or carcinoma, should lead to the woman's rejection. Indurated inguinal glands or cicatrices in the groins must be looked for, and their relation to chancre and chancroids borne in mind.

A protracted lochial flow indicates usually subinvolution, with or without some other lesion, such as laceration, ulceration, or polyp. Such conditions render the woman unfit in proportion to their effect upon the general health.

The woman who refuses to submit to an examination of the genitals must be declined. The breasts must be examined, to determine their capacity for the formation of milk and their fitness for giving suck.

The well-shaped breast of the primipara is conical, and does not drag. If a multipara, the breast hangs somewhat downward as a result of previous nursings. A large breast may be merely a mass of adipose tissue, with but little of the true tissue of the mammary gland in it. When kneaded with the fingers, the large mammary gland gives a sensation of greater resilience, and the lobules may be recognized. The breast that consists chiefly of adipose tissue diminishes but little in size as the child nurses, whereas the mammary gland furnishing a good supply of milk becomes decidedly smaller and less tense after the child has emptied it. The latter breast also enlarges and becomes more tense at the expiration of two or three hours. The breast must be examined for fibromata, cysts, carcinomata, and tuberculosis. The contagiousness of carcinoma and of tuberculosis of the breast through the milk is at least so probable that no risks should be taken. Lancereaux describes a diffused and a circumscribed syphilitic mastitis. The diffused form is usually bilateral, and consists of an indolent induration without discoloration of the skin, almost painless, but attended with enlargement of the axillary glands.

The circumscribed or gummatous form may exist either in the superficial fascia or in the gland itself. It is of slow growth, firm and somewhat lobulated, produces but little pain, and may not be attended with enlargement of the axillary glands. Softening may occur, and, after ulceration, the débris escape. With any syphilitic lesion of the breast other than the primary, a careful scrutiny of other portions of the body will probably furnish corroborative evidence. Neuralgia and hyperæsthesia of the nipple or breast may exist as a late result of syphilis: a hyperæsthetic or neuralgic condition of a non-syphilitic character may be present and of itself render the woman unfit to become a wet-nurse. A not infrequent site of syphilitic ulceration is the under surface of a large and pendent breast.

The nipple may present syphilitic fissures or ulcerations. Even if the mother should have escaped infection prior to and during pregnancy, she may contract a primary sore on the nipple or breast from a syphilitic lesion of her child, such as a mucous patch of the mouth, or a fissure of the lip. Any syphilitic lesion of the breast, whether primary or secondary, the latter especially if moist, is liable to infect the child wet-nursed.

Tuberculosis of the breast not infrequently escapes observation. The most usual forms are the cold abscess and the chronic fistula. A disseminated form exists in which the nodules are of various sizes and are hard to the examining fingers. They are liable to caseous degeneration and softening, or to calcification. In this variety the breast is but slightly enlarged from the deposits, and may be movable over the ribs. There is a confluent form of mammary tuberculosis in which the swelling is more marked. Nodules can be felt as irregular, somewhat lobulated, and, it may be, immovable masses. Fistulæ are liable to occur. A true miliary form may exist as an early manifestation.

In the disseminated variety the nipple remains quite unaltered. Palpation reveals the nodules. The local tuberculous deposits may or may not be associated with constitutional evidences of tuberculosis. Kolessnikow's investigation shows that there is actual danger of infection when the milk of a tuberculous breast of a cow is used; and the possibility of such infection occurring from the putting of an infant to a tuberculous breast should be most carefully avoided.

Mastitis and cicatrices, or indurations with a history of previous inflammation, render the woman unfit. It is very rare that a breast in which parenchymatous suppuration has once occurred is ever again entirely trustworthy. The nipple should be neither too large nor too retracted: if too large, a feeble child cannot draw it; a depressed nipple is not only suckled with difficulty, but is liable to become fissured and ulcerated, with the consequent risk of mastitis developing.

The quantity of milk furnished may be judged of by the extent to which the breast diminishes in size when the child suckles, and also by noticing the degree of distention at the expiration of two or three hours after the suckling. The trickling of milk from the child's mouth, the act of swallowing, and the satisfied manner in which it remains at the breast until falling asleep after twenty or thirty minutes, aid in determining the quantity and the character of the milk. A healthy, well-developed, and vigorous child of a few weeks or older indicates that the milk is abundant and of good quality. Still, it must be remembered that a syphilitic child may present the appearance of health during the first few weeks. The quantity of milk taken at each nursing may be determined more accurately by weighing the child before and after it has taken the breast.

Good human milk has an alkaline reaction, is of a dull-white color, and has a specific gravity of 1032. The microscope shows a large number of medium-sized fat-globules. According to Bouchut, if a drop of the milk is added to one hundred drops of a one-per-cent. solution of sodium chloride, and a drop of this be placed under the microscope, each square millimetre should contain from eight hundred thousand to one million milk-globules. Pressure on the breast even after the child has nursed should cause the flow of a few drops of milk. It is not necessary that the child of the wet-nurse should be of the same age as the one to be nursed. It is usually better merely that the wet-nurse's child shall be several weeks old. Women of twenty-five to thirty-five years are to be preferred.

The diet of the wet-nurse should be generous, and any article known to be nutritious, easily digestible, and easily assimilable may be allowed. That diet which tends to the preservation of vigorous health in the woman will lead to the formation of the largest supply of nutritious milk. An excess of meats must be guarded against if the accustomed amount of exercise is no longer partaken of. Such things as occasion flatulence or other evidence of indigestion must be avoided. Tea must not be drunk in excess. Milk taken during the meals is advantageous. Beets have been recommended as peculiarly serviceable in procuring an abundant flow of milk.

It will be sometimes advantageous to give a moderate amount of porter or other malt liquor; but the liability of wet-nurses to become intoxicated must be remembered always. An increase in the amount of liquids taken tends to increase the amount of milk secreted, but the liquids should be of a nutritious character, such as meat broths, gruels made with milk, and milk itself. It will rarely be advisable to resort to stimulants.

The nurse should be required to take a considerable amount of out-door exercise. The sleeping-apartment should be well ventilated, and not too greatly heated. The normal action of the bowels must be secured, and abundant ablutions exacted. The child should sleep in a crib, not with the wet-nurse, and the mother should be always on the alert that the wet-nurse does not give an anodyne in some form to the infant.

In Prussia there are special laws bearing on the relations existing between the employer and the wet-nurse: but I know of no such special laws in this country.

DIET AFTER WEANING.

BY SAMUEL S. ADAMS, A.M., M.D.

WEANING may be defined to be the period of infancy when the child is deprived of breast-milk, and such changes are made in its alimentation as are rendered necessary by its independent existence. The time of weaning cannot be arbitrarily fixed at the same age for all infants. There is no uniform opinion as to the exact time for weaning, although most authorities assert that it should take place between the twelfth and eighteenth months. Under normal conditions the infant should not be weaned before the twelfth month, nor should lactation be continued after the eighteenth month. In ninety-one observations made by the writer but four were found nursing after the first year. There is such a general conformity between dental evolution and age that weaning usually takes place at the evolution of the eight incisor teeth, which is completed about the twelfth month.

For convenience it is necessary to assume that the child has been deprived of the breast-milk at the twelfth month, and to formulate a dietary accordingly. The eruption of the lower central incisors, during the seventh or eighth month, seems to be the indication to mothers to begin supplemental feeding. Very few infants pass far beyond this physiological epoch without it.

During the period of dentition developmental changes gradually take place in the digestive apparatus which fit the child for an independent existence. The glandular structures become more active, and the muscular tonicity increases, so that at the period of eruption of the anterior molars the alimentary tract is prepared for semi-solid food.

The object of this paper being to prescribe a suitable dietary for the child, in health and disease, from weaning to puberty, it will be best attained by making divisions to conform to the recognized anatomical and physiological changes in the organism. The following divisions seem, therefore, to meet all the requirements: 1, twelfth to eighteenth month; 2, eighteenth to thirty-sixth month; 3, third to fifth year; 4, fifth to eighth year; 5, eighth year to puberty.

While most mothers will appreciate the value of milk as the chief food for infants during the first year, very few will be convinced of its value as such after weaning. Several months before the child is weaned, in many instances, it has had some of the farinaceæ, and also, probably, meat broths. If weaning takes place before the eruption of the molars, the diet should be milk. If it is weaned during the summer months, milk should be its only food, although the molars and, perhaps, the canines have appeared. If, however, the child does not seem to derive sufficient nourishment from the milk, it may be given some additional food, provided the weather be cool, but always remembering that its chief constituent must be milk. If it seems to thrive on milk alone, it will be advisable to limit it to it until the eighteenth month. It is the exception, however, when a child will be satisfied with milk until this late period. It is generally necessary to supplement its food by adding some farinaceous aliment. If there is a tendency to loose bowels, barley-water is preferable. It is made by grinding a tablespoonful of the grain barley and adding to it six ounces of water: this should be boiled for fifteen or twenty minutes, salt added to suit the taste, and the mixture strained. This decoction should be made fresh twice a day and kept cool. It should be added to the milk in the proportion of one to three or one to two. The prepared barley may be used in the same manner, but it is not so reliable. If constipation is the rule, oatmeal may be used by preparing a decoction similar to that of the barley. Arrow-root should not be used, on account of the large proportion of starch it contains.

Bread jelly has been highly recommended by Churchill and others as an excellent food for children just after weaning. It is made by taking a quantity of the soft part of stale bread, breaking it into small pieces, covering it with boiling water, and allowing it to soak for some time. The water is then strained off, and fresh water added. This should now be boiled until it becomes soft; the water is then pressed out, and the bread on cooling will form a jelly. A portion of this should be mixed with sweetened milk.

In some cases beef tea will be well borne. That made in a bottle swimming in a water-bath does not contain soluble albuminoids. It contains large quantities of salts, and should not be given when there is a tendency to diarrhœa. Excellent beef tea is made by mincing one pound of lean beef and adding a pint of cold water and ten drops of dilute hydrochloric acid. This should stand for two or three hours, with occasional stirring. It should then be left to simmer for fifteen or twenty minutes, when it will be ready for use.

Beef broth is not very nutritious, and is not recommended. Mutton, veal, and chicken broths are nutritious, and are applicable in many cases. It must be borne in mind, however, that mutton causes constipation, and veal diarrhœa.

Cow's milk is that most generally used in feeding infants. We should not delude our patients with the idea that they can secure *one cow's milk*, because that will not be done if the dairyman has more than one. He may promise to keep it apart, and will accept an additional price for doing so, but he will more than likely deliver a part of the "general milking" in the can that has been provided for the *one* cow's milk. If the dairyman has but one cow, a thorough examination into its keeping may disclose that it is not the best. The cow may be kept in a small, badly-ventilated, and foul stable; it may scarcely ever run at large or browse, and probably its food will be mainly swill; though, even without exercise or browsing, if it is fed on long food and brans, with an occasional feed of fresh green grass, it may furnish a good quality of milk. Again, the particular cow may be a sickly one, but the milkman will not let it be known so long as he is receiving an extra price for its milk. If we are sure of getting good, sweet milk, twice daily, from properly-fed cows, let us be satisfied. Probably a great many more children would be saved if more attention were paid to the preparation and dispensing of milk. Unmethodical and irregular feeding is quite as bad as feeding with improper aliments. The child should be fed regularly with enough milk to satisfy its appetite; but giving milk to appease its anger should be positively prohibited. The quantity must necessarily be increased as the child advances, but due regard should always be paid to its digestive and assimilative powers. Overloading its stomach impairs its digestion.

The most satisfactory general rule is to secure good sweet milk from a country dairy, delivered twice a day if possible. As soon as it is delivered, pour on the requisite amount of boiling water to scald it; put this in the refrigerator, to be used when required. Until the fifteenth month at least, the milk should be given from a bottle, to insure steady feeding; after this it may be given from a cup or glass. Do not permit the bottle to be used as a soothing apparatus; when thus employed it does harm. Never let the child sleep with the nipple hanging to its lips. It should be fed not oftener than once in four hours. With every feeding add a tablespoonful of lime-water or from one-half to one grain of the bicarbonate of sodium. When it is through feeding, throw away the remaining portion, never allowing it to stand in the bottle. Scald the nipples, tubes, and bottle, and keep them in a solution of soda until the next meal. The simplest and most conveniently cleansed feeding-bottle is always the best.

Of the various substitutes for breast-milk, condensed milk is probably the most extensively used. Owing to its apparent cheapness, and its ease of preservation and preparation, it is a popular food with the lower classes. Very many cannot afford to purchase cow's milk, and cannot spend the time necessary for its preservation and preparation, and, consequently, feed their children on this unstable article. The weight of authority is against the use of condensed milk, owing to the lack of nutrient ingredients. Children fed with it will grow, but are deficient in muscular vigor. Under some circumstances we may be compelled to use it. During very warm weather, when poor people cannot buy ice to keep cow's milk, or when infants are travelling, and it is impossible to obtain sweet cow's milk, it may be advisable to use it; but its use should never be sanctioned when good cow's milk can be secured.

The writer's experience with peptonized and pancreatized milk has not been favorable. It cannot be recommended as generally as was at first supposed. These methods have given way to the sterilizing process, which is by no means new.

The writer desires to enter a protest against the use of the various "infant foods" as substitutes for or aids to cow's milk. The agents employed in introducing them to the medical profession are skilful in pointing out the advantages each possesses over the others. The manufacturers resort to many artful devices to increase their sales. The physician is constantly annoyed by confidential letters asking him to try this food which has been so successfully used by the professional gentlemen whose names they exhibit on the fancy cards, calendars, and books. Mothers are attracted to them by the warnings posted in the street-cars, and the pictures of plump, rosy babies distributed by the druggists. Analyses by competent and honest chemists—not paid by the manufacturers—have shown them to be rich in the ingredients they are guaranteed not to contain and to be deficient in those which are lauded as being present in larger proportion than in any other food. Rotch, in a valuable paper,[1] discusses the merits of the different "infant foods," and demonstrates their unreliability as substitutes for milk.

With the sixteen teeth the child should be allowed a more liberal diet. Its digestive apparatus is now capable of digesting food which has been masticated. It may be allowable to give it stale, well-cooked bread, and butter, or crackers. It may also be given a little mashed white potato, with gravy. A sandwich of scraped lean beef and bread, seasoned with salt or sugar, will be relished, and is very nutritious. It may have a piece of rare beef or a chicken-bone to suck, care being taken that it does not swallow the pulp or bone.

Peptonized beef preparations have been recommended by the recognized authorities.

In regulating the regimen of a healthy infant during this period, very little change is required in its food. It should be fed five or six times, at the same hours, every day, but should not be awakened for the purpose. If it desire its food before its accustomed time, it should have it.

First meal, 6 A.M.—A cup of milk, with cream biscuit or a slice of buttered bread.

Second meal, 8 A.M.—Stale bread, broken and soaked in a tumblerful of rich milk.

Third meal, 12 M.—A slice of buttered bread, with about half a pint of weak beef tea or mutton or chicken broth.

Fourth meal, 4 P.M.—A tumblerful of milk, with crackers or a slice of buttered bread.

Fifth meal, 8 P.M.—A tumblerful of milk, with bread or crackers.

---

[1] Archives of Pediatrics, vol. iv. No. 44, p. 458.

Towards the latter part of this period, when the child has sixteen teeth, it may be desirable to substitute the following:

First meal, 6 A.M.—Bread or crackers, with a half-pint of milk.

Second meal, 8 A.M.—A tablespoonful of oatmeal, cracked wheat, or corn-meal mush, with milk, and a couple of slices of buttered bread.

Third meal, 12 M.—Bread-and-butter, milk, and a soft-boiled egg.

Fourth meal, 4 P.M.—A piece of rare roast beef to suck; mashed boiled potatoes, moistened with dish-gravy; bread and milk; and a small portion of rice, bread jelly, or farina.

Fifth meal, 8 P.M.—Milk and bread or crackers.[1]

This is a modification of the diet laid down by Louis Starr; but the writer usually insists that the infant should be confined to milk, milk and barley-water, or milk and oatmeal-water, during this entire period. When his advice has been followed, the perils of the "second summer" have been avoided.

Fruits and berries of all kinds should be interdicted.

Every case of infant-feeding must be regulated by its own indicated requirements. There is no uniform rule applicable to all. Each must be studied carefully, and that mode of feeding must be adopted which proves best suited to it.

The child should not be permitted to sit at the family table. It may have a separate table, where it can have its frugal meal without being tempted by unwholesome dishes.

The diet in sickness during the first period must be regulated by the nature of the case. It is impossible to prescribe a regimen suitable to all sick children.

Vomiting is unquestionably the most frequent symptom to be controlled. It may be due to overfeeding, or to some fault in the quality of the food. When it is caused by overfeeding, a diminution in the quantity of food, as well as a longer interval between meals, will usually correct it. If it should be caused by a defect in the quality, this should be discovered and remedied. If the ejected matter is sour-smelling, the alkali must be increased. Frequently, forced abstinence will correct it; and in many cases small quantities of food given every fifteen, twenty, or thirty minutes will have a salutary effect.

Diarrhœa is often the result of improper feeding. The food may be too concentrated, or its quality may be poor. When it is due to too much solid food, the indicated treatment is to confine the patient to a liquid diet. If the quality of the food is not good, it should be improved. In many cases the addition of barley-water to the milk will prove effectual in checking the diarrhœa.

---

[1] Often it would be preferable to give the fourth meal at three P.M., and the fifth meal at six P.M., especially in winter, so that the child can be put to bed by seven o'clock.—Ed.

Constipation may often be corrected by adding oatmeal to the second meal, or oatmeal-water to the milk.

It should be the invariable rule to confine children to a liquid diet as soon as any impairment of digestion or assimilation is noticeable or they become ill. Milk should always have the preference. It may be given pure, diluted, boiled, or, perhaps, predigested. In rare instances milk will not be retained by the stomach, or will be passed from the bowels only partially digested. In such cases a mixture of equal parts of milk and lime-water, given in teaspoonful doses every ten or fifteen minutes, will not infrequently be retained and digested. In some cases where milk cannot be retained, barley- or rice-water may be temporarily substituted. In other cases beef tea, beef essence, or beef juice may be administered in small quantities, frequently repeated, with marked benefit. Tea and coffee should not be allowed.

In weakly children the following may be given:

Chicken jelly.—Clean a fowl that is about a year old, and remove the skin and fat. Chop it, bones and flesh, and put it in a pan with two quarts of water. Heat slowly, and skim often and carefully. Let it simmer for five or six hours; then add salt and mace or parsley to taste, and strain. Set away to cool. When cold, skim off the fat. The jelly is usually relished cold, but may be heated. Give this in small quantities, very often.

Wine whey.—Boil three wineglasses of milk, and add a wineglass of sherry or port wine. Strain, and add a wineglass of warm water. A wineglassful of this may be given once or twice a day.

White wine whey.—To half a pint of boiling milk add a wineglassful of sherry; strain through a fine muslin cloth, and sweeten. A tablespoonful of this may be given every two or three hours.

It is quite as important to regulate the diet of the second period as that of the first, but much more difficult. At this period the child is walking, and often helps itself to indigestible substances. It now has all its milk-teeth, and is capable of mastication. Its mind is generally sufficiently active to be taught what edible articles it should have. Its power of masticating, its flow of saliva, its good digestion and assimilation, and its increasing bodily growth demand a greater variety of food. If it reach its second period during the summer, and have the appearance of health, and seem satisfied with its milk and simple food, it will be prudent to wait until cool weather to change its diet to a more substantial kind.

It is now admissible to allow it to eat at the family table, because the opportunity to begin its training early should not be overlooked. It can be taught to eat slowly, that certain articles are not suitable for it, and that it can have enough of the proper kind of food. When a child frets for different articles of food on the table it is generally because some imprudent person has allowed it to taste them. If it is not tempted by tasting other, it will be contented with its simple food. It should be fed at least four times daily, and occasionally will require a few crackers or a slice of bread-and-butter between meals.

First meal, 8 A.M.—A portion of well-cooked oatmeal, wheaten grits, or corn-meal mush, with a liberal supply of milk; cold bread-and-butter; and a piece of finely-cut, tender beefsteak, or a soft-boiled egg.

Second meal, 12 M.—A bowl of chicken or oyster soup, or weak beef tea; milk, with bread or crackers, and butter.

Third meal, 4 P.M.—Roast beef, mutton, chicken, or turkey; fresh white fish; mashed white potato, moistened with gravy; bread-and-butter; and rice and milk.

Fourth meal, 8 P.M.—Milk, with bread or crackers.

It may be necessary to give a glass of milk and a piece of bread-and-butter between the first and second meals; and if the child is particularly hearty the same may be occasionally required in the early morning. Towards the latter part of this period fresh ripe fruits are admissible, provided due care is taken to prevent the ingestion of seeds and rinds. A popular fruit is the banana; but the writer's experience has been such that he considers it more productive of eclampsia than any other fruit, and consequently he cannot recommend it.

The meal-hours vary in different communities, so that those for children will be governed by the local customs. It may be necessary to give the principal meal earlier than four P.M. It must be remembered, however, that most children sleep the greater part of the afternoon, so that if they eat dinner at two o'clock they will be asleep during the digestion of the bulk of the day's solid food; on the other hand, if the meal be at four o'clock there will be active exercise after it to aid digestion and assimilation.

It will need constant watching to prevent it from obtaining unsuitable food.

Frequently the neuroses, as eclampsia, "night-terrors," petit mal, and the numerous symptoms attributable to "worms," may be directly traceable to the presence of indigestible food in the alimentary tract. A brisk purgative seldom finds the "worms," but generally allays the excessive exaltation of the nervous system by removing the offending material.

When the child is suffering from an acute disease, its diet should be limited to milk and beef tea. In chronic ailments, or in protracted convalescence from acute disease, each case must be treated by its individual requirements, while good judgment will render valuable assistance in the selection of those foods which are easily digested and which possess the maximum quantity of nutritious matter to the quantity ingested. In sickness, tea and toast are favorite articles, but only load the alimentary tract with innutritious matter.

During the third period—from the third to the fifth year—the difficulty of regulating the child's diet will be great. It has now reached the age when its friends will humor it with knick-knacks and table-food of difficult digestion. It has twenty teeth, and its friends cannot understand why it should not have such food as a healthy adult can digest. A devoted mother, or usually grandmother, will argue that all her children, at this age, were fed from the table and were not injured. Such children lived in spite of mismanagement. Granting that its diet must be more liberal at this age, it must still be restricted, for even now the presence of indigestible or undigested food in the alimentary tract may be productive of reflex nervous disturbances.

Its activity and waste and repair demand an increase in the quantity of nutritious food. Three substantial meals a day will usually suffice, but occasionally a snack between meals will be required. While it is well to apply the rule of regularity, it is not always prudent to enforce it, especially if the child is hungry. The practice of children running to the pantry and helping themselves should be discouraged. In such cases children do not eat enough at the regular meals.

It is impossible to lay down "a bill of fare" for this period, but a frugal meal can be selected from the following:

BREAKFAST.

Corn-meal mush; oatmeal; wheaten grits; hominy; with plenty of cream.

Potatoes, baked and stewed.

Eggs, poached, soft-boiled, and omelet.

Fish, fresh, broiled.

Meats.—Beef hash; broiled steak; stewed liver and kidney; lamb-chops; chicken fricassee.

Tomatoes, sliced (occasionally).

Bread.—Cold, light; Graham; entire wheat; corn; muffins, plain and Graham (occasionally); corn and rice cakes.

Fresh ripe fruit may be given in moderate quantity.

Highly-seasoned food must be avoided.

LUNCHEON.

Soups.—Oyster; bean; chicken; consommé.

Vegetables.—Potatoes, baked and stewed; sliced tomatoes.

Meats.—Beefsteak; lamb-chops; cold lamb or mutton.

Bread.—Cold rolls and soda-crackers.

Fruits in season.

Rice and milk.

DINNER.

Soups.—Consommé; noodle; oyster; cream of barley; potato; chicken; and chicken stew.

Fish.—Fresh, baked, broiled, and boiled.

Meats.—Beef, chicken, lamb, and mutton.

Vegetables.—Potatoes, rice, cauliflower, macaroni, peas, tomatoes, beans.

Bread.—Well-cooked wheaten.

Desserts.—Rice and milk; light puddings; ice-cream occasionally.

Fruits and berries in season (fresh and sound).

The regimen of the sick during this period does not differ very materially from that of the preceding, except that, generally, a more generous diet may be allowed. If the illness be of a nature demanding liquid food, the principles already set forth will be applicable. In all cases of illness the food should be reduced in quantity and changed in character, although the patient may not be confined to liquids. As soon as the appetite becomes impaired, the child should be put upon a simple diet. Frequently, in children of this age, too much or deteriorated fruit will cause digestive disturbances. Withholding the fruit for a few days will usually effect a cure. The child should always have its fruit selected for it. When sick, knick-knacks, jellies, and fancy dishes should be forbidden. If the illness be protracted, and the food be digested and assimilated, it should have the most nutritious aliment. This rule is especially applicable to scrofulous, syphilitic, rachitic, and tuberculous children. We need not wait for the manifestation of these diatheses. If there is good reason for suspecting their presence, the sooner the select diet is begun the better; and, even if they are not latent in these children, the care in feeding will prove beneficial.

New troubles seem to arise during the fourth period which require close vigilance over the child's dietary. At this time the milk-teeth begin to decay, and the first of the permanent teeth make their appearance. The child has frequent attacks of toothache, the dread of which prevents him from properly masticating his food. Consequently, indigestion and diarrhœa, from bolting food, are of frequent occurrence.

Again, the child is old enough to be indulged by its parents with everything they eat: hence the impossibility of restricting the diet as long as it is healthy.

It is advisable to select its food from the articles recommended for the third period, with the addition, perhaps, of game, corn, string-beans, sweet potatoes, lima beans, hot bread and cakes, and light custards and puddings.

In sickness the general rule of restricting the diet according to the nature of each individual case is also applicable.

The physiological changes which take place during the fifth period would seem to warrant the statement that extraordinary care should be exercised in regulating the child's regimen between the eighth year and puberty.

The ingestion of highly-seasoned or very rich food may unduly excite the passions and pervert the physiological phenomena of boyhood and girlhood. It is also apt to cause lascivious dreams and sexual excitement.

The rules governing the dietary during sickness are similar to those for adults.

The use of wines and beers should be prohibited, and that of tea and coffee discountenanced.

In discussing the diet for children in the preceding pages the writer has not lost sight of the fact that some regard should be paid to the important factor of the circumstances of life. It is well in prescribing a regimen which has stood the tests of the laboratory to remember that such advice is given to a large number who are not able to incur the necessary expense of typical feeding. To prescribe such food as that hereinbefore recommended for the child of the laborer, whose wages are scarcely adequate to support his large family, would entail hardships on those whose affections are strongest for the weak and afflicted. The expense necessary to obtain cream, milk, and milk-sugar will not be considered by people of even moderate circumstances, but will be difficult for the mechanic and impossible for the laborer. Therefore it is important in selecting a food for children, either well or ill, in the lower walks of life, to recommend that which will be healthful and of reasonable cost.

If the following good advice is impressed upon the nurse, the success of treatment may be greater:

"Never give re-cooked meats, fish, or vegetables to an invalid, and cook only small quantities for him. Simplicity, variety, and healthfulness are the things to be considered in preparing food for the sick. What is good for one person is frequently injurious to another. One must not become impatient or discouraged because the invalid is changeable in his tastes.

"The eye as well as the palate of the patient is to be considered. The tray always should be covered with a fresh napkin; the china, glass, and silver should be the daintiest the house affords.

"Only a few things should be served at a time: it is better that the patient should think that he has not had enough to eat, than that he should lose his appetite on the appearance of a large quantity of food."

NURSING OF SICK CHILDREN.

BY MISS CATHERINE WOOD.

THERE can be no doubt that in this department great strides have been made during the last twenty years or so, and perhaps this improvement may be entirely set to the account of the children's hospitals which have now sprung up everywhere. The children are important little people, and set so much store by themselves that they demand study and thought on the part of those who would understand their treatment; and all who have had experience in the care of sick children will admit that a special education and training are required for those who aim at nursing them successfully. To a casual observer passing through a ward of sick children they may seem all much alike,—alike in their restlessness, in their weariness, in their perpetual demand on the patience and care of their attendants,—and yet to the practised eye there is every shade of difference in that row of cots; the diversities of character are as strongly marked as among adults; even the little babes differ one from the other. In the study of these idiosyncrasies, and in the adaptation of means to an end, the real child's nurse will at once declare her aptitude for her task; she will see at once that though her duties should be performed methodically and with regularity, each child must be the subject of special study, and rules and red tape made sufficiently elastic to cover all.

It is curious to review how by degrees the little sick ones have been forcing themselves to the front. At first they were grudgingly admitted into the wards of a general hospital, and then the nurses and patients only tolerated them; but now, as in a well-ordered house, the children have their own department and their own attendants, their special nurses, and harmonious arrangements, or they may be promoted to the dignity of their own hospitals: whatever may be the plan pursued, the children have trained their nurses and instructed their doctors, to the manifest advantage of all parties. There can be no question that many a sick child in its own hospital, or ward, is infinitely better circumstanced for its recovery than in its cot at home. A child is very sympathetic and receptive, and when one among a number of other sick children, receiving only a portion of a nurse's time and attention and being acted upon by the various harmonious influences around, its physical and vital energies are aroused and directed to a healthy action, it is taken out of itself, and its mind is diverted from its ailments, a result most satisfactory for either adult or infantile sick; moreover, it is saved from the inevitable spoiling that is the natural end of home nursing, or it is rescued from the mischievous and sometimes fatal indulgence of its whims and fancies; it is tided over the fretful stage of convalescence almost unnoticed, and returns to its home a little hero, fit to take its usual place in the family. The sad experience of every doctor who has had to treat a spoiled child at home will confirm this: the child refuses to take its medicine or food, and the united efforts of the parents and household are useless; it remains master of the situation, a hardened little sinner. Or some particular posture must be maintained to allow an inflamed joint to recover itself; it screams and kicks at all efforts to place it aright, it frets if fastened down, and refuses its food, until at last it is allowed to lie as it likes, and valuable time is wasted. Place that same little one in a children's hospital in the hands of doctors and nurses accustomed to children, and it at once becomes tractable; all friction and contests are avoided; it follows the example of the next cot, and swallows medicine and food quietly; it submits to be laid down, for it seems the fashion of this nursery and so must be right. Then the atmosphere of play and merriment carries it over the tedious hours of a chronic illness almost unnoticed. Dr. West, who was certainly the pioneer in initiating a specialty in the treatment of sick children, says in his opening lecture to students, "Children will form at least one-third of all your patients; so serious are their diseases that one child in five dies within a year after birth, and one in three before the completion of the fifth year. These facts, indeed, afford conclusive arguments for enforcing on you the importance of closely watching every attack of illness that may invade the body while it is so frail." If the medical attendant is being educated by his patient, the nurse also is its pupil; for the child will not be nursed by any one, it is as elective in its tastes as the most experienced valetudinarian, and those who aim at nursing sick children must have the art of winning the child's love and confidence at the onset. Then on this the special training may be based, the power to observe, to interpret aright these observations, to understand and anticipate the wants of the patient, to comprehend the emphatic but unspoken language of the aspect, manner, cry, posture, etc., of sickness; it must be the first object of the nurse to learn these, or she will fail in her task; and she must also bring to her aid invincible patience, gentleness, cheerfulness, good temper, and self-restraint. She will not only have to learn how to feed a refractory child, but she must grasp the method and science of giving food so as to sustain the strength and yet not overtax the powers; she will have to adjust her foods to the most irritable stomach as well as the rebellious one; and above all she will have to steel her heart to the pathetic petition for indulgences or treats. She must also learn how to combine firmness with gentleness, how to insist without coercion, how to win obedience without friction, how to take her patient along with her; and all this can be accomplished only by love and truthfulness. Once win the child's trust, and then it will yield itself a willing slave. The most fearless truthfulness should be insisted on from all those who have to tend the sick child, even when it wrings the loving heart to speak the truth. The pain the child will feel will be far less than upon finding itself deceived, especially when among strangers.

It is very touching when among sick children to see the quiet and contented way in which they lie in their cots, thankful to be only let alone; and it is this letting-alone which is so important in nursing a sick child. The less the child is handled the better. The poor mothers in their own homes make quite a toil of their children; they will hardly put them out of their arms, and they certainly will not believe that the child can be thriving unless they are dandling it on their knees; both are quite wearied, mother and child. But this is all mistaken kindness. A sick child will thrive best if laid quietly in its own cot, so that the fresh air may play around it, and that it may rest. Its little face will soon lose the worried look that is so often marked on the faces of the children of the poor, and a look of happiness and content will take its place. It is not difficult to accustom the children to lie quiet; at first they will be restless and fret at not being taken up, but when they see that their frettings are of no avail, with the ready adaptability of childhood they make the best of it, and soon find how much the best it is.

It is most essential in the care of sick children that they should be supplied with plenty of light and fresh air, in neither case pouring in directly on them, but flooding them all round in generous profusion. They by no means appreciate the darkened room and hushed voice: like the plants in the garden, they expand under the rays of light; and there can be no doubt that the light has a physiological influence on their growth and development, especially so in the case of illness. Therefore in arranging the sick-room, let it have as much of the light as possible, a southern or western aspect, and a free circulation of air through it, by maintaining an interchange with the outer air without making a draught; and this should be kept up by night as well as by day, especially in crowded cities. Of course in certain fever cases, in acute diseases of the brain or diseases of the eye, a darkened room may be required.

I. THE SICK CHILD.

The sympathetic nature of the child is at once affected by any deviation from the standard of health; its organism is like a delicate machine, disturbed by the presence of a minute grain of sand; it at once gives token that there is some morbific influence at work. The severe onset of an illness, its rapid course, and its speedy termination either in recovery or death, are always matters of surprise to those unaccustomed to sick children; and so it requires that the attendants should be fully on the alert to catch each new symptom, give it its value, and be prepared with appropriate treatment. It is hardly a safe course for the mother to wait until her child's illness has declared itself before she takes action; and even then, as a mild domestic ailment and an acute disease may alike assume the same symptoms, she would act more wisely to seek some skilled assistance, for, however experienced a mother may be, she can hardly read symptoms aright. Or it may be that one of the infantile infectious complaints is declaring itself; and then for the sake of the other children some system of isolation is necessary.

Illness at first shows itself in a child by listlessness and loss of appetite; the eyes look heavy; the child may be fretful, especially if disturbed, or it may be drowsy; it will feel hot, and if the temperature be taken the thermometer will generally show an elevation above the normal; but this in itself must not be regarded with disquiet, as a very little suffices to disturb the normal heat of the body; in nearly all cases there will be vomiting and some bowel-disturbance, and then special symptoms will declare themselves. In the older child, one able to give some account of itself, the symptoms generally set in in the same sequence, and they must be taken as a warning that something is amiss. The best treatment is to wait and see what is coming, at the same time placing the child in favorable circumstances,—that is, keeping it quiet and away from its fellows, giving it light food, of easy digestion, seeing that the bowels are not overloaded, and then waiting for the diagnosis of the medical attendant.

Age has much influence upon the diseases of children, and if it is borne in mind that before the age of seven years the body is being built up rapidly, and this means a great expenditure of vital force, it is more easily understood that a small disturbing cause will seriously upset the equilibrium of its powers. It is of more importance to keep a child in health than to restore it from illness to its normal condition; and very much may be done by regularity in all its habits. Appropriate food at regular intervals will drill the digestive apparatus into strong, healthy ways; regular hours of rest and exercise will soothe and strengthen the nerve-centres; the muscular powers will be developed by use, and the mental faculties develop themselves in harmony with the animal vigor. There are certain crises in the child's life that must always be reckoned with as causes of disturbance,—notably, the period of teething. This is a sea of troubled waves, over which the little bark must be sensibly steered and it will voyage in safety; but then it is a natural process, for which provision has been made in the child's constitution, and if its surroundings and habits are healthful it will pass over the storm with but little danger. Of more serious moment are the hereditary defects that are ever-present dangers to the child life and will break out into flame with the least spark and will modify acute disease by their influence. If the mother has a good knowledge of her child's constitution, she may do much to defend the weak point by engendering a wholesome habit of living. This much is quite certain, that no two children are alike, and that they will thrive the best who receive the most individual thought.

Of secondary importance, but by no means to be forgotten, is the child's nursery: here three-fourths of its day will be spent, and its aspect and traditions will never die out of the child's life. It is of paramount importance that it should be bright, cheerful, clean, and wholesome, that its presiding genius should be a lovable, common-sense woman, and that order and method should rule its habits. The little ones will then look back upon their nursery days as some of the brightest in their life.

It may become the sad necessity to turn this bright room into a sick-ward. In such case, turn all the unnecessary articles of furniture out of the room, take up the carpet, remove the hangings if there are any, and have at hand everything that is likely to be wanted, extra basins, jugs, cups, and feeders, small pans for the linen, a plentiful supply of water and liberal means of making it hot, baths, and a ready supply of linen. Keep all these appliances handy, but outside the room, and also outside have vessels for receiving the slops, so that nothing offensive may be about the patient. Provide a good supply of some disinfectant in a concentrated form, to be readily weakened, and let this be freely used on the floor, in the vessels, and for soaking the linen from the patient. Last, but of no less importance, is the choice of the sick child's nurse. It does not follow that either the mother or the nurse is the most fitting; the one may be too nervous and excitable, the other too indulgent or ignorant: what is wanted is a steady, reliable woman, who can manage the patient with kindness and firmness, who can be trusted to carry out orders and yet have a discretion of her own, cheerful and even-tempered, physically strong in the face of an extra demand on her powers, cool and self-possessed in an emergency, and, above all, with a love for her work and her patient. If added to these qualifications there is hospital training, then the right attendant is found. It is very essential that as far as possible the management of the sick-room should be kept in the hands of one person, so that there may be a unity of treatment and that methodical harmony which is of more importance in sickness than in health; and then, if the assistants are obedient, good work may be done. There are few things more harmful than the fidgety nursing that one so often sees in the family. The nurse, if she shows herself to be a woman of tact and sympathy, will soon infuse her spirit into the members of the family, and they will readily work under her guidance.

There are many little niceties of method and order that form part of the training of a nurse in the hospital wards, that will add to the comfort of the patient. A child with any form of joint-disease or fever is easily washed in the recumbent position on a blanket, being rolled gently from side to side, and in the case of an injured limb it must be steadied with one hand or by a second person, and then there is very little pain or displacement. It is a great husbanding of the strength in fever, especially typhoid, to keep the patient always lying down, and the whole of the person can be properly washed in this way. This is the sovereign preventive of bed-sore, especially in cases of paralysis, where the evacuations are not retained, and enables the nurse to see at once any weakness of the skin. Complete drying of the skin must be insisted on, and the liberal use of dusting-powder, and then a child may lie for months on its back without any ill effects. An important part of the nurse's work is to prepare her patient for a physical examination, and to do this quickly and readily without undue exposure is a sign of good training. It is very irritating to a doctor to watch a nurse fumbling at buttons and strings, and it wearies the child. Before the time of the doctor's visit she should have all the clothing loosened, and a blanket warming at the stove to wrap the child in if it is to be taken out of bed. If the child is to be examined in bed, the night-gown and vest are drawn over the head and placed on the stove to keep warm: this is a little detail, but it is important for a delicate child to be saved the chill of cold garments when exhausted by the examination. A loose wrap will serve to cover the parts not under observation; and if the doctor uses the towel whilst examining the chest, be sure that it is one well aired. If the patient is removed from the bed, the nurse will take the child in the warmed blanket on her lap and be ready to adjust it to the doctor's needs. Some little gentleness and coaxing are required to prevent the child from being frightened, and a few moments of time must be spent in winning the patient's confidence and allaying its fears; and if this is successfully accomplished, the patient will probably look upon the whole as a game for its amusement. Should some operation be necessary without an anæsthetic, it is far better to tell the child that it will be hurt a little bit, and if the instrument be kept out of sight the fright will be very momentary. It is marvellous how patiently children submit to painful remedies if only they are treated with candor. In putting on hot applications it must be borne in mind that the child's skin is more sensitive and tender than an adult's, and that the test of the nurse's hand is not sufficient; the child's sensations must be the guide. It is a cruel thing to put on a fomentation or poultice too hot, and it does no good if it excites the child. If a blister is ordered, it can be put on more efficiently with blistering fluid, care being taken that it does not run, and if put on at night the child will generally sleep through it; the after-treatment will be according to the instructions of the medical attendant. Children are very tolerant of blisters, and in wise hands they are useful remedies. The application of leeches is more complicated: the sight of them must frighten the child, so they should be dealt with quickly and decidedly. The easiest way is to turn the box on to the part, which should be first well washed, and then wait until they have all taken; or take them up in cotton-wool in the mass and hold them on; in either way the child does not see them moving about, and if the cotton-wool is left on they will not be very evident. They must be left until they drop off, and then the part washed, pads of lint or absorbent wool put on, and the whole bandaged up. The nurse must be on the watch for after-bleeding and report to the doctor.

Leeches should not be placed on the prominence over a bone, nor on a vein, nor on any part that receives pressure. The pain is not severe, and the fright is caused more by the sight of them than by their bite. If possible, the child should be kept quiet after the application, or the bleeding may become troublesome.

A very frequent remedy ordered is an enema, either as a medicine in diarrhœa or constipation, or as a means of giving food. Its nature and quantity will be prescribed by the doctor, but its administration will be in the hands of the nurse. Supposing it is to check diarrhœa, it will probably consist of starch and opium, and should be made as small in bulk as possible,—not more than two or four teaspoonfuls of mucilage with the quantity of opium prescribed; if to deal with constipation, it will be large in quantity, such as a pint of soapy water warm, or gruel and castor oil, or soap and castor oil; and after the injection has been given leave the patient quiet until there is a desire to return it. The tube should be well oiled and passed up the rectum gently as far as it will go. In giving nutrient enemata, the food must be made as concentrated as possible, and be a little thickened with starch powder or arrowroot. Four ounces is as much as the bowel will retain with advantage.

A nurse who knows her work will know that she has to put out the urine for testing by the doctor: it should be a small quantity taken from the first passed in the morning, and let it be put aside in a clean vessel and covered over.

It may be that she is instructed to measure and record the amount of urine passed in the twenty-four hours: in such case she must have a suitable vessel, such as a marked jug or glass, provided for her, and then should begin her observations thus. Let her fix on an hour, say nine A.M., at which to take the observation: on the first morning let the child pass water at that hour, and then throw it away: all the water passed subsequently is to be saved, and at nine A.M. the next morning the child is to be invited to pass water, and then the whole quantity is measured, recorded, and thrown away. If the specific gravity is to be taken, the nurse must be shown how to use the little instrument that weighs it, and how to record it.

It is also part of the nurse's duty to examine the evacuations and report on them, and in any case of doubt to save them for inspection. On this point there is a great deal of ignorance and diversity of opinion: one nurse will call that diarrhœa which another nurse will name only "a little looseness," and so on. The presence of slime and blood in the evacuations should be at once reported, and the stool saved for inspection; also the presence of undigested food. The frequency of the action and the quantity must likewise be observed, and intelligent answers given to the doctor's questions.

II. MANAGEMENT IN SICKNESS.

The diseases of young children are so frequently induced by bad management that the medical attendant will rely very much for the success of his remedies upon the intelligence and good management of the nurse. He may lay down theoretical rules for feeding and rest which may be entirely upset by the wilfulness of his patient. What is to be done with a child who will not take milk, where that is the special diet indicated by its complaint? What is to be done with a child who will sleep by day and feed by night? Or with one who refuses all food? In diarrhœa and vomiting, the administration of food has much to do with the recovery: first of all it must be suitable, then it must be given in such quantities as will suit the digestion, then it must be given regularly and with patience, and it must be freshly prepared. All the vessels used for it should be scrupulously clean, for the least trace of decomposition will upset the stomach.

The popular feeding-bottle with the india-rubber tube is a great offender: it is almost impossible to prevent particles of food from clinging to its inner surface, and as these decompose they will taint the most carefully prepared food. The bottle and nipple need careful scalding and rinsing, and should be kept in cold water betweentimes. If an infant hand-fed is troubled with diarrhœa and vomiting, look to the bottle first. As a subsidiary measure, and one of some importance, see that the loins and the abdomen are quite warmly clothed. In feeding a child who has a delicate or irritable stomach it is of great importance to give the food in small quantities and as frequently as the digestion will bear.

In all diseases of the respiratory organs the child requires a warm, even temperature, not made stuffy or poisonous by want of efficient ventilation, but a constant temperature kept up with a free interchange of fresh air. This requires a little management, but it can be done. What is essential is that the external air, which is the freshest, should be admitted steadily, and the temperature kept from falling below 60° F. The means by which this is to be brought about must be left to the nurse's ingenuity, but she must remember that letting in the used-up air off the staircase and passages is not ventilating with fresh air.

At times it is a great relief to the patient to moisten the air with steam; this is best done by surrounding the bed with some light curtains or screen and then letting the steam come into the bed from some suitable apparatus, care being taken that there is an escape from the top of the bed, or the curtains will become damp. This is an essential in the treatment of laryngitis and diphtheria after the operation of tracheotomy, it being advisable to moisten and warm the air before it enters immediately into the lungs through the tube.

It will not be necessary in an article of this nature to give instructions for dealing with diphtheria or laryngitis, as these cases require incessant care on the part of both medical attendant and nurse. Still, a few hints of arrangement may be of use. There are few cases that demand more skilled nursing than diphtheria, and the attendance on such cases should be always put into the hands of old, experienced nurses, especially after the operation of tracheotomy, as careful feeding and watching by an experienced nurse are essential to recovery. One small precaution may prevent the nurse from taking a disease which is propagated by the breath, and that is to keep her mouth closed whilst standing over the patient, and to use a disinfectant for washing the hands before taking her meals. A basin of weak carbolic solution should be put near the cot for washing the sponges, etc., used about the patient, and all feeding-cups, spoons, and glasses must be kept apart. Linen over a piece of waterproof to make a bib and pinned over the neck of the child's night-dress is a clean way of keeping the neck dry and wholesome; for it must be remembered that the diphtheritic discharges are most irritating to the skin. To sum up, a nurse in dealing with these cases must be prepared for a work that will tax all her skill, patience, and vigilance: her patient will require incessant watching, and will make endless demands on her ingenuity. Instead of pocket-handkerchiefs, some rags that can be burnt at once are advisable.

When infectious diseases are in the house, very much may be done by way of precaution in the use of disinfectants for the linen and the discharges before they are taken out of the room, as it is in these that the germs of disease are conveyed. A sheet kept moistened with some disinfectant, and hung over the outside of the door of the sick-room, is very effective. Then, of course, there should be no intercourse between the inhabitants of the sick-room and the rest of the household, and the nurse and friends should change their garments before going out. The floor of the sick-room should be swept with saw-dust moistened in the disinfectant, and all dust and refuse should, if possible, be burnt. In scarlet fever in the desquamating stage, it is the practice of some doctors to have the patient rubbed over with an ointment; some, on the other hand, say that the oil retards the process of desquamation and closes the pores of the skin; but, whatever treatment is adopted, it is essential that the skin be kept clean by frequent sponging with warm water, and by baths, and that the patient be kept warm in bed until the process is over. Every precaution should be taken to hinder the dust from the bed or room from being scattered about.

Measles is a much more unruly disease to deal with; it starts infection in the early stage, before the eruption has declared itself, and so spreads among a household almost unchecked. The same rules of disinfection will apply to this; it is of importance to keep the patient in a warm room, in bed, until the eruption has quite disappeared, and longer still if there is any tendency to lung-disease, as shown by a continued high temperature and the state of the breathing.

In this and in all other eruptive diseases the diet should be light and nourishing and with but little animal broth or tea in it, as this is apt to be over-stimulating, except when contra-indicated by great prostration. Careful observation of the temperature is of great assistance to the nurse. It begins to fall about the third day; but if it persists high or rises above 103°, then the nurse must be on the alert for some complications and look out for all symptoms that may aid the doctor in detecting the mischief, and for her part she must keep her patient warm, lying down, pay attention to the evacuations, and support the strength with careful systematic feeding.

Whatever may be the nature of the illness, one great essential in its nursing is scrupulous cleanliness in the person of the patient and in all its surroundings. A sick child should be washed all over every day, and sometimes twice a day; every part of its body should be examined, that the first sign of a sore may be detected, or any change in its condition, such as a swelling, discoloration, or enlargement about a joint, and such information should be handed over to the doctor at the earliest opportunity. In the case of young babies, their skin requires washing and drying each time the napkin is changed; a nurse who knows her work well will be able to keep her charges clean and their wants anticipated without giving in to these lazy ways. If the patient is to be clean, so must also the bed, and all soiled linen at once be taken away, not pushed under the bed out of sight, nor one wet end of the sheet tucked under the mattress, but absolutely put in its proper receptacle, where it will do no harm. The hospital draw-sheet is very useful on the sick-bed; it can be quickly drawn away without much disturbance to the patient, and another substituted. A draw-sheet is a narrow, long sheet, about one and one-half yards long by three-fourths of a yard wide, of a coarser material than the linen, and is placed under the body of the patient, sometimes with a square of mackintosh under it; it tucks in well and keeps things straight.

III. FEEDING.

It is hardly possible to exaggerate the importance of the subject with which this section deals. Nine-tenths of the ailments of children are caused by erroneous feeding; and it is not too much to say that many lives are lost in sickness that might have been saved if only the nurse had understood something of this art. And it is an art; for when we consider that the free will of man is very rampant in infancy, and that it is quite possible for the wilfulness of the babe to puzzle the skill of its elders, it will be understood that the feeding of a wilful sick child is a problem of no ordinary complexity. In the first place, there are varying ideas as to the quantity that a child should consume, and as to the frequency with which food should be given, and also as to its component parts. Nature has given a standard as to what is the proper food and proper quantity for the infant, and from this she intends us to work out the problem.

In the case of a child constitutionally weak, that has to be brought up by hand, the problem of feeding is very difficult; the child's diet must be carefully studied, and then that food which seems to suit it best must be adhered to. In dealing with these difficult cases, all theories must be laid aside, and that food used which agrees the best. The addition of five drops of brandy to each feed for twenty-four hours or so will often give the tone and vitality to the stomach that it has lost through weakening diarrhœa and vomiting. A little gentle friction over the abdomen with carefully-sustained warmth may bring about an improvement.

Another essential in rational feeding is that it should be systematic. Supposing that a child has to take one and a half or two pints of food in the twenty-four hours, then let this be divided into equal quantities, to be given at equal intervals of time. Suppose that the diet consists of milk one pint, beef tea one pint, with some stimulant, then it will be found that an alternate feed of two ounces every hour will use up the quantity in the time. This mode of feeding naturally applies in its frequency to serious illness, where the strength requires such sustenance; but where the child's condition permits, it is of importance to allow the stomach its night's rest with the rest of the body. It is a mistake into which hospital nurses fall to consider that night-feeding is as essential as day, unless otherwise ordered: better than all food is a good sound sleep, and a little nourishment given early in the morning, when vitality is low, is then of great value.

In typhoid fever systematic and rational feeding is nine-tenths of the treatment: as long as the temperature keeps high,—and that will be generally for three weeks at the shortest computation,—the patient must be kept strictly to a fluid mild diet, such as milk and beef tea, with no admixture of bread or starch foods. The well-meant but mistaken efforts of relatives to interfere with the diet of a typhoid patient must be sternly set aside.

A useful mode of feeding when a child is refractory, or when from any other cause it cannot take its food, is through the nose. In skilled hands this is a useful accessory, but in over-ambitious hands its use may be fatal.

IV. BATHING.

Some children dread the water: the origin of this fear is very often caused by roughness in washing them, or by hustling them too suddenly into a bath. Rickety children are essentially tender to the touch, and they require gentle handling when in the bath.

As the bath is essential for both the healthy and the sick child, the nurse must use her ingenuity to overcome the fear. An ordinary warm bath should be of the temperature of 98° F., and this should be decided by the use of a thermometer and not by the nurse's hand. Let the child be quite ready for the bath when the bath is ready for the child, or it will be cooling. When the bath is over, have at hand a warmed blanket on which to place the child whilst being dried; let the drying be done quickly and the child be put into its warmed garments for bed. If a douche-bath is ordered and the regular appliances are not handy, place the child in an ordinary warm bath, standing if possible, and then pour a jug of cool water down the spine from a height, or on to that particular part for which the douche is ordered. Rub the part well with a rough towel, so as to get up good circulation, and knead it with the hand. If a bath is ordered to reduce the temperature, its temperature should be 65° F. The bath being brought to the bedside, the patient is lowered in on a blanket and kept in it five or ten minutes, according to the doctor's orders; then the patient is dried quickly and put back to bed.

In cases of skin-disease an oatmeal bath is very useful, of course using no soap. A sulphur bath, used in cases of scabies, is made by dissolving half a pound of sulphur in a bath of ordinary size and warmth. The patient remains in it from twenty to thirty minutes. The sulphur turns all metal black, and the offensive smell from the bath suggests that it be given in some out-of-the-way place.

In very young children and in babes a warm bath should be a part of the daily programme, morning and night, for it is most essential that the pores of the tender skin should be kept freely open and healthy. Use a soft Turkey sponge, and then dry the body tenderly but carefully; and let the soap that is used be the purest. As the child grows older, the bath may be made tepid, until at last it may be strong enough to take a cold bath. In administering the cold bath, keep the feet from the cold water, then give a good dash of cold water all over the body with a large sponge, and quickly dry the body. If the reaction is imperfect and the surface is blue, then the tepid must be substituted for the cold bath, as the shock is too great for the system. No child under seven years of age should take a cold bath; and it is an essential in all bathing that the skin and the hair be dried thoroughly. A cork or a toy in the bath will often reconcile the nervous child to the inevitable.

V. CLOTHING AND THE BED.

It is an essential in the clothing of a sick child that it should be loose, light, easily changed, and sufficient. A sick child does not make the same use of its bedclothes that the sick adult does, and so some warm jacket must be put on to keep its chest and shoulders protected. The bedclothes also must be light and warm, and not doubled in a heavy fold over the chest, perhaps already overweighted with some difficulty in breathing.

When the patient first gets up it is necessary that the surface of the body be thoroughly well covered with light warm clothing, put on quite loosely. Woollen clothing is more warmth-giving than cotton, and is lighter. Neither in sickness nor in health should the child's body be confined in stiff clothing; binders and stays of all kinds are a mistake; they interfere with the free use of the muscles, and do not improve the shape. In construction also the clothing should be simple and easily put on and off. It would not be advisable in this chapter to advise any patterns or styles for the children's dress; but, laying down these rules as above, feminine ingenuity may easily devise a shapely garment that will harmonize with them and with the child's requirements. In sickness, the flannel vest and bed-gown require frequent changing; and it is a great soother of the night's rest to change the garments entirely at the child's usual bedtime. In dealing with surgical cases which must be kept in one position, it facilitates the process to have a night-gown open down its whole length; the same applies to patients with typhoid fever.

In arranging the bedclothes for a child that has been cut for stone, a circular bedpan placed under a circular air-cushion and the sheets arranged accordingly will serve to keep the little patient quite dry. The same can be done when the patient has paralysis with no control over the sphincters.

In preparing a bed for an operation, or where the patient must remain on it for a long time, a firm hair mattress should be selected, and a continuous board placed under it, two sheets folded straight down the centre, so that they can be easily withdrawn and kept in their places with a draw-sheet, and then the rest of the clothing arranged so as to give the most warmth. All creases must be carefully avoided, as they will cause a sore; and if pillows are required to support a limb, they should be firm, like sand pillows, and as small as possible. A good feather pillow is often of use for slinging a limb; but every appliance must be adjusted to the restlessness of childhood and to the tender nature of its skin. It is wonderful how tolerant children are of one posture and of long confinement in bed, if only they are placed in a comfortable position and well amused; and they will maintain an ordinary standard of health under such circumstances, if fed sensibly.

It may strike the reader that many of these details are needlessly minute; but it is by attention to such minutiæ that the work of nursing a sick child back to health may be accomplished. Nothing is too small that contributes to such an object, and those who have had much experience in the care of sick children know that all their success will depend upon careful thought for these details.

There can be no doubt that it is very hard work to nurse a sick child; but there also can be no doubt that the hard work amply repays itself in its results.

NURSERY HYGIENE.

By L. M. YALE, M.D.

NURSERY Hygiene in its full sense includes the same topics and covers the same ground as does general hygiene, with such variations as to details as are required by the ages of the occupants of the nursery. But in the present work these subjects have been assigned to various hands, and this article will not consider the care of the new-born, the feeding of infants and young children, nor dentition, but will be restricted to suggestions concerning the nursery itself, its situation and surroundings, its warming and ventilation; nursery nuisances and their avoidance; the dress, bath, and toilet of children, and the care of their food.

While it is true that as regards many of these topics medical advice is rarely asked, it is also true that to the mass of persons the family physician is the only sanitary authority, and that by opportune suggestions he may do much, in the aggregate, in the way of the prevention of disease. However much such guidance may be necessary in general, it is still more imperatively demanded in nursery matters, owing to the exaggerated susceptibility of young children to the depressing influence of unwholesome surroundings, and the far-reaching effects of such influence upon their development. It seems proper, therefore, to call attention, even at the risk of insisting upon truisms, to details which are often relegated to the discretion of nurses.

The Nursery Itself.—Of course no nursery can be thoroughly healthful unless the house itself is such,—is well placed upon good soil, and so constructed in detail that the rules of sanitation have been consciously or unconsciously considered. The space at our command does not permit any discussion of these rules. It will be assumed that the house is as well situated, as well drained, as well built, and as well lighted as the means of the owner or occupant will permit. The details which follow are such as assist in making the nursery the most healthful part of a good house, and as wholesome as practicable in a defective one.

In selecting a room for a nursery, that should be chosen which is the sunniest, best aired, and driest; and in deciding between two or more houses in other respects equally eligible, distinct preference should be given to that one admitting of the best arrangements for nursery purposes. In houses where no room is to be specifically set apart as a nursery, and children are to occupy the general living-room by day and the parents' bedroom by night, the same rules should govern the selection of these rooms, the sanitary benefit in such case accruing to adults and children alike. When the nursery is separate it is preferably to be placed above the ground-floor, unless the latter be unusually well raised from the ground, but it should not be immediately under a roof, on account of the difficulty of regulating the temperature in such a situation.

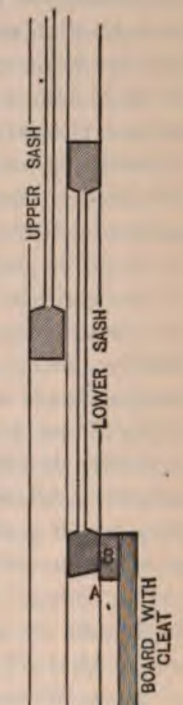
The beneficial influence of sunlight needs no insisting upon; nevertheless it is constantly overlooked. The nursery should, if possible, look to the south, or as nearly so as the situation of the house permits, with a morning exposure in preference to an afternoon sun, if but one can be had. The windows should be ample in size, and more than one if possible, as they not only serve for the admission of light, but in the ordinary dwelling are the only avenues of ventilation. The sensibility to the loss of sunlight seems to vary somewhat with adult individuals, but we believe that all children suffer from its absence; and the physician should insist upon the daily complete sunning of the apartment. In summer, even, it is usually better to have the sun and to mitigate its power at proper times by means of awnings and blinds than to have a room upon which it does not shine. There may be circumstances of climate or of prevailing winds which will modify this rule, but it holds in general. The room should be of ample size, particularly if it serve, as is the rule in ordinary houses, the double purpose of night and day nursery. The precise amount of space required for each child will vary with the arrangements for ventilation, but not less than fifteen hundred cubic feet of air per hour should be allowed, and preferably double that amount.

As only in the houses of the wealthy can a room be specially set apart as a sick-bay or hospital, the nursery must ordinarily serve that purpose whenever illness occurs. For this reason, as well as for others, the furnishing of a nursery should be as simple and as easy of cleansing as is consistent with comfort. The floor should be of smooth, closely-joined boards, preferably of hard, close-grained wood. The seams, if they open by shrinkage, should be closed either by relaying or by calking well done. Poor calking is worse than useless, and any calking is inconvenient in rooms the floors of which must be raised to reach gas- or water-pipes, as is unfortunately often the case. Carpets are necessary to comfort, but movable carpets or rugs are far preferable, as permitting more frequent cleansing both of the carpet and floor. At the present time even cheap grades of carpet are made in rug form, or the desired pattern can be made up with tasty borders without much expense. In case of actual illness of a contagious nature the rugs may be taken away at once, and their contamination be prevented, which in view of the difficulty of subsequent disinfection is very desirable. The same precaution against dangerous dirt leads to the preferring of painted and varnished to papered walls for the nursery, even at the loss of some beauty in the apartment. If paper is strongly insisted on, it should be of a kind that can be thoroughly varnished and will admit of being washed, and all old paper must be first removed before new is laid. The furniture of the room should be as light as consistent with serviceability, in order that the pieces may be easily moved from place to place to admit of frequent cleansing; and for the same reason, every bulky or heavy article should have large and strong casters. It is further desirable that all furniture should be as plain and simple as possible, carved wood and thick upholstery stuffs being objectionable as receptacles for dust. Taste may be gratified without violating this requirement. Further, all cupboards, closets, and similar places of deposit should be as open to inspection as possible, in order that offensive or untidy things may easily be detected and removed. On account of this facility of examination and cleansing, the writer usually prefers shelves with a removable curtain in front to closed cupboards and deep drawers. The latter are suitable enough for clean linen, but are temptations to careless attendants to indulge in "tuck-away neatness."

Warming and Ventilation can only be touched upon in this article. If the nursery is in a house with a good system of heating and ventilating, nothing in particular will be needed except a grate or a stove for use in emergencies. Ordinarily, however, even houses which have a fairly good furnace or other heating-apparatus have no specific arrangements for ventilation beyond what are afforded by the windows, chimneys, and imperfections of structure. The ordinary methods of warming in use in this country are open fires, stoves, and hot-air furnaces. The hot-air furnace, if properly constructed, is quite satisfactory. Its commonest faults are the delivery of too small a quantity of air at too high a temperature (a larger quantity at a lower temperature being preferable), and such an arrangement of its cold-air flue that the supply is from an impure source. The former difficulty is overcome by having the furnace considerably larger than necessary and by keeping the fire moderate; the latter, by using a tight metal flue the outer end of which is free from unwholesome surrounding and preferably raised some feet from the ground, by which means some of the foul air of dark city back-yards or of the gutters is avoided. The same precaution is of use in many country houses. The outer end must be protected by a wire screen, to prevent mischief being done by children or small animals. If the screen be fine-textured it will diminish the amount of dust drawn into the house. For a nursery it is of advantage to have the registers for warm air rather high, as this arrangement makes a better general circulation of air, diminishes the intensity of floor-draughts, and renders meddling with the register by small children more difficult.

The open fireplace has for advantages cheerfulness of aspect and a fair amount of ventilating-power, for disadvantages great wastefulness of fuel in proportion to its heating-power, so that ordinarily, when this is the only source of heat, great differences of temperature exist in different parts of the room; if the neighborhood of the fire is comfortable, the remoter parts are cold. Further, it ventilates by the production of draughts, particularly of floor-draughts, which are especially dangerous in the nursery, where little children spend so much of their time upon the floor. The wood fire is very beautiful and useful when a short, quick heat is needed, but, aside from its costliness, it is not so good for steady heating as a coal fire. In the nursery any open fire must be carefully guarded by a strong wire screen, to prevent accidents from sparks or from the clothing of children taking fire. Stoves of the ordinary close varieties, the "air-tights," are very economical of fuel, but nearly useless as ventilators, and if used make especial watchfulness as to ventilation necessary. The ventilating stoves which are the offspring of the old "Franklin" make a compromise by which all the ventilating value of the open grate is preserved with far less waste of fuel, about three times as much of the heat-value of a given amount of fuel being utilized by these stoves as by the open fire. The principle of construction in its simplicity is to surround the stove and its smoke-flue for some distance with an air-chamber; to this chamber air is admitted, preferably from out of doors, and as it is warmed it is poured into the room at a higher point,—for instance, near the mantel. As regards all stoves, it is perhaps safer to have no damper in the smoke-flue, or else to fasten it so that it cannot be closed without difficulty, since by so doing the danger of the gases of combustion being forced into the room is removed.

Ventilation, in a house which has been constructed with no reference to it, usually must be accomplished by simple devices or not at all; any radical improvements would be practical reconstruction. The most natural thing to do to let in fresh air and let out foul is to open a window; but this of course in cold weather involves dangerous draughts. We cannot, with our fickle and often severe climate, even do as some English writers suggest should be done,—fasten the upper sash so that it cannot be quite closed. Window-ventilation, therefore, must in winter be carried on by means of some contrivance which will break the force of the current of air and direct it upward so that it may be diffused about the room. The "elbow-tube" ventilator placed under the lower sash is well known; so are various wire screens, either vertical or rotating like a transom. A very useful one, and the simplest and cheapest, is the common window-board, which, fitting against the inside of the lower sash, allows the latter to be raised and the current of air inward is sent directly upward at the overlapping of the two sashes. The board should be at least eight inches high. The plan of a stout cloth nailed across the lower part of the window allows, when the sash is raised, two currents, one above and one below. The writer thinks



he gets the same advantages by modifying the window-board as in the figure. The strip B runs the whole length of the board. If it is desired to shut off the lower current, the sash is left in contact with the strip; if the lower current is desired, it is left below the strip, as at A.

The stove with jacket used in barracks seems to be well adapted for nursery use. The stove is surrounded by a jacket of sheet zinc or iron, with necessary doors, leaving space between the stove and the jacket. This should come to the floor, and the cold air be brought from out of doors to within the jacket by means of a small pipe; the air escapes warmed at the top of the jacket. For the nursery the jacket has the advantage of being a safeguard against burns, at least against severe ones.

The getting of foul air out is a rather more difficult problem, especially with stove heat. An open window with the double current described will do fairly well under favorable circumstances, but is rarely sufficient when an air-tight stove is used. If in the construction of chimneys a ventilating-flue is included, or if the smoke-flue is enclosed in a space which may serve as a ventilation-flue (as, for instance, a stove-pipe running up within a chimney which has a fireplace at the bottom), it is easy to ventilate a room. If the chimneys are already closed in, the cheapest and at the same time an efficient method is to have an air-flue leading from near the floor into the chimney higher up. The upward current of air in the latter draws the air through the ventilating-shaft. It is more efficient if placed near the stove, so that the air within it is heated and its upward movement hastened. Its mouth is placed low, to save unnecessary waste of warm air.

It should be remembered that the artificial light of lamps or gas in a room rapidly spoils air for breathing. Lighting-capacity is usually measured in candles, and an average adult produces rather less than twice as much carbonic acid as one candle: as a consequence, a large kerosene lamp or gas-burner often equals the production of five or six adults. It is very desirable, therefore, if a night-light is necessary in the nursery, that its carbonic acid be got rid of; and by the device, often used for ventilating purposes, of putting the burner or lamp within or beneath a tube or flue going to the roof or chimney, the results of combustion are carried away and an outward current of small power is also established. By having at the bottom of the flue a box, with a door, to contain the light, the latter may be shut off partly or wholly except when needed.

As to the temperature of the nursery authorities are not quite agreed; but it is certain that in American cities it is usually too high, in common with that of the rest of the house. Children and adults are often forced to endure in winter apparel a temperature (70° F. and upward) which in summer is considered to demand much lighter dress. There are good reasons why the standard of house-temperature is usually set high in America, but it is carried too far. The discrepancy between in-door and out-door temperature is made too great, the skin and mucous membranes are made sensitive, and the multitudinous forms of "colds" favored. We believe that if a room can be uniformly heated, 65° F. will be found, on the whole, more comfortable and healthful than the usual 70° F., which latter should not be exceeded. Where intelligent supervision of the temperature can be relied upon, we believe that a still lower degree than 65° F. will be healthful to children old enough to play about. At night the temperature should not be allowed to fall too far below the day standard; and especial pains should be taken to guard against the uncovering of children in bed.

A word should be added concerning windows. As is well known, the loss of heat from the cold glass is very great: Mr. Hood puts it that by each square foot of glass more than one and one-quarter cubic feet of air (1.279 cu. ft.) will be lowered each minute as many degrees as the difference between the internal and external temperatures. If, for instance, the thermometer outside showed no colder than freezing temperature, 32° F., and within no higher than 67° F., the discrepancy would be still 35° F. A window three feet by six feet would expose eighteen feet of glass surface, and according to this rule it would cool each minute ($18 \times 35 \times 1.279 =$) 805+ cubic feet one degree, or about two hundred cubic feet four degrees. This makes a constant current of descending cold air near a window, very sensibly felt by any one obliged to work in such a place in cold weather. It is important, then, that children should not play immediately near a window in cold weather, and a low article of furniture may be often so placed as to keep them away without the trouble of constant oversight. The ingenuity of the attendant will similarly devise means of keeping them from sitting on the floor if it be draughty.

Besides the admission of pure air and the discharge of foul air, purity of atmosphere demands that no nursery nuisances be allowed to exist. It is better that no plumbing of any sort should be in the room itself. Bath and closet conveniences are very necessary, but should be a little removed and well ventilated. In houses that are not plumbed, a place to which all offensive or soiled articles can be directly removed should be provided, which place should have free ventilation. In especial all soiled napkins and vessels containing evacuations or urine should be promptly removed, and in case of sickness a vessel should be provided in which the napkins or stools can be disinfected.

Under ordinary circumstances, however, disinfectants, in the usual sense of the word, have no place in the nursery nor in hygiene generally. A place that cannot be made wholesome by sunlight, air, and cleanliness should not be occupied. Whenever emergencies demand their use, they should be of the safest kinds consistent with efficiency, and after a contagious illness only the more costly contents of the nursery should be disinfected; the cheaper ones can be burnt with greater ultimate economy. For this reason we always urge that toys be of the cheapest description, particularly if of such a kind as readily to conceal supposed sources of contagion. The painting of walls and ceilings and the closely-laid floor already urged are of great assistance in promoting efficiency of disinfection.

Toilet.—*Baths* have many uses in the nursery as remedial agents, both as lessening temperature and as quieting nervous irritation of various sorts. They are here considered only in their hygienic uses as a part of the toilet: thus employed, their object is simply cleanliness and the aiding of the proper functions of the skin, with practically little intent to produce the stimulant effect incident to the cool morning bath. Such a bath needs to be of a moderately high temperature; that is to say, not very much below the usual skin-temperature, so that no great effect shall be had upon the general system. By using warm water, moreover, a smaller amount of soap and of friction is necessary for cleansing; both of which in excess tend to irritate the delicate skin of infancy. Only the best, purest, and blandest soaps should be used. While undue coddling is to be avoided, all "hardening" or "toughening" regimen is distinctly pernicious in infancy, and is to be used with judgment according to individual constitution throughout the developmental years. The power of a bath at a given temperature (according as the effects of a hot or a cold bath are sought) is much greater when the body is immersed than when it is sponged for the same length of time. For this reason, in children at all feeble the immersion should be brief or omitted altogether. The bath should never be allowed to become a domestic fetich, but its objects be kept in mind and its results noted. For young infants in ordinary health the method of administration followed by intelligent nurses is entirely satisfactory. The bath-tub contains water of about 95° F., which may cool a few degrees during the operation. The child, lying upon the bath-blanket spread upon the nurse's lap, is sponged with soap and warm water, particular attention being paid to those parts most likely to have sebaceous accumulations or to be otherwise soiled, such as the scalp, armpits, groins, and seat. This done, the child is dipped into the bath for simple rinsing, laid in its blanket, and dried with it without rubbing. As it grows older its back is supported by the hand of the nurse and it is allowed to frolic in the water for a few minutes, the exercise of kicking and its pleasure insuring a healthy reaction after the bath. If a child is alarmed at its bath, the immersion should be omitted or very brief, as fright will counteract any benefit from the immersion, and may often be accepted as evidence that from some cause the procedure is unsuitable. If it enjoys the bath, its immersion may be gradually prolonged and the temperature somewhat diminished, say to 85° F.

Toilet-powders are not necessary. Their purpose is only to dry the skin. This is better done by careful pressure with soft cloths with little friction. If irritation exists around the seat or in the groins or in other places where moisture is usually excessive, powder may be useful. We prefer mineral to vegetable powders, on account of their freedom from fermentive changes. Powdered talc we think the best.

If a *cold* bath is to be used for its stimulating effect upon a young child, before the full bath the bath by affusion should be tried, the child standing in a tub while the water is applied by squeezing it from a full-sized sponge.

The shower and douche baths have no place in the nursery except as therapeutic resources.

For very young children, sea-bathing, unless ordered as a remedy, is rarely desirable. When, however, a child is old enough to take pleasure in it, at the age of about three years on the average, it may be begun, warm weather and a place where the surf is very gentle being chosen. It is of the utmost importance that the child should not be alarmed: fright under such circumstances is deleterious physically and morally. The bath for a child of this age should consist of but a few—at first but one or two—dips, a warm wrap being at hand and the child taken directly away for drying. If the "reaction" from the bath is satisfactory,—*i.e.*, if the skin promptly becomes warm and the child not depressed,—the number of immersions may be gradually increased, and the child be allowed to play a few minutes in the water, always beside an adult and in shallow water. But it must not be allowed to wade in the water with the upper part of the body dry: it should be first immersed. As soon as the child is old enough to comprehend the method, it should be taught to swim.

Neither the in-door nor the out-door bath should be given soon after a meal, nor when the child is really hungry. In the one case indigestion is likely to follow, in the other the shock of the bath is not well reacted from.

The care of the *hair* consists in infancy chiefly in the care of the scalp, which must be kept strictly clean. If the vernix caseosa is as completely removed from the scalp at birth as from other parts of the person, there is usually little difficulty in preventing future accumulations. A soft brush should be frequently used upon the hair, a comb only as a separator for parting the locks and in emergency for disentangling.

The *teeth* require the same care as in adult life, but brushing should be of the gentlest sort, for fear of irritation of the gums, which may cause their subsequent retraction. In infancy after each feeding or nursing the gums should be washed, to prevent the formation of aphthous growths, and the teeth treated likewise as they appear. When the child is old enough to be quiet while the cleansing is done, a soft badger-hair tooth-brush should be used.

Dress.—The hygienic essentials of dress are—sufficient warmth without burdensomeness, uniformity of protection as far as consistent with activity, freedom, and, for children at least, softness. The problem of warmth without undue weight is best solved by the use of woollen garments. By reason of the poor conducting power of wool, such garments retain the bodily heat longer than those made of other materials. This slowness of conduction is greater in loose-textured fabrics. That is to say, a given weight of wool is warmer if loosely than if tightly woven. Hence the warmth of knitted garments. The difference is due to the retention in the interstices of a certain amount of air, which is a poor conductor. For the same reason, two garments, two shirts for instance, are warmer than one shirt of weight equal to the two, and loose-fitting garments are warmer than tight ones. In hot weather, however, tight garments are distressing for other reasons. Linen stands at the other extreme of ordinary dress-materials, being the best conductor of heat. It follows that woollen garments give the best protection against change of temperature and chilling, and in proper weight they make the safest dress in all places where temperature may vary or for all children who may become heated in play. Fashion or taste usually calls for outer garments of linen, but the protective garments should be beneath. The absorption of heat from the sun varies very much according to the color of the garment, the material and texture being unchanged, white taking the least heat, or being the coolest, while black will absorb about twice as much. Singularly enough, the "cool-looking" light blue is found by some experiments to be very nearly as hot as black. For very young children who are little exposed to the sun's heat this question of color is of minor importance.

Softness of material is essential for children on account of the sensitiveness of their skins. To most infants fine soft woollen shirts, either knitted or of "baby flannel," are seemingly entirely comfortable. Some, however, manifest unusual irritability of skin, and for such a shirt of fine linen should be placed within the flannel. This precaution is more often necessary in hot weather, when the flow of perspiration is increased.

The ordinary dress of very young children is objectionable in several ways. It is ordinarily unnecessarily confining about the body and limbs, although it has never in this country reached the degree in this respect that seems to be usual in some Continental countries. There is also an unnecessary number of layers of fabric involved, as they are not required for the child's warmth under ordinary circumstances. The process of dressing or undressing is really an ordeal to the infant, as it is alternately rolled upon its back and belly in the nurse's lap, in order that one band after another shall be fastened by pins or stitches. Very much of this dressing is unnecessary, if not harmful. First of all is the "band," a girdle enveloping the trunk from about the nipples to the iliac crest. Such an appliance may possibly be useful during the healing of the navel; afterwards it is not of use if tight. The abdomen needs no support in health, the compression of the ribs is not advantageous, and so far as such a girdle affects the question of hernia (which it is popularly supposed to prevent) at all, it rather favors the production of the inguinal or femoral variety. A loose girdle worn to prevent chilling is, however, often advisable in hot weather; and in cold weather a flannel girdle, or binder, "cut bias" to secure elasticity, makes a useful envelope for the entire trunk of very young children as a preventive of bronchitis.

As a means of getting rid of the objectionable features of the ordinary dress, the writer has for some years recommended the following plan[1] or some modification of it. There are three garments (besides the napkins), all covering the neck and shoulders and reaching ten or twelve inches below the feet. The outer garment, as well as the middle one, is a little larger in every dimension than that beneath it, so that no binding shall take place. They are all cut in the girdle-less pattern called "Princess." The inner one has sleeves, and may be made of cotton flannel or very soft wool flannel: if wool is used, care must be taken against shrinkage in washing. The next garment has no sleeves, and no seams at the arm-holes, to insure against pressure there; the material is wool flannel. The outer one is the usual dress, with high neck and sleeves, the details of which may be modified to suit taste. Thus, except the sleeves, the thickness is the same throughout. At night a garment like the inner one above described and a napkin only are worn. These three garments are placed one within the other before the dressing commences, pains being taken to avoid wrinkles and folds, and they are put upon the child as one garment with very little trouble. They are removed with equal ease.

---

[1] This plan was originally devised by Dr. Grosvenor, of Chicago, for use in his own family, and subsequently published by him.

The napkins may be of any suitable kind; *i.e.*, soft and absorbent material, easily washed. Linen has no real advantage ordinarily over cotton, except æsthetically. Old linen is soft, but likely to be thin. It is desirable to diminish the bulk of the napkins as far as possible, to prevent uncomfortable pressure: this is accomplished by having a small napkin simply to cover the seat and genitals thick enough to retain the urine or fæces, covered by another one not thick, but large enough to envelop the hips. The age at which napkins may be discontinued depends upon circumstances. Among English families of the better classes, apparently, children are taught to make their needs known earlier than is usual with us. Much can be done by an attentive and intelligent nurse who holds the child over a vessel with suitable frequency. But children vary greatly in this particular, and under no circumstances is any severity justified, or even scolding, as nervousness or anxiety on the part of the child simply aggravates the trouble. As soon as the child can regularly give notice of its wants in this respect it is better to discontinue the diaper, as its absence gives greater freedom to the limbs. Of course at all times napkins should be changed as soon as discovered to be damp or soiled. Rubber or other impervious covers for diapers should not be used. Even the exigencies of a railway-journey, with the conveniences usual in this country, do not require their employment. They simply convert a wet napkin into an unclean fomentation.

When a child begins to use its limbs freely, the clothing should be shortened. In fact, there is no real need of long clothes at any time, except to save labor in keeping the infant's feet covered. When it begins to creep, its manœuvres are facilitated by slipping over its skirts a loose baggy pair of breeches of woollen which is tied around its waist and buttoned about its knees. This keeps the skirts from impeding its progress, and protects it against floor-draughts in a measure.

The dress of older children should conform to the same hygienic requirements as given above. The two most frequently disregarded are freedom from constriction and uniformity of protection. The former is violated by the use of tight girdles, or even by corsets, tight sleeves, garters, and misshapen stockings and shoes. Their harmfulness is well understood: the neglect is usually a wilful preference of fashion to healthfulness. The same might perhaps be said of the fashion of unevenly distributing the clothing over the person; but the injurious effects of this are less understood. Chilling is resisted far better if the whole person is exposed to the same temperature than if one part is exposed to a lower temperature than another. It is a matter of universal experience that many persons who rejoice in outdoor life even in severe weather are directly injured by a draught and by sitting near a window. Yet formerly more than now low-necked dresses were used for children, the entire shoulders being exposed, while the remainder of the trunk was burdened with dress. At the present time fashion exposes the legs more. Shoes and stockings are often too thin, but in particular children are too often dressed with the lower limbs bare from above the knee to a little way above the ankle, the foot being covered by a slipper. The difference is often aggravated by too much clothing on the body and a sash over all. The lower limbs should be thoroughly clad,—not cumbrously, but warmly. The stocking of a child old enough to run about should be long enough to meet or be overlapped by the next article, napkin or drawers, as the case may be. Stockings of wool, for the reasons already given, are to be preferred. They should be soft. They should not be pointed at the toes, but be wide enough to admit of ample play in every direction of the anterior part of the foot. Color is not indifferent, as some dyes have been found to produce eruptions on the skin. Public attention has, however, been so thoroughly drawn to this subject as to have led in some instances to legislative enactments, and such dyes are probably less frequently used than formerly. Aniline reds have been thought to be especially irritating.

Shoes of proper shape are not easy to get for children; not nearly so easy as for adults. This comes probably partly from the supposed necessity of making them for a low price and partly from a belief, often openly expressed, that "a baby's foot has no shape." The real shape of the human foot is followed in the true "waukenphast" shoe, but this we have never seen of proper sizes for infants or young children. It is not enough that a shoe should be as wide or wider than the foot, but it should have its width rightly disposed: space where the foot does not demand it in no wise compensates for pressure elsewhere. The result must inevitably be a distortion. In choosing shoes for infants it is better that they should be unduly long, if that be necessary to obtain the requisite width in front, than that they should be narrow.

The Care of Food.—The feeding of children will be treated of elsewhere in this volume, but it remains to say a few words concerning the care of their food and of drinking-water. Food for children who are on a general diet is to be cared for in the same way as that for adults, but with additional scrutiny, owing to the greater susceptibility of children to the injurious effects of unwholesome articles. These effects are in many instances now attributed to the development of ptomaines in the food. Most of the ptomaines already recognized are developed in articles of food, such as sausages, ham, and canned goods, that do not form a proper part of the food of young children. One only is of importance in this connection, namely, tyrotoxicon, which may be developed in milk or any food of which it is a component part. To prevent its development, scrupulous cleanliness must be observed as to every vessel that may contain milk, the room in which it is kept, and every utensil which may be used in the preparation or administration of food; for all these things have been distinctly shown to be the conveyers of the poison. Food should not be allowed to stand about the nursery except as immediately needed, and especially it should never (and this is emphatically true of milk) remain in the room with soiled napkins or alvine discharges.

The sterilization of milk often becomes necessary, although it cannot be said that it is ordinarily so. Whenever there is any special reason for suspecting the purity of milk, when the weather or other conditions are particularly favorable to changes in it, when the occupants of the nursery are already affected with diarrhœal diseases, or when such ailments are prevalent in the neighborhood, sterilization is advisable. The elaborate apparatus for the purpose devised by Dr. Soxhlet is now manufactured in this country;[1] but the process may be simply and effectually carried out by placing the milk in suitable bottles, such as ordinary nursing-bottles or the stout bottles used for soda-water or ginger ale, and putting these into an ordinary kitchen steamer and keeping them at the temperature of steam for at least fifteen minutes. If no steamer is at hand, the bottles may be partly immersed in water kept boiling for the same length of time. The bottles are then seized by a well-protected hand and tightly stopped with corks which have been boiled,—rubber corks are preferable,—and put away till needed. If the milk is to be kept for any length of time, boiling or steaming on successive days gives greater surety of permanent sterilization.

An additional word may be said about the care of nursing-bottles. The long-tubed bottle has been pretty generally condemned, owing to the difficulty, amounting in practice to impossibility, of keeping the calibre of the tube clean. The rubber nipples are quite readily kept clean, and so are the bottles with care. The writer has used with satisfaction the bottle devised by Dr. Haven, of Boston, which is really a feeding cup rather than bottle. It is made of stout glass; its shape is much the same as that of the beaked cup used to feed the sick. The bottom is flat, the upper side open, at one end is a handle, at the opposite end the tubular nose terminates in a thickened end which keeps the rubber nipple in place. Its advantages are that it is as easily cleansed as a cup, and that the child cannot be left to play with its bottle, to eat when it pleases, stop, and resume again, drinking sometimes warm and sometimes cold food. It must be held by the nurse during the entire time of feeding. As the flow of milk is very free, the holes in the rubber nipple should be very small.

---

[1] In New York by C. Riessner, 406 Pearl Street, and R. Van der Emde, Second Street and Bowery.

To the care of drinking-water the same general rules of cleanliness apply as to the care of food. But if the supply of water is not good, the consumer is usually less able to remedy the difficulty than he is in the matter of food.

If water is too hard, it can usually be improved somewhat by boiling, which causes the deposit of a part of the lime. If the water is impure from organic matter, the impurities may or may not be deleterious to health. Water from ponds is often high-colored and even at times disagreeable in odor from vegetable matter without any mischief following its use. We have observed an instance where an active outbreak of typhoid fever seemed to be directly due to a mistaken dread of discolored and unpleasant water from an aqueduct, leading many persons to resume the use of neglected wells which were contaminated from privies, although the well-water seemed to the eye and nose to be pure. Perfectly efficient filters which yield any considerable amount of filtered water (porcelain filters, etc.) are too costly for general use. But water can ordinarily be made safe by thorough boiling for fifteen or twenty minutes—better still by boiling on two successive days—and subsequent coarse filtration through filter-paper, or a wad of absorbent cotton packed neatly into the bottom of the funnel. The entire outfit of a large funnel and a water-vessel costs but very little. It may be of tin if constantly watched and cleansed.

The use of iced water is undesirable for various reasons: the ice may be impure, and freshly-made iced water is not proper for children's consumption. Both difficulties may be overcome by putting the household drinking-water into large corked bottles or into closed jars and placing these near the ice or in the refrigerator. In this way water may be had that is cooler than ordinary spring-water and safe to drink. If the taste of water that has been boiled seems insipid, as it is apt to do at first, the addition of a minute quantity of salt generally renders it palatable.

Out-door Exercise.—Except in inclement weather, most children are better for being abroad daily to receive the influence of the sun and the pure air. Exceptions, of course, exist, particularly in winter. Young children—under six months of age—should be carried in arms in cool weather, that they may have the warmth and support of the nurse's arms and person. In very cold weather or in inclement weather we believe it is preferable to open for a while the windows of a room that faces the sun, until the air is as pure as that out of doors, then to close the windows and to allow the children to play there or be carried about there attired as if for out of doors.

DENTITION.

By JOHN DORNING, M.D.

Definition.—The term dentition, as generally used, refers only to that stage of development when the tooth is penetrating the superficial tissues of the gum. The period between the seventh month, when the first teeth appear, and the end of the second year, at which time the second temporary molars erupt, is spoken of as the dentition epoch.

By the second dentition is meant the eruption of the permanent teeth.

Development of the Teeth.—The space allotted to this article forbids more than the briefest general description of the development of the teeth. For a full account of the evolution of the teeth the reader is referred to the different modern treatises on dental histology.

In the human subject two sets of teeth appear in the course of life. The first set, consisting of twenty teeth, appear during the first two years after birth, and are known as the temporary, milk, or deciduous teeth. The second set, thirty-two in number, appear after the fifth year, and are called the permanent teeth.

The first trace of the future tooth is perceptible about the sixth week of intra-uterine life. There is an active proliferation of the epithelial cells covering the rudimentary gum, which becomes centred along a line marking the location of the future arch of teeth. This rapid cell-multiplication causes a depression or groove in the jaw, which deepens as the cell-growth advances. In other words, we have a groove (the dental groove of Goodsir) in the jaw, filled with epithelial elements. The collection of epithelial cells filling the groove is called the epithelial cord, and from it is developed the enamel organ, which furnishes the enamel for the future tooth. As the epithelial cord extends into the gum, its distal end expands into a club-shaped enlargement, and, meeting the dentinal papilla which springs from the deep connective tissue of the jaw, becomes invaginated by the latter, so as to form a complete cap for the papilla. The dentinal papilla is the future pulp, and from it is developed the dentine of the prospective tooth.

From the side of the epithelial cord is given off a secondary process, the epithelial cord of the future permanent tooth. Extending from the base of the papilla upward along the outer side of the enamel organ is seen a thin layer of fibrous connective tissue, which becomes condensed into what is known as the tooth follicle, or sac. The cementum of the tooth is also probably developed from this connective tissue. As this follicle-wall grows up over the expanded end of the epithelial cord, or enamel organ, and encroaches upon the neck of the cord, the latter atrophies and the connection between the mucous membrane of the mouth and the enamel organ is consequently severed. The tooth-germ is now, about the sixteenth week, enclosed in its follicle. Very soon, about the seventeenth week, calcification of the dentine and enamel begins, and is followed in a few weeks by commencing ossification of the crypt which encloses the tooth-germ.

"The germs of the milk-teeth make their appearance in the following order: at the seventh week, the germ of the first molar of the upper jaw appears; at the eighth week, that for the canine tooth is developed; the two incisor papillæ appear about the ninth week (the central preceding the lateral); lastly, the second molar papilla is seen at the tenth week, behind the anterior molar. The teeth of the lower jaw appear rather later, the first molar papilla being only just visible at the seventh week, and the tenth papilla not being developed before the eleventh week."[1]

According to Dr. Pierce,[2] calcification of the dentine and enamel of the central and lateral incisors begins at the seventeenth week of embryonic life, and calcification of the cuspids and molars commences at the eighteenth week. At the fortieth week, or at birth, calcification of the crowns of the incisors is quite complete and the roots are beginning to calcify. Three months after birth, the cuspid and molar crowns are complete and calcification commences in their roots.

With the completion of the crown and beginning calcification of the fang, the process of eruption commences. The growth of the root propels the crown towards the surface of the gum, the superimposed tissues, first the margin of the bony crypt, and then the soft structures of the gum, disappearing by absorption. Synchronously with the development of the root, the jaw increases in depth by the addition of new osseous material. The bony crypt is rebuilt around the neck of the tooth, and forms the alveolus or socket of the milk-tooth.

By many there is thought to be some force in addition to, or independent of, the elongation of the fang, in impelling the tooth-crown forward. Among the facts offered in substantiation of this view are: first, in teeth prematurely erupted the roots are sometimes undeveloped; second, a tooth may be completely formed, and still remain buried in the jaw, and erupt later in life; and, third, when a normal tooth erupts, its crown travels a greater distance than is represented by the increase in the length of its fang during the same time.

---

[1] Gray's Anatomy, 8th ed., p. 753.

[2] American System of Dentistry, vol. iii. p. 636.

Eruption of the Teeth.—Between the sixth and eighth months after birth, the two lower central incisors erupt, usually simultaneously.

Between the eighth and tenth months, the two upper central incisors appear, followed shortly by the two lateral incisors.

Between the twelfth and fourteenth months, the two upper anterior molars, the two inferior lateral incisors, and the two lower anterior molars appear, in the order mentioned.

Between the sixteenth and twenty-second months, the four canine teeth erupt.

Between the twentieth month and the end of the third year, the four posterior molars erupt.

The eruption of the twenty milk-teeth is now complete, and no more teeth appear until the fifth or sixth year, when the eruption of the permanent teeth commences.

Shedding of the Deciduous Teeth.—The temporary teeth drop out in about the same order as they appear.

Scarcely a year elapses after calcification of the milk-teeth is complete before absorption begins. There is still some obscurity about this most interesting physiological phenomenon. The process of absorption would seem to be quite independent of the presence and pressure of the permanent tooth, as is evinced in the fact that, not infrequently, absorption of a milk-tooth is carried on in the absence of its successor; and, again, decalcification is known to commence on that side of the fang opposite to the successional tooth, and also in several places at once.

Normally, absorption begins at the apex of the root and advances towards the crown. Shortly after the root has disappeared the crown is removed either by the advancing permanent tooth or by an accidental rupture of the attachment between the neck of the tooth and the mucous membrane of the gum.

Development and Eruption of the Permanent Teeth.—The germs of the first permanent molars appear during the fourth month of embryonic life; at about the same time may be noticed the first steps in the formation of the twenty anterior teeth of the second set. The germs of the second permanent molars do not show themselves until the third month after birth; and those of the third molars (wisdom-teeth) not before the third year.

The epithelial cords of the twenty anterior teeth spring from the epithelial cords of the corresponding temporary teeth. The cords for the twelve permanent molars arise either from the epithelium of the mouth or from successive extensions backward of the epithelial cords of the posterior milk molars.

The development of the permanent teeth is similar to that of the deciduous teeth.

Calcification of the permanent teeth begins in the first molars about the sixth month of fœtal life.

"First year after birth, central and lateral incisors begin calcification. Four years of age, cuspids, bicuspids, and second molars begin calcification. Eight years of age, third molars begin calcification."[1]

To accommodate the developing molars, the jaw increases in length by the addition of bony material at the posterior border. As the permanent teeth erupt, the sockets and roots of the temporary teeth disappear by absorption, and new alveoli are built for the second set.

Ordinarily, the permanent teeth erupt at the following periods, the teeth of the lower jaw preceding those of the upper:

Sixth year, first molars.
Seventh year, central incisors.
Eighth year, lateral incisors.
Tenth year, first bicuspids.
Eleventh year, second bicuspids.
Twelfth to thirteenth year, canines.
Twelfth to fifteenth year, second molars.
Seventeenth to twenty-first year, wisdom-teeth.

ANOMALIES OF THE TEETH.

Precocious Dentition.—It is not uncommon for dentition to begin prior to the sixth or seventh month. Some children are even born with teeth. Many interesting examples of this singular anomaly have been placed on record. The younger Pliny states that the Roman consul Manius Curius had a full set of teeth at birth, on account of which he was named Dentatus. Louis XIV., Richard III., and Mirabeau are said to have had congenital teeth. I have seen two infants both of whom were born with a lower central incisor through the gum.

In some cases congenital teeth are less dense than normal teeth, have no roots, become loose and drop out during the first few months of life, and are replaced by the deciduous teeth proper. In other cases these teeth have been known to remain until displaced by the permanent teeth, and were, therefore, undoubtedly genuine milk-teeth.

Precocious dentition is usually associated with premature ossification of the bones, particularly those of the head. As a consequence, there is early closure of the fontanels and sutures, which may interfere with the normal development of the brain.

After the premature eruption of one or more teeth, dentition may cease for from four to twelve months, or even longer, as a result of malassimilation from some cause.

Premature dentition is believed by some observers to be evidence of a tubercular, scrofulous, or syphilitic diathesis. It is, however, sometimes observed in children in whom no inherited taint can be discovered.

Retarded Dentition.—It is very common for the beginning of dentition to be deferred for several months after the normal period. In some rare cases teething does not commence until the second year or later. I have had under my care three children who did not cut their first teeth until the twenty-fifth, twenty-seventh, and twenty-eighth months respectively. Jacobi mentions the case of a boy whom he had under observation until the age of two years and ten months, "at which time he had not a tooth, nor a symptom of approaching dentition." Churchill reports a case in which the first tooth appeared at seven years of age. Smellie records the case of a patient whose first tooth erupted at twenty-two years of age.

---

[1] Dr. Pierce, American System of Dentistry, vol. iii.

Delayed dentition is an indication of a late general development,—in the vast majority of cases the result of rachitis. As a rule, in cases of protracted teething the anterior fontanel closes later than the seventeenth month, the normal period, and ossification of the bones is also delayed. Teeth that are cut late are frequently marked by imperfections of the enamel, lack density, and decay very early.

Absence of Teeth.—Deficiency in the number of teeth is of more frequent occurrence in the permanent than in the temporary set. A milk-tooth may fail to appear because of the destruction of its germ by traumatism or disease. In some rare instances there is an absence of a number of teeth. Such abnormities have been accounted for on the ground of heredity; sometimes they are found in connection with other anomalies of the dermal system. In the permanent set, the upper lateral incisors are most frequently found missing. Cases are reported where a missing tooth has been found lying horizontally in the jaw. Two such cases have fallen under my own observation. The total absence of teeth is an exceedingly rare anomaly. There are but a few cases on record, and some of these, judging from their histories, are questionable.

Multiple Dentition.—In medical literature, a number of cases of a third and even a fourth dentition are narrated. Theoretically, a third dentition is not impossible; but in the cases reported, the statements of the patients and their relatives constitute the only evidence of this abnormity, and such testimony is not always to be relied upon.

Supernumerary Teeth.—The number of teeth is not infrequently increased by one or more additional teeth. Supernumerary teeth are generally small, and, although usually distinct, are sometimes attached to other teeth. They are more frequently located in the anterior part of the mouth, and are more common in the upper than in the lower jaw. Supernumerary milk-teeth have been followed by corresponding supernumeraries in the permanent set. The mouth of a medical student whom I recently examined contained five lower permanent incisors. The teeth were all in the dental arch and but slightly twisted on their axes.

Irregularities in the Order of Eruption.—It is not uncommon for the normal order of eruption to be violated. The upper incisors often erupt first; and when such is the case their appearance is usually delayed. The lateral are sometimes cut before the central incisors. In rare instances the molars or canines precede the incisors, a posterior molar erupts before a canine, or a canine protrudes prior to an anterior molar. In the case of a child brought to my clinic, the upper anterior molars were the first to erupt at the age of sixteen months. That there is a normal order of eruption is a fact recognized even among savages. Dr. Livingstone tells us that among some of the tribes of Central Africa a child that cuts the upper teeth first is believed to be *moiko* (unlucky) and certain to bring death into the family. Such a child is sold to the Arabs. In some civilized countries the eruption of the upper incisors first is considered a bad omen.

Malposition of the Teeth.—Malposition of individual teeth is of much less common occurrence in the deciduous than in the permanent set, and when found is usually limited to slight torsion or overlapping of the upper or lower incisors. The permanent teeth most frequently malposed are the inferior incisors and canines; next, the superior incisors; after these, the third molars.

All sorts of irregular arrangement are seen. The involved teeth may be twisted on their axes, overlap one another, or be displaced within or without the dental arch.

Displacement of the teeth occurs when the jaw is too small for their proper accommodation. A disproportionately small jaw is a common consequence of rachitis. In a certain percentage of cases heredity seems to be an undeniable factor. The blending of types by intermarriage of different races is a well-recognized source of small jaw and large and displaced teeth.

Persistent thumb-sucking is said to cause a forward direction of the upper anterior teeth and a backward inclination of the lower front teeth, with more or less deformity of the jaws.

Malformations of the Teeth.—There are numerous departures from what may be regarded as the typical form of a tooth. Large teeth with very small roots, an increased number of cusps or fangs, outgrowths from the crown or fang, twisting, bending, division or coalescence of the roots, are among the variations in shape.

The surface of a tooth is often marked by transverse or vertical ridges and furrows, or pittings, the enamel being apparently perfect. Under the microscope, however, the enamel is generally found to be defective. These ridges and furrows are analogous to the ridges and grooves seen on the nails, both the result of interrupted nutrition.

The enamel of a tooth may present a few excavated spots or a general honey-combed appearance due to a disorganization of this structure. Sometimes the crown of a tooth is entirely devoid of enamel.

Pigmented spots, and spots having the appearance and consistency of chalk, are not uncommonly observed.

Defects in structure are due to some morbid condition of the organism during the developmental period of the teeth,—in the course of intra-uterine life, when the milk-teeth show imperfections, and in the first few years after birth, when the permanent teeth are faulty.

A large proportion of artificially-fed children have faulty permanent teeth later in life. The condition of the teeth, therefore, would in a general way seem to be an indication of the previous health of the individual. When the defects are seen on only one tooth, the cause may be local.

There is sometimes an absence of the enamel at the middle of the biting edges of the upper central incisors. The exposed dentine is soft and but partly calcified, and is soon worn away, leaving a crescentic notch in the edge of each tooth. Notched milk-teeth are of no special diagnostic import. But when the permanent upper central incisors are notched, they are almost invariably an indication of congenital syphilis. Mr. Hutchinson was the first to call attention to this condition of the teeth in inherited syphilis. They are known as "Hutchinson teeth." This peculiarity in the upper central incisors was at one time thought to be caused by stomatitis, but at present it is believed to be the result of an arrest of development in the central or first-formed portion of the tooth.

In subjects of congenital syphilis both the temporary and the permanent teeth may be crescentic. A number of such cases have come under my observation.

SYMPTOMATOLOGY AND ALLEGED DISORDERS OF DENTITION.

At one time dentition was held accountable, directly or indirectly, for nearly all the ills of infancy. At the present day, owing to a more extensive knowledge of the etiology and pathology of disease, and greater proficiency in methods of diagnosis, the symptomatology of dentition and its power as an etiological factor are, with the majority of the profession, becoming more and more restricted, and by some totally ignored. There are, however, others who still adhere to the old doctrine of dentition as tenaciously as do the laity, and in their practice among the infant population find more use for the gum-lancet than for common sense.

Dentition is a purely physiological process, and, like other physiological processes, is subject to irregularities from local and constitutional disorders. Its affirmed etiological potency, however, is questionable.

It is true, functional derangements and organic disease are more common, and the mortality greater, between the ages of six months and two years than at any other period of childhood; but hereditary, dietetic, hygienic, and educational influences furnish causes more rational and demonstrable than the presumed irritation of a hidden tooth-germ.

There never has been any unanimity of opinion on the subject of how teething produces the numerous disorders attributed to it. It is said that dentition is more severe in the winter than in the summer, and *vice versa;* more so in large cities than in the country; and its consequences more serious in badly-nourished children, and among the poor; that diseases occurring during dentition are rendered more dangerous by this process; that teeth erupt with more difficulty during the course of any severe malady; that the cutting of the incisors, on account of their sharp edges, is more painful than the extrusion of the molars; that the eruption of the molars causes most pain because of their broad crowns; that the eye-teeth, owing to their long fangs, are liable to give rise to cerebral disturbances; that the protrusion of the stomach-teeth is likely to be attended with vomiting and diarrhœa or cough; that it is the evolution of the molars that causes most cerebral and intestinal troubles. Then, again, the forward pressure of the advancing tooth-crown on the superimposed gum, the backward pressure of the fang on the nerves of the subjacent parts, and the lateral pressure of all the teeth together are thought, by their respective advocates, to account for the many complicating ailments of dentition.

In the estimation of many writers the semeiology of dentition embraces drooling, rubbing of one jaw on the other, biting on the fingers or any hard substance that can be carried to the mouth, fever, restlessness, peevishness, fretfulness, disturbed sleep, flushing of the cheeks, itching of the nose, dilated pupils, conjunctivitis, otalgia, pain and inflammation of the gums, aphtha, thrush, anorexia, vomiting, diarrhœa, bronchitis, convulsions, local spasms and paralyses, and cutaneous eruptions.

Drooling is said to be the first indication of approaching dentition, and is thought to be the result of a stimulation of the salivary glands by an irritation transmitted through the chorda tympani from the gums. It is believed that drooling keeps the gums soft, relieves the congested capillaries of the gums and mouth, and "derives the blood from the brain and moderates its irritative condition."

Slavering is observed to commence in all healthy and normally-developed infants between the third and fifth months, and generally ceases before the eighteenth month. In sickly and backward children it usually begins later, and may continue for several years.

While the infant is fed at the breast there is no requirement for either teeth or saliva; still, the development of both the teeth and the salivary glands must of necessity be well advanced towards completion before the period of weaning. Hence, instead of regarding this copious flow of saliva as a manifestation of a morbid action of the salivary glands dependent upon dental irritation, it would be more reasonable to assume that it, like the eruption of the teeth, simply betokens a stage of developmental activity in which there is a preparation of the digestive organs for the reception and utilization of the aliment that is to succeed the maternal milk.

Rubbing of one jaw on the other and biting on the fingers or any substance that can be carried to the mouth are supposed to be indicative of a feeling of uneasiness, or itching, in the gums, induced by the upward pressure of the teeth; and some smooth and hard material is recommended, for the child to bite on, with the view of allaying the pruritus and hastening the absorption of the superimposed gum.

Jacobi says, "Is it astonishing that an infant will during the time of dentition take everything to its lips and into its mouth, after it has done so all its life? The principal impressions an infant obtains depend on its relation to foods and drinks; eating is the only real propensity an infant has, and the mouth is known by experience to be the great receptacle destined for the reception of everything around; not to speak of the lips being used as a means of touching, grasping, and learning the qualities of things."

The grinding of the teeth in children who have completed their first dentition is evidently, at times, due to some derangement of the economy. The biting motion of the jaw in infants before and during dentition may likewise be occasionally excited by some irritation, but not necessarily seated in or reflected from the gums. If there be any sensation at all attending the eruption of a tooth, it probably amounts to nothing more than a moderate degree of pruritus. It should be remembered that muscular action is essential to muscular development; that a healthy child is in almost constant motion while awake, and that the masticatory movements may be, and probably are, but a part of the general gymnastics in which the child indulges. An infant cannot walk, neither can it masticate food, yet it exercises both the muscles of locomotion and those of mastication, developing and educating them for their respective functions when, at a later period of existence, these shall become necessary.

Fever, restlessness, peevishness, fretfulness, and disturbed sleep are the commonest manifestations of infantile derangements. Not infrequently they are coincident with the eruption of a tooth or a group of teeth. When such is the case, a superficial examination may lead the physician to conclude that a relationship exists between the two; whereas a careful and thorough investigation will generally bring to light some associated condition which at another time would be considered quite adequate to produce these symptoms. If fever and general irritability be symptomatic of dentition, they should be continuous throughout its whole duration, or coincident with the eruption of each group of teeth, instead of appearing at uncertain times; and, furthermore, they should be present in at least a mild degree in every child.

Slight disorders, presenting a few indefinite symptoms, occur at all ages, and the diagnostician is now and then at a loss to account satisfactorily for them. To rely upon the age of the patient in determining whether they shall be attributed to a physiological process or to some much more probable cause is certainly illogical. Peripheral impressibility is very pronounced in the infant, particularly in one whose power of resistance is lessened by some constitutional vice; and any slight irritation, as from indigestible food or parasites in the alimentary canal, constipation, disarranged clothing, a misplaced pin, or soiled napkins, may give rise to a greater or less degree of fever and general uneasiness.

Very often, trifling disorders that are viewed as evidence of difficult dentition are, directly or indirectly, dependent upon rachitis. This is one of the most common of children's diseases, and frequently a mild form of the affection passes unrecognized because its symptoms have received a [illegible]. The local and general disturbances, in the estimation of the parents, and not infrequently in that of the physician too, merely mark the dreaded teething epoch, the attending perils of which every infant is destined to encounter. The tardy dentition and lateness in walking are regarded as nothing but harmless freaks of nature, and instances are cited where the same peculiarities have been noticed in other members of the family. When rachitis is recognized,—and it should be, before any deformities of the bones are visible,—and an appropriate line of treatment adopted, recovery generally follows; the teeth are cut rapidly, and, owing to the extra attention bestowed on the child, few if any of the ordinary derangements of infancy occur.

Vaso-motor disturbances, as the transient flushing of the cheeks, or sudden pallor of the countenance, are often noticed during the time, and, it is said, in consequence, of dentition. But it should be remembered that there are many conditions in which these symptoms are present, and they must receive careful consideration before making a diagnosis of difficult dentition. It will then seldom be necessary to fall back on teething.

Conjunctivitis is said now and then to occur on the same side on which the teeth are protruding. More than likely this is a coincidence. But the opinion has been advanced that it may be the result of dental irritation, the extension of the irritation to the conjunctiva being favored by the proximity of the apices of the fangs of the canine and first molar to the floor of the orbit,—a rather unsatisfactory explanation.

Otalgia, as indicated by crying and the carrying of the hand to the side of the head, has been declared one of the reflex disturbances of dentition. In congestion or inflammation of the middle or external ear, meningitis, or cerebral hyperæmia, the child carries its hand to the neighborhood of the ear and gives evidence of suffering. Most of the earaches in children are dependent upon acute otitis; and many an otitis is neglected until the organ of hearing is irreparably damaged because "the doctor said the ear would [illegible] when he [the child] cut all his teeth." "The doctor" had [illegible] forgotten that the same predisposing and exciting causes could be [illegible] before the eruption of the last of the twenty milk-teeth, as well [illegible].

[illegible] swelling, and tenderness of the gums during the time of dentition, are generally held to be symptomatic of some difficulty in the eruption of the teeth.

[illegible] writing on difficult dentition, remarks, "Redness, pain, swelling, [illegible] increased secretion (or, in short, catarrhal stomatitis) are present [illegible] Jacobi says, "There is no stomatitis; certainly no thrush; [illegible] pathological conditions."

[illegible] healthy child are of a pale pink hue. As a tooth [illegible] the surface, the gum in that locality becomes more prominent, [illegible] until it is almost white, and is anything but sensitive. [illegible] tooth just before it reaches the surface a depression is often observed, due to a disappearance of the epithelial and subepithelial layers, by a necrotic process.

Sometimes the gum over the crown of an erupting tooth becomes inflamed and tumid, and an incision may give exit to a drop or two of thick, black blood. The gum around the top of a tooth that is partly through the gum is oftentimes inflamed. This condition I have repeatedly seen follow attempts at "rubbing the tooth through" with a thimble, finger-nail, or other hard substance. Ulceration of the gum over a tooth now and then occurs from the impingement of a sharp corner of a corresponding tooth that has erupted in the opposite jaw.

It is doubtful if dentition be ever the sole cause, or, indeed, a cause at all, of a gingivitis. If the highly sensitive nerve-filament that forms a part of the pulp of a milk-tooth becomes obliterated together with the substance of the fang, without pain or inflammation, to make way for its successor, it is not unphilosophical to infer that the less sensitive tissues that lie in the path of a deciduous tooth are disposed of in a manner as painless and as free from inflammatory action; for in both instances the same result (absorption of vitalized tissue) is achieved by processes that are, so far as the principle of action is concerned, identical.

When stomatitis is present, some cause other than dentition should be sought. The vast majority of cases of stomatitis occur in bottle-fed children. It is generally associated with some derangement of the organism, particularly the digestive tract. The child's diet or hygiene is usually at fault. The use of foul nursing-nipples, dirty teething-rings, and filthy sugar-teats, thumb- and tongue-sucking, and irritants taken into the mouth, as hot fluids, principally tea and coffee, drugs, or substances the child may pick up while wandering around on the floor, may give rise to stomatitis.

Diarrhœa in teething children has by some writers been attributed to the swallowing of large quantities of saliva, the salts contained in it being supposed to act as a mild aperient. By others the reputed dental diarrhœa is thought to be of a neurotic character,—an irritation being transmitted through the sympathetic nerves to the vagus, influencing the glandular secretion of the digestive tube or producing a hyperperistalsis of the intestines.

Vogel says, "A mild diarrhœa, five or six evacuations in the twenty-four hours, is very beneficial to teething children, for cerebral affections are thereby most surely prevented." Many children are sacrificed annually through a belief in such an erroneous doctrine. Diarrhœa may occur at the time a tooth is protruding, or at successive periods of dental evolution, but never in consequence thereof. Children who are fed exclusively at the breast at proper intervals, and whose hygiene receives careful attention, seldom suffer with diarrhœa before the period of weaning. Then, again, diarrhœa is strikingly more prevalent in one season than in another, notwithstanding the eruption of teeth at all periods. These two facts rather militate against the theoretical existence of diarrhœa from dental irritation.

The causes of intestinal derangements are improper feeding, bad hygiene, and the changes produced in the atmosphere, especially in a city, by a high degree of solar heat. The most significant of these factors is improper feeding. Most babies at the breast are nursed too often. Bottle-fed infants, in addition to being fed too frequently, labor under the disadvantage of not being provided with a suitable substitute for their natural food.

Too commonly, undue importance is attached to the appearance of the first tooth. Its presence is hailed as the beginning of a new era in the child's existence, and no opportunity is lost in putting the anxiously-watched-for organ to a legitimate use.

Bronchitis, it is said, may be the result of the saturation of the coverings of the chest with the saliva that flows from the child's mouth,—a plausible view. It is also thought to be due to a nervous irritation reflected from the gums.

Because an attack of bronchitis will now and then subside with the eruption of a tooth, it does not follow that the cutting of the tooth is the cause of the bronchial inflammation; for a mild attack of bronchitis will get well spontaneously in a child free from any predisposition, whether a tooth be coming through or not. After a child has begun to creep or walk it is more exposed to atmospheric changes than earlier in life. Hence the greater frequency of attacks of bronchial catarrh during the second year. Rachitic and scrofulous children are subject to recurring attacks of bronchitis; and the great prevalence of rachitis should not be overlooked.

Convulsions, varying in form from slight twitchings of particular groups of muscles to a general eclamptic attack, are said to have an origin in dentition. Frequently a child will sleep with the eyes half open and the eyeballs rolled upward, presenting a most appalling spectacle to the inexperienced mother. Or a smile will occasionally flit over the infant's countenance, caused by the contraction of the facial muscles,—a pleasing sight to the sentimental mother whose creative imagination conjures up a vision of angels whispering to her sleeping babe. Her apprehension in the one event or her happy delusion in the other is, however, soon dispelled by some wiseacre who knowingly unfolds to her some of the mysteries of teething.

Now and then a general convulsion will occur,—perhaps with the eruption of a tooth, or at successive periods of dental protrusion.

But it must be remembered that during the dentition epoch the whole organism is in a state of active development; that the nervous system has not acquired the stability of equilibrium of the youth or the adult, and is therefore extremely susceptible to external impressions, as is evinced in the marked manifestations of disturbed function that are produced by what, in the more mature individual, would be considered trifling affairs. The etiology of convulsions is consequently much more extensive in infancy and childhood than later in life, and it is very doubtful if it should include dentition.

Jacobi[1] remarks, "We must not forget that peripheral irritability increases from the fifth to the ninth month considerably, and that the inhibitory centres do not perform all their functions as in the adult. Thus it is even possible that now and then a convulsion will occur; but, so far as I am concerned, I have not seen convulsions dependent upon difficult dentition in the course of the last ten years."

In the majority of cases convulsions are traceable to some irritation in the alimentary canal. Rachitic children are peculiarly liable to convulsions. In some cases the most painstaking examination fails to reveal the cause of the convulsion; and a careful and thorough autopsy may even be barren of results: so that it is much better to acknowledge candidly that occasionally we are unable to determine the cause of a convulsion than to attribute it to some presumed cause for want of a more real one.

Cutaneous eruptions—notably, eczema, lichen, urticaria, and impetigo—are very common between the sixth and twenty-fourth months, and, like diarrhœa and convulsions, may appear contemporaneously with the cutting of a tooth.

The delicate and sensitive nature of the child's skin renders it susceptible to disorders from slight irritation. Inherited or acquired predisposition, derangement of the digestive organs, usually from some fault in the diet, some disturbance of the nervous system (not always to be accounted for even in the adult), lack of cleanliness, immoderate bathing, the use of strongly alkaline soaps or impure toilet-powder, rough handling in washing, drying, or dressing the child, irritation from the clothing because of either its quality or its arrangement, but not dentition, may give rise to cutaneous eruptions.

Since the first dentition has been considered the source of so much local and constitutional trouble during the first two years of life, it is only natural that in the second dentition should be sought an explanation of many of the disturbances occurring between the sixth and twenty-first years.

The various forms of stomatitis, tonsillitis, sore throat, gastro-intestinal derangements, febrile disturbances, bronchitis, internal rhinitis, diseases of the eye, of the ear, of the skin, chorea, epilepsy, neuralgia of the fifth nerve, facial paralysis, spastic contraction of the muscles of mastication, tetanus, aphonia, hysteria, etc., have all been imputed to the second dentition.

The fact that a dead tooth in the jaw is not infrequently a cause of some reflex disturbance, as neuralgia or a local paralysis, is offered in support of the belief that the above-mentioned maladies can have an origin in dentition. But it is difficult to perceive an analogy between the two conditions. There is a vital connection between a growing tooth and the structure in which it is enclosed, with an harmonious adjustment of its growth and the absorption of superimposed tissues; whereas the root of a dead tooth retained in the gum is a foreign body, likely at any time to set up local or reflected disturbance.

---

[1] Intestinal Diseases of Infancy and Childhood.

Generally there is no pain or gingivitis attending the eruption of the permanent teeth. Even the eruption of a tooth at an abnormal point, because of either lack of room in the dental arch or faulty direction of the tooth-germ, is, as a rule, unaccompanied by pain, uneasiness, or local inflammatory action. The eruption of a wisdom-tooth is, however, not infrequently attended with pain or discomfort. In such cases there is usually insufficient space between the jaws at the back part of the mouth, a result of imperfect development of the bones consequent on rachitis in early life, so that as the tooth advances towards the surface the overlying gum is subjected to enormous pressure each time the jaws are closed, as in the act of biting, and a, sometimes severe, gingivitis is the result. The irritation of the gum by the rough edge of a milk-tooth that is about ready to drop off sometimes produces inflammation and ulceration of the gum and contiguous parts.

MANAGEMENT.

Tradition furnishes many absurd notions and superstitions in relation to the care of children during the dentition epoch. It is said that the mothers of Brittany will not touch their infants' gums, lest the teeth grow crooked. It was also said that the first teeth must not be thrown away when they drop out, for if any animal got such a trophy the next tooth would be like that of the animal finding the old one. To facilitate the eruption of the teeth and lessen the severity of concomitant ailments, various nonsensical and obnoxious procedures have at one time or another been held in high repute,—necklaces of beads made of amber or different roots placed on the child's neck, daily frictions of the gums with the fresh brains of hares, or with blood from the recently-wounded cock's comb, unguents, lotions, and hard and soft substances.

The modern treatment of dentition with many practitioners comprises the use of teething-rings, drugging with the bromides or opium,—the mother very often administers the opium on her own responsibility in the form of a death-dealing soothing-syrup,—local applications of laudanum or cocaine to the gums, and the unlimited and unwarrantable use of the gum-lancet. The management of the child should, in fact, begin before conception. Healthy, vigorous, and normally-developed parents beget healthy children. Freedom from hereditary taint, and proper care as regards diet and hygiene, will insure the child against most of the ills of the dentition period. In a large city, however, it is next to impossible to carry out all the requirements necessary to the welfare of the child. An unnatural environment increases the susceptibility of the infant organism to functional inharmony.

The clothing, particularly that in contact with the skin, should be well fitting, made of some soft and non-irritating material, and in amount suitable to the season. An abundance of undisturbed sleep, plenty of fresh air and sunshine, and cleanliness, are indispensable to the well-being of the child. In the use of the bath anything that may irritate the skin, as too hot water, impure soap, coarse wash-cloths or towels, or rough handling, must be scrupulously avoided.

Too much care cannot be bestowed on the nourishment of the child. It is a well-known fact that the maternal milk of each species of mammalia, because of certain chemical and physical properties, is peculiarly adapted to the sustenance of its own particular progeny, and the lacteal secretion of any other race proves but a most imperfect substitute. This is exemplified in the feeding of very young infants on pure cow's milk. So that, unless contra-indicated by some constitutional disorder that cannot be speedily removed, as syphilis (?) or phthisis, the mother should, if her breasts contain milk, nurse her own baby, observing proper intervals, two, three, or four hours according to age, in so doing. Occasionally between nursings the child should have water to drink.

The indication for weaning is the presence of at least twelve teeth in the mouth. Solid food may then be gradually added to the child's diet; but milk should still be the principal aliment until the second year.

When for any reason it is impracticable to supply the infant with the milk of its mother or that of a wet-nurse, cow's milk in a modified form must be substituted. A discussion of the very important subject of the artificial feeding of infants will be found in the article on Infant Feeding, to which the reader is referred.

The slight ailments of infancy should always receive immediate and proper attention. A mild diarrhœa must never be regarded as salutary unless it be due to the irritation of some indigestible substance in the intestines. Nature may then be assisted in removing the offending matter, after which the diarrhœa must be checked. When a group of indefinite symptoms, as fever, restlessness, and fretfulness, loss of appetite, etc., appear in a child, and are not traceable to derangement of the digestive organs, a careful and thorough examination of the whole body, and particularly of the thoracic organs, should be made daily until a correct diagnosis can be reached. Very often cases of so-called difficult dentition turn out to be nothing less than pneumonia, pleurisy, diphtheria, or some other serious malady, much to the surprise of the parents and to the mortification of the medical attendant. When dentition is delayed, the child's general condition requires attention. Errors in feeding must be corrected, and cod-liver oil, alone or in combination with small doses of phosphorus, administered. There are no local measures that can facilitate the eruption of the teeth. Nor, indeed, are any necessary. A teething-ring furnishes the child with something on which to exercise its masticatory muscles, and, if clean and perfectly smooth on the surface, is probably harmless. But it does not hasten the absorption of the gum. The vile practice of some mothers of supplying children with cake and sugar-teats for constant sucking is both filthy and injurious. The fermenting sugar is a cause of sprue, stomatitis, digestive derangements, and caries of the milk-teeth.

It is not many years since the gum-lancet was considered the catholicon for nearly all infantile maladies. At the present day it has fallen into well-merited disrepute. As J. Lewis Smith remarks, it is used more by the ignorant practitioner, who is deficient in the ability to diagnosticate obscure diseases, than by one of intelligence, who can discern more clearly the true pathological state. Gum-lancing is not objectionable because of pain, the possibility of severe hemorrhage, injury of the tooth, or any difficulty attending the operation, but because it is an absolutely useless procedure so far as it affects the eruption of a tooth. There is no tension of the overlying gum; for an incision, whether linear, crescentic, or crucial, does not gape. A pretty story is related of a child having experienced such great relief, on one occasion, from having her gum lanced, that, not feeling well, at a subsequent visit of her benefactor, she toddled over to him with a table-knife in her hand and by various gestures expressed a desire to have the operation repeated. Marvellous, if true. Every one has heard of cases where a cutaneous eruption disappeared, convulsions ceased, diarrhœa stopped, fever abated, irritableness subsided, and quiet slumber ensued after the gums had been lanced. But it is more than likely that in every instance it was a pure coincidence, or the result of the hemorrhage (a blood-letting), or of imagination on the part of those interested. Every intelligent observer knows that infantile derangements frequently get well spontaneously,—good ground for being sceptical about many of our vaunted therapeutic measures.

PUBERTY: ITS PATHOLOGY AND HYGIENE.

By THOMAS MORE MADDEN, M.D., F.R.C.S.

Puberty has been defined as the period of life within which reproductive capacity becomes established. But, as in many instances that development does not occur throughout the whole course of existence,—however long protracted and in other respects normal it may be,—in the following observations the term puberty will be used as signifying merely the epoch intervening between childhood and adult age or manhood. Under ordinary circumstances, this period is marked by the evolution of the organs of generation, together with those protean physiological changes and new etiological relations that are connected therewith. So important and complex are the latter that of all the successive stages of growth, maturity, and decay, into which the brief span of human existence is biologically divisible, there is perhaps no one epoch the pathological aspects of which are of such frequent interest to the medical practitioner as that which forms the subject of the present memoir.

In infancy and childhood the vital powers are occupied exclusively with the nutrition and growth of organs essential to the existence of the individual. During puberty, on the other hand, in addition to this, there now, as a rule, occurs the still more remarkable evolution, structural and functional, which controls the perpetuation of our species. The physiological actions which are necessary for this object are, as was well observed by Dr. Roget, "great and commensurate with the magnitude and importance of the design," and they give rise to that rapid and varied succession of changes, mental as well as physical, which are essential for the perfected development of that marvellous trophy of creative power,—"the living microcosm of man's body."

Nor are these developmental changes purely physiological, but, on the contrary, inasmuch as "the seeds of death are inseparably intermixed with the germs of life," they are closely connected with, or productive of, numerous special pathological proclivities or tendencies to disease, which will be separately considered in the succeeding pages.

Circumstances affecting Evolution of Puberty.—The age within which the vital changes usually included in the term puberty may take place does not admit of any rigid limitation, as their occurrence is neces-

[illegible] or family temperament, consti-[illegible] and circumstances of life, in each [illegible] of disease, and above all by the [illegible] the possibility of more than a [illegible] in reference to the normal date of

[illegible] **Puberty in Females.**—The advent of [illegible] the first appearance of the catamenia, [illegible] in warm climates, sanguine tempera-[illegible]rious states of society, and is retarded [illegible] this climate the period of first men-[illegible]en by the table on the opposite page, [illegible]fluence of the various extrinsic or acci-[illegible]. Thus, to say nothing of the precocious [illegible]rmal in more sunny regions, and of which [illegible] during my long residence in Southern Spain [illegible] Africa and other tropical or semi-tropical [illegible] have observed cases of regular menstruation [illegible] year, and have attended the accouchement, [illegible]teen. Many other similar and still more [illegible] reproductive power in this climate are well [illegible] recorded by Dr. Macnaughton Jones, in his [illegible] Women" (London, 1888), of maternity at the [illegible] cases mentioned in the *British Medical* [illegible] year.

[illegible]stance of the same kind may be found in the [illegible] 1888, in which an account is given of a case [illegible] of a young girl who began to menstruate [illegible] two years: the menses took place up to a short [illegible]sion, and lasted for five days. The child was [illegible] end of twelve months had cut all its teeth. In [illegible] pounds, and now in its sixth year it weighs [illegible] time ago it was robust and well. The head and [illegible] to those of children of its age, but the buttocks [illegible]veloped. The breasts are prominent, and the [illegible] with hair. The child speaks in a bass [illegible] 1888, the health of the child continued good, but [illegible]sed; at each menstrual period the child is [illegible], and the latter have tended to become [illegible] and in severity.

[illegible] in cold or temperate climates such as ours the [illegible]rded beyond the usual period. In several [illegible] first appearance of the catamenia in persons [illegible], and in one of them this function was not [illegible] the patient in her twenty-sixth year, nor did she then again menstruate until after her confinement in the following year, from which time she menstruated regularly.

In reference to the ordinary period of the commencement of puberty, I may here avail myself of a table taken from a former article of mine in Dr. Quain's "Dictionary of Medicine," showing the result of my own inquiries into the date of first menstruation. This investigation, I may add, extended over a considerable period and a large field of inquiry, having been commenced during my connection with the Rotunda Lying-in Hospital, and subsequently being continued in the gynæcological wards of the great institution to which I have been attached for the last twelve years. Small as the results may appear, they were not obtained without some difficulty, as in the great majority of cases the statements of those whose menstrual history was investigated proved so indefinite or unreliable that in only an infinitesimal proportion of them—namely, in four hundred and ninety-seven instances—was I able to obtain any accurate data on this point. In these latter cases the ages at which menstruation first occurred were as follows:

Under	12	years	of age	4	menstruated	for the	first time.
At	12	"	"	17	"	"	"
"	13	"	"	50	"	"	"
"	14	"	"	94	"	"	"
"	15	"	"	138	"	"	"
"	16	"	"	105	"	"	"
"	17	"	"	65	"	"	"
"	18	"	"	10	"	"	"
Over	18	"	"	14	"	"	"

From the foregoing table it appears that of four hundred and ninety-seven cases where the date of the first catamenial period was ascertained, menstruation occurred between the fifteenth and seventeenth years in three hundred and thirty-seven instances, and that in this triennial period its first manifestation most commonly took place at the sixteenth year, which may therefore be regarded as the average normal date of the commencement of female puberty.

Evolution of Female Puberty considered.—The transition from girlhood to puberty, the normal date of which has been just referred to, is, notwithstanding the far greater complexity of the physiological changes involved, much more direct and sudden in its accomplishment than is the case with the corresponding period in the opposite sex. In the primary stages of life the functional differences between the sexes are comparatively slightly marked, but on the occurrence of puberty in the female these become sharply accentuated, and are denoted by the sudden development of the reproductive or sexual organization, including the accessory parts, such as the mammæ and external genitals, as well as the essential organs of generation, and more especially the enlargement of the ovaries, the maturation of their Graafian follicles and contained ova, and, in fine, the evolution of the entire utero-ovarian system, the predominant influence of which on the general economy is tersely summed up in the old aphorism, "*Propter uterum est mulier.*" From this moment the girl passes at once from childhood to full procreative maturity, as evinced by the establishment of menstruation. This function, which results from the processes of ovulation and uterine denudation, leads to that periodic sanguineous discharge by the regular monthly recurrence of which, during the ensuing thirty years or so of life, the term of woman's distinctive sexual or reproductive vitality is measurable.

Period of Puberty in the Male.—The commencement of this epoch in man is less definite in its characteristics, and in the age of its occurrence, than is the case with the opposite sex. In Great Britain, and I believe a similar law generally prevails in the United States, a boy is not legally considered as arrived at puberty until the age of fourteen, when supposed sexual capacity and legal responsibility for the crime of rape commence.

By the old Roman law, however, another and a better standard of adolescence was provided, this term being thereby considered synonymous with the period at which liability to military service began,—namely, at the age of fifteen, the ordinary date at which the physiological change from boyhood to puberty occurs in all temperate climates. The approach of this epoch is now denoted by a characteristic modulation of the voice, which becomes altered from "the thin childish treble to the deep manly base," caused by the development of the larynx and vocal cords, the enlargement of the pomum Adami, and the elongation of the thyroid cartilage and the thyro-arytenoid muscles. About the same time is also noticeable the first appearance of that downy growth on the face, so fondly watched and cultivated by its proud possessor as the badge of emancipation from the pedagogue's stern rule, and the evidence of the advent of the bright spring-time of life, when

> "A young man's fancy lightly turns to thoughts of love."

There now also occur the growth of hair on the pubes, etc., the commencement of the structural and functional development of the testes and other parts of the genital organs, and too often the abnormally early first manifestation of the sexual instincts. These successive changes, however, proceed so gradually that their full completion is not accomplished until some years have elapsed, and is often delayed until long after the legal term of manhood has been attained.

Premature Puberty in Males.—Although, as already observed, the vital changes connected with the transition from childhood to adolescence are, under ordinary circumstances, seldom accomplished before the sixteenth year, and are frequently delayed until a much later period of life, occasionally this customary course is departed from, and in these fortunately exceptional instances the whole system, physical and mental, or, as more frequently happens, particular powers or organs, become prematurely developed at an abnormally early age. Thus, numberless instances of mental precocity are on record, from the time of "the Admirable Crichton" down to the present day, which has been so prolific in over-gifted children,—such as Joseph Hoffman, Pape, and the many other infant prodigies whose exhibitions of precocious musical or histrionic talent have recently proved so attractive to the sensation-seeking audiences of the Old and New Worlds.

If the mental faculties may in such cases be thus early developed, with an almost absolute certainty of their subsequent failure at a correspondingly untimely age, it is not to be wondered at that a like extraordinary precocity should in some unfortunate instances exhibit itself in a premature evolution of the sexual functions, the unhappy subjects of which, instead of growing up with gradually-increasing vigor to the possession of a healthy manhood, sink into a premature old age, mentally imbecile and physically decrepit at what should normally have been the period of vital maturity.

In the writings of the older physiologists many examples of physical as well as mental precocity, and of their generally untoward termination, may be found. A most interesting collection of such cases is contained in Dr. Mason Good's erudite but now forgotten "Study of Medicine;" and, as this work is not generally accessible at the present day, a reference may be here permissible to some of these, taken principally from the *Journal des Sçavans* for 1688 and the *Philosophical Transactions* for 1745. In the former Boiset gives an instance of this disgusting anticipation in a boy of three years; in the latter, the subject in the case recorded was two years and eleven months old. Similar examples at a similar age may be found, together with various others, minutely described in the first volume of the *Medico-Chirurgical Transactions*. In the year 1748, Mr. Dawkes, a surgeon at St. Ives, near Huntingdon, published a small tract, called "Prodigium Willinghamense," or an account of a surprising boy who was buried at Willingham, near Cambridge, upon whom he wrote the following epitaph: "Stop, traveller, and wondering know, here buried lie the remains of Thomas, son of Thomas and Margaret Hall, who, not one year old, had the signs of manhood; not three, was almost four feet high; endued with uncommon strength, a just proportion of parts, and a stupendous voice; before six he died, as it were, of an advanced age. He was born at this village, October 31, 1741, and in the same departed this life, Sept. 3, 1747." See, also, *Phil. Trans.*, 1744–45. As Dr. Elliotson has observed, this perfectly authentic case removes all doubts respecting the boy at Salamis mentioned by Pliny (Hist. Nat., lib. vii. c. 17) as being four feet high and having reached puberty when only three years old, and respecting the man seen by Craterus, the brother of Antigonus (cited in Blumenbach's Physiology, 4th edition, p. 535), who in seven years was an infant, a youth, an adult, a father, an old man, and a corpse!

Diseases of Puberty.—Of the various factors in the etiology of disease, there is none more obvious in its effects than the influence of age in the causation of the chief maladies to which each period of life is specially susceptible, and which seldom occur at other epochs. "Thus," as Dr. Eliotson remarked, "we rarely see gout in an infant, nor is it common for old persons to have the symptoms of acute hydrocephalus." This elective affinity of certain disorders for particular ages is strikingly exemplified during puberty by the special tendencies then manifest in both sexes to the development of strumous or tuberculous disorders and gastro-intestinal complaints, as well as by the various acute inflammatory and hemorrhagic diseases—pulmonary, cerebral, and hepatic—which are then so prevalent; whilst in females the special pathological proclivities accompanying puberty are, as will be seen in the next section, still more directly connected with the newly-developed functional activity of the utero-ovarian system.

Special Disorders of Female Puberty.—The various morbid susceptibilities or special predispositions to disease connected with the establishment of menstruation have been elsewhere discussed by myself as well as by countless other writers since the time when they were clearly described by Sir Thomas Laycock and Dr. Williams. Many, indeed, and serious, as the latter points out, are the evils liable to be produced by external causes which check the development of this function. So also, when established, this function has its nervous as well as vascular relations, and where it is disordered or irregular a predisposition is given to various maladies affecting the blood-vessels and their contents, the secreting organs, and the nervous system. It would be impossible to discuss the pathological relations of the special function that marks the establishment of female puberty without some reference to the physiological processes by which this evolution is accomplished, and to any disturbance in which—whether by excess, diminution, or arrest—the complaints to be now considered may, in many instances, be traceable.

Some doubt has been thrown by Mr. Lawson Tait and some other recent writers on the hitherto generally accepted doctrine, to which I myself still adhere,—viz., that menstruation is dependent on ovarian action or ovulation; and hence, as the question is one bearing directly on the pathology of female puberty, I shall here briefly recapitulate some of my former observations on this point.

The chief characteristic of the change from girlhood to puberty, which in our climate generally occurs at the fifteenth year of age or thereabouts, consists in the regular establishment of that periodic action of menstruation for the accomplishment of which the conjoint functional activity of the ovaries, Fallopian tubes, and uterus is essential. This process commences in ovulation, or the maturation of a Graafian follicle, followed by the escape of the contained ovum and its transmission by the Fallopian tube into the uterus, whereupon there also occurs a disintegration, or shedding of the endo-uterine lining membrane, which, the subjacent surface thus unsealed, leads to a hemorrhagic exudation or discharge per vaginam, amounting to six or eight ounces, and extending over a period of from three to five days. Immediately before this catamenial epoch, the patient suffers from more or less general malaise, languor, and heaviness; she is indisposed to exertion, and complains of pain in the back and loins, and down the thighs. Occasionally there is uneasiness and a sense of constriction in the throat and about the thyroid gland. There is a peculiar dark shade over the countenance, and especially underneath the eyes. The cutaneous perspiration and breath have a faint sickly odor. The mammæ are enlarged and often painful, digestion is somewhat impaired, and the appetite is fastidious. After these symptoms have been present for a day or two, under normal circumstances the menses appear, and the uneasiness subsides.

In a large number of cases, however, the nervous disturbances connected with the establishment of menstruation are of a more serious nature than in those just referred to, and these will now be considered.

Hysterical Disorders of Puberty.—The frequent occurrence of hysterical and other cerebro-nervous disorders in females about the age of puberty is evidently strictly consequent on the complex structural and functional changes then in process in the reproductive system, the predominant influence of which is manifest in every vital action from the dawn of puberty until the termination of the period when utero-gestation is possible. The commencement of this epoch is marked by a sudden and complete revolution in the female mental as well as physical constitution. At each succeeding ovulation there also is a coincident recurrence of constitutional and nervous disturbance acting on the general economy through the wide-spread ramifications of the vaso-motor sympathetic system, so that comparatively few women whilst menstruating can be said to enjoy the *mens sana in corpore sano* in their integrity.

When menstruation has become established, and is regular in every respect, the accompanying nervous disturbance may be so slight as to escape observation. But the earlier catamenial periods, as well as every subsequent deviation from normal menstruation, are so frequently attended with some manifestation of hysteria that under the guise of nearly every complaint then incidental to female youth, and whether the trouble be spinal, cardiac, pulmonary, or indeed any of those obscure complaints common at that age, and for which no obvious physical evidence is apparent, the experienced practitioner may very frequently be able to trace the *fons et origo malorum* to the sympathetic nervous disturbances that are connected with the evolution of puberty. It need scarcely be added, however, that, whilst thus prepared to meet with the protean forms of hysteria, simulating and complicating the most common diseases prevalent during this epoch, the physician must be no less forewarned against the much graver error of ignoring or neglecting the obscure evidences of actual physical disease in any patient, however hysterical she may be.

It would be impossible, within the limits of this article, to refer here in any way to the widely-extended list of authors, of every age and country, by whom the hysterical disorders of puberty have been described. Still, as a matter of literary rather than of practical interest, I may venture to cite the brief allusion made to this subject by the earliest of them, viz., Hippocrates, who observes, "Nubile virgins, particularly about the menstrual periods, are affected with epileptic paroxysms, apoplexies, and groundless fears and fancies." He attributes these to a congestion about the heart and diaphragm ("noble parts"). "When these organs are oppressed, rigors and feverishness supervene; the patient raves about the acute inflammation, cries out on account of putridity; is terrified and anxious on account of her dimness of vision; and, from the oppression about the heart, thinks suffocation is impending. The mind is harassed by anxiety and weakness, and becomes diseased. The patients call out in great alarm, desire to leap down or throw themselves headlong into pits, and order themselves to be strangled, as if it were a thing beyond all others to be desired. Spectres haunt them, and they earnestly long for death, as for a pleasure. The disease is easily cured if nothing retard the flow of the menses. To those young females affected by it, I recommend that they marry as quickly as possible, for if they conceive they will escape the disease. Unless this be done, they are sure to suffer from it, about or a little after puberty." In another part of these writings ("De Morbis Mulierum," lib. i.) is a graphic description of the aggravated form: in this ischuria, spinal and abdominal tenderness, tympanites, aphonia, syncope, etc., were observed, just as they are at present in such cases.

At the present time the forms of hysterical disease which are commonly observed in connection with the evolution of puberty, though less aggravated than those described by Hippocrates, are nevertheless of sufficient importance to deserve more consideration than is generally accorded to them. Thus, even the ordinary hysterical paroxysm associated with early menstrual derangement, and usually regarded as too trivial to require any special medical care, may be an indication of serious utero-ovarian irritation or disease, the neglect of which may possibly eventuate in the gravest forms of cerebro-nervous disorder,—viz., epilepsy and insanity.

The Voice in Hysteria.—As a general indication of hysteria, the changed character of the patient's voice in such cases may be mentioned. This alteration consists in a loss of that peculiar softness and melody which distinguish the female from the male voice. In hysteria the patient's intonation either becomes more rough and masculine than normal, or else becomes more shrill and piercing or metallic than usual, as well as more rapid in the sequence of its modulations. The hysteric voice is not easily described, but once recognized it is, I believe, an unmistakable evidence of nervous functional disturbance consequent on some derangement of the utero-ovarian functions.

The earlier nervous symptoms that frequently occur at puberty may for a time be unrecognized, but as the local disease progresses these come into such prominence as in many cases to obscure all the evidences of their physical exciting cause. The most important of these manifestations of hysteria are increased nervous susceptibility, or general hyperæsthesia, and diminution of inhibitory nerve-force, together with perverted moral or mental excitability, and in some cases actual delusions.

Hysterical hyperæsthesia is more frequently coexistent with amenorrhœa, or dysmenorrhœa, resulting from uterine disease or displacement, than with any pathological increase in this function.

Hysterical Insanity.—The connection between mental disease and menstrual disorders, more especially amenorrhœa, has been frequently observed by alienists as well as by gynæcologists. Thus, in a case related by Pinel, a girl suffering from insanity was placed under his care shortly before the ordinary age of puberty, which passed over without the occurrence of the usual changes connected with this period. After a considerable lapse of years, however, one day on rising from bed she ran and embraced her mother, exclaiming, "I am well!" The catamenia had just flowed for the first time, and her reason was restored, both the mental and reproductive systems thenceforth permanently resuming their normal functional conditions.

Hysterical Epilepsy.—As I pointed out in a former memoir of mine, "On the Cerebro-Nervous Disorders peculiar to Women," this disease is frequently observed in girls of an hysterical temperament at the period of puberty, nor can I call to mind a single case of any of the different forms of epilepsy or hystero-epilepsy, so met with, that was not accompanied with some derangement, and more generally suppression, of the menstrual functions.

Hysterical Trance.—Hysterical evidences of utero-ovarian disorder, connected with the evolution of puberty, may also manifest themselves by diminished nervous activity and general or local anæsthesia, and as well by the opposite condition. Perhaps the most remarkable illustration of this fact is afforded by hysterical trance, or cataphora, in which the ordinary phenomena of vitality are apparently suspended by a morbid condition, undistinguishable in some instances from death. A brief account of some instances of lethargy of this character that have come within my experience will perhaps best serve to illustrate the general course of these interesting cases. Fuller details may be found in two papers of mine on this subject in the *Dublin Journal of Medical Science.*

The first of the following cases is an instance of so-called hysteric trance. A young lady, Miss R., recently arrived at puberty, of an hysterical temperament, but otherwise apparently in perfect health, went into her room after luncheon to make some change of dress. A few minutes afterwards she was found lying on her bed in a profound sleep, from which she could not be awakened. When I first saw her, twenty-four hours later, she was then still sleeping tranquilly, the decubitus being dorsal, respiration scarcely perceptible, pulse 70 and extremely small; her face was pallid, lips motionless, and the extremities very cold. At this moment so death-like was her aspect that a casual observer might have doubted the possibility of the vital spark still lingering in that apparently inanimate frame, on which no external stimulus seemed to produce any sensorial impression, with the exception, however, that the pupils responded to light. Sinapisms were applied over the heart and to the legs, where they were left on until vesication was occasioned without causing any evidence of pain. Faradization was also resorted to without effect.

In this state she remained from the evening of the 31st of December until the afternoon of the 3d of January, when the pulse became completely imperceptible, the surface of the body was icy cold, the respiratory movements apparently ceased, and her condition was to all outward appearance undistinguishable from death. Under the influence of repeated hypodermic injections of sulphuric ether and other remedies, however, she rallied somewhat, and her pulse and temperature again improved. But she still slept on until the morning of the 9th, when she suddenly woke up, and, to the great astonishment of those about her, called for her clothes, which had been removed from their ordinary place, and wanted to come down to breakfast, without the least consciousness of what had occurred. Her recovery, I may add, was rapid and complete.

In the second instance of the same kind that I have seen, the patient, after a lethargic sleep of twenty-seven days, recovered consciousness for a few hours, and then relapsed into her former comatose condition, in which she died.

In another case of hysteric lethargy in a young lady under my care, the trance lasted for seventy hours, during which the flickering vital spark was preserved from extinction only by the involuntary action of the spinal and ganglionic nervous centres. In this instance the patient finally recovered.

The last instance of profound cataphora or lethargy that has come within my own observation occurred last autumn in the Mater Misericordiæ Hospital, in the case of a young woman under the care of my distinguished colleague Dr. Boyd. In that instance, despite all that medical skill could suggest or unremitting attention could do, it was found impossible to arouse the patient from the apparently hysterical lethargic sleep in which she ultimately sank and died.

I have referred to the foregoing cases, occurring in one physician's experience, as disproving the general opinion that hysterical lethargy or trance is so rarely met with, and is then of such trivial pathological importance as to be of little if any practical interest. On the contrary, from my own experience, I can vouch that these conditions are of far more frequent occurrence than is generally supposed to be the case, as well as for the fact that all the ordinary external signs of apparent death may occasionally be thus counterfeited with wonderful similitude. I would therefore take this opportunity of urging the necessity of bearing this in view, so as to avoid what I fear is the not infrequent possibility of living interment in some cases of too hurried burial under such circumstances,—a calamity the horrors of which, I may here repeat, no effort of imagination can exaggerate, and for the prevention of which no pains can be excessive and no precautions superfluous.

Hysterical Paralysis.—In many instances the nervous symptoms of utero-ovarian functional disturbance at the period of puberty may also be manifested in the simulation of every form of paralysis, from the most trivial local loss of power to complete paraplegia. Of the latter I recently met with a well-marked example in the case of a young lady, aged nineteen, who had never menstruated, and who, when I first saw her, had been for nearly eighteen months confined to bed with apparent complete loss of power of the extremities. During this period she had been actively treated by several practitioners, by whom she had been alternately submitted to faradization, the various nerve-tonics, blistering, cold and hot baths and douches, as well as ultimately being enclosed in a plaster jacket, to remedy the supposed spinal cause of her condition. None of these remedies, however, proved of the smallest use until, after an interval of nearly two years from the commencement of the attack, her changes for the first time made their appearance, and from that date she rapidly regained her former health and strength.

Menstrual Disorders of Puberty.—The normal course of the evolution of puberty is specially liable to derangements arising from the various morbid conditions by which the due performance of the function of menstruation may be interfered with. This disturbance is most frequently occasioned by amenorrhœa, or the total absence or diminution of the catamenial discharge; secondly, by dysmenorrhœa, or the difficult and painful accomplishment of this function; and thirdly, and less commonly, by menorrhagia, or abnormal activity in the utero-ovarian changes connected with ovulation, and consequent excess in the resulting menstrual discharge. But although the effects of these disorders are more marked during puberty than perhaps at any subsequent epoch, inasmuch as their occurrence is by no means restricted to this period, it would be beyond the scope of the present article to attempt any discussion of their general pathology and treatment.

With regard to the first-named of these disorders—viz., amenorrhœa—it may, however, be here observed that very undue importance is commonly ascribed to the non-appearance of menstruation at the usual age, or to its subsequent interruption or diminution, as the supposed general cause of nearly all the ills to which female flesh, about the period of puberty, is heir. In the great majority of the cases of amenorrhœa for which at this epoch we are so frequently consulted by anxious mothers, the functional irregularity is merely symptomatic of systemic morbid conditions, to the rational treatment of which, by appropriate constitutional remedies, rather than to any futile, if not injurious, utero-ovarian or local stimulation, the efforts of the physician should in such cases be directed.

Dysmenorrhœa is hardly less frequently associated than amenorrhœa with the special hysterical and other constitutional disorders incidental to puberty. Under these circumstances, difficult menstruation, although occasionally resulting from uterine flexions or displacements, or from stenosis or other obstructive causes, as well as from local inflammatory conditions, is far more commonly merely a complication of coexisting general nervous disorder or constitutional hyperæsthesia, on the cure of which the dysmenorrhœal trouble will at once subside.

In this connection I may add a word of warning against the popular custom, so prevalent among all classes, of treating the dysmenorrhœa of puberty by wine or brandy. From long experience I am convinced that, as I have elsewhere observed, intemperance in women may very frequently be traced back to the first painful menstrual period, when alcoholic stimulants are often forced on the young sufferer. The pain of dysmenorrhœa being thus relieved, the girl at the next similar epoch naturally and no longer reluctantly seeks the same solace, until in this way the victim of dysmenorrhœal alcoholism may gradually become an habitual and perhaps an incurable drunkard.

Space forbids any further allusion in this place to the disorders of menstruation, to the influence of which the period of puberty is so susceptible. In the next section we must, however, refer at greater length to a condition generally connected with menstrual derangements, and the occurrence of which is peculiar to the epoch that forms the subject of the present article.

Chlorosis—or chloranæmia, as green-sickness is more properly termed—is the most frequent of all the morbid conditions specially incidental to female puberty. This complaint may be regarded as a specific form of anæmia, the aglobulism in these cases being primarily dependent on a neurosis of the ganglionic nervous system, and as a rule is connected with either amenorrhœa or dysmenorrhœa. The history of the disease, its symptoms, and the line of treatment by which these may be relieved, all point to the accuracy of Kuchenmeister's conclusion,—viz., that the essential cause of the chlorotic condition is the retention of carbonic acid in the blood. This theory is sustained by the fact that the chlorotic are very commonly persons of the poorer classes, who have been subjected to privation of fresh air, sunlight, and exercise, and in whom by the consequent diminution of pulmonary exhalation, aided by the lessened menstrual evacuation in such cases, the blood is surcharged with carbonic acid as well as poor in red corpuscles.

Chlorosis is characterized not so much by the sallow or slaty pallor of the cutaneous surface (for this is not essential to the disease, and the skin may be deadly white, without a greenish tinge), as by a universal and decided debility of the whole frame, and sometimes even a degree of torpor of particular organs. There is a general weakness of the muscular system, and weariness and languor of body, with listlessness of mind, the patient being indisposed for any exertion, easily overcome by fatigue, nervous, low-spirited, and frequently a prey to singularities of temper. There is generally severe recurrent headache or vertigo, and sometimes an impaired state of the memory and of the faculty of attention; the sleep is disturbed, the chlorotic sufferer being either preternaturally wakeful or abnormally drowsy. The eye, in well-marked cases, is dull and heavy. The lips and tongue are at first exsanguine and pallid, and subsequently present a peculiar slaty hue. The temperature, more especially that of the extremities, is depressed. The pulse is small and weak, often rapid, and easily fluttered. There is frequently palpitation, recurring in attacks, or of a more permanent character; more frequently still, there is a sense of sinking in the præcordia, with irregular action of the heart, or imperfect syncope. There is usually a degree of breathlessness experienced on any exertion; sometimes fits of dyspnœa; sometimes a sonorous cough. The appetite is abnormal; occasionally it is morbidly increased, but more usually anorexia is present, and the patient loathes food, or is sick after eating, or much troubled with flatulence and gastrodynia. Often there is a desire for indigestible substances, particularly chalk, magnesia, or even cinders. The bowels are costive, often obstinately so; or, if not, the stools are dark and offensive. The abdomen is not uncommonly tumid, swollen, and variable in size. The hands and feet swell at night, with œdema of the eyelids, if not of the whole face, particularly in the morning. The urine is scanty, though clear.

In addition to the foregoing, many other of those obscure symptoms which in girls are so frequently met with about the time of puberty may also be found connected with chlorosis. Of this kind is that severe left-side pain, otherwise inexplicable, so often complained of at this age, as well as those intense nervous headaches, breathlessness, and, in fine, that host of hysterical symptoms by which all the features of organic and functional disease, whether pulmonary, gastric, or cardiac, may be simulated.

Treatment.—With regard to the treatment of chlorosis I would here repeat that our attention should be primarily directed to the rectification of that error of digestion which is a chief cause of the characteristic aglobulism, and, secondly, to the depuration of the vitiated blood by the excretory organs, rather than, as is too often done in such cases, to the restoration of the catamenial discharge, the suppression of which should be regarded as merely a symptom—albeit a primary and most important one—of the constitutional disorder.

In accordance with this view, the rational treatment of chlorosis must therefore be approached by means capable of strengthening the general system, and more especially improving the tone of the organs of digestion and excretion. For the first purpose, open-air exercise, free exposure to sunlight, and suitable food are obviously more essential than any of the pharmaceutical resources at our command. Even if our main object were the cure of the amenorrhœa connected with the chlorotic condition, and even if we had medicines more certainly emmenagogue than we possess, we might, when we succeeded in this, attribute our success chiefly to such means as tend to improve the general health and strength. We would, then, recommend regular exercise, proportioned to the ability of the patient; the use of the warm or tepid salt-water bath every day, succeeded by friction with dry flannel or a soft brush; sufficient clothing, and particularly a flannel dress; a nourishing and digestible diet; the administration of bitter and tonic medicines in varied forms, preparations of iron, such as chalybeate waters, tincture of muriated iron, or the subcarbonate of iron, alone or combined with myrrh, or sulphate of iron with quinine, or a grain of iodide of iron in a bitter infusion, and arsenic.

The use of chalybeate mineral waters, internally as well as externally, is of self-evident service in the chlorotic state, unless the patient be of a full habit, in which case purgatives must be premised, and afterwards conjoined, so far as necessary.

Tuberculosis and Strumous Disorders of Puberty.—Having discussed in the preceding sections the most important of the special disorders directly connected with the organic and functional evolution of the reproductive system, we must now briefly consider some other forms of disease to which the period of puberty is specially liable, even although their occurrence is not limited to this age and their etiology must in some instances be sought in causes operating at an antecedent epoch. Of these maladies the most important in this connection are the various forms of strumo-tuberculous complaints which constitute so large a proportion of the diseases of puberty.

Within the last few years the frequency of such affections, as observed in my hospital practice, has become notably increased. The explanation of this fact must, I think, be looked for in circumstances preceding the development of puberty, and is mainly referable to the dietetic and hygienic mismanagement of childhood, more especially to the frequent employment of unsuitable condiments, such as tinned and other artificial so-called milk-preparations as substitutes for the natural food essential for the healthy nutrition of children.

The acute forms of tuberculosis which are most common during youth have been observed by Cohnheim and Klebs to resemble the infective diseases in their zymotic origin from a specific virus, whether generated in the body from caseous matter or introduced from without. The latter is probably generally the case in the tubercular diseases so common among the children of the poorer classes, into whose dietary tinned or preserved milk now enters largely; for there can, I think, be no guarantee that the cows furnishing this supply are not suffering from *perlsucht*, or bovine tuberculosis, as the disease is very prevalent and does not materially affect the quantity of milk.

The foregoing views as to the frequent origin of tuberculosis from infected milk, to which I called attention in "The Transactions of the International Medical Congress of 1881" (vol. iv.), have been strongly corroborated by the recent researches of Prof. Bollinger (*Wiener Medizin. Presse*, September 16, 1888). Regarding the frequency of tuberculosis, Prof. Bollinger maintains that in large cities from forty to fifty per cent. of all deaths may be attributed to this disease. Recent experiments in his laboratory show that milk may prove infectious, whether taken from cows suffering with local or from those suffering with general tuberculosis.

As in other infectious diseases, the quantity of tuberculous matèrial introduced into the economy strongly influences the severity of infection. Only a few drops of undiluted milk from a tuberculous cow proved sufficient to produce typical miliary tuberculosis in animals, but when this quantity underwent any material dilution its effects were negative. This latter observation would suggest the use of milk taken from many cows rather than from one cow. The non-infectiousness of meat from tuberculous animals was proved by taking the muscles of twelve tuberculous cows and injecting the liquid obtained into the peritoneal cavities of sixteen guinea-pigs: no tuberculosis resulted.

With regard to the curability of tuberculosis, Bollinger refers to many necropsies made on individuals in whom lesions of a former tuberculosis are found.

Inoculations made with material obtained from twelve capsulated caseous patches on twenty-six animals resulted in the development of tuberculosis in twenty of the animals. The experimental results show that in twenty-six suspected cases of tuberculosis of the pulmonary apices, seven only (twenty-seven per cent.) were non-infectious. For the microscopical anatomical diagnosis the fact is important, that all apical affections of the lung are to be regarded as infectious, as long as caseous or caseous-calcareous patches are present. Those cases only can be denominated as cured in which simple cicatrices are present, with or without calcareous infiltration.

Another cause of the increasing frequency of scrofulous and tubercular disorders is the physical deterioration of our people arising from that widespread intemperance which is almost as general among women as it is among men, and the consequent toxicological effect of alcoholism on the wretched offspring of these drunken parents, who further pay the penalty of their progenitors' excesses by the development of scrofula and tuberculosis as the result of semi-starvation and neglect during the first years of life. Probably also local circumstances have much to do with the prevalence of scrofulous and tubercular diseases in other large towns as well as in Dublin. Nor can there be any doubt that the situation of that city in the low-lying, badly-drained valley of the Liffey, and the densely-inhabited squalid tenements, too generally devoid of the most necessary sanitary requirements, in which the poorest class of a poverty-stricken population are crowded together, have a deteriorating influence on the physical condition of the ill-lodged, ill-fed, and ill-clad scrofulous children who from thence recruit our hospitals and prematurely fill our cemeteries.

It would be impossible to consider here so wide a question as the relation of scrofula to tuberculosis; but I may venture to reiterate my adherence to the older doctrine on this subject, which I have elsewhere discussed, and which was first impressed on my mind when a student in the scrofula-haunted hospitals of Algiers,—namely, that the scrofulous diathesis is the prolific and primary source of all tuberculous disease, whatever part of the body may be thus affected, whether it be the lungs, the meninges or substance of the brain or spinal cord, the mesenteric glands, the cancellous structure or articulating surfaces of the bones, or the external glandular system.

In my work on "Change of Climate," the first edition of which was published many years ago, I dwelt on the evident connection between scrofula and phthisis, and also pointed out the contagious or infectious character of these diseases under certain conditions. I may therefore be permitted to claim some interest in finding similar views now adopted on these questions by the most eminent recent pathologists.

One of the most frequent forms of pulmonary tubercular disease that come under treatment at the Dublin Children's Hospital is miliary tuberculosis, or, as the old writers well termed it, acute or galloping consumption. In many instances I have seen miliary tuberculization of the lungs pass through all its stages, from its first recognition until the patient's death, within less than a month. The rapidity of the race towards death, and the accompanying similar tubercular infiltration of the meninges and substance of the brain, peritoneum, liver, etc., in such cases, leave little room to question the fact that acute tuberculosis is essentially an auto-infective disease. Within the last few years a new light has been thrown on the causes and method of development of tubercular disorders, concerning which, until recently, the views of Buhl as to their origin from auto-inoculation with caseous matter in the body were generally accepted. This doctrine has been disturbed by the discovery by Koch (in 1882) of the specific bacillus of tubercle, and by the more recent researches of other pathologists in the same direction, which enable us in some measure to understand the extraordinary rapidity with which pulmonary tuberculosis too often supervenes on an attack of broncho-pneumonia, particularly in strumous patients at the age of puberty. Nor is it to be wondered at if, in children thus previously enfeebled by diathesis, the struggle for existence between the specific micro-organisms of disease and the colorless blood-corpuscles or leucocytes, which have been recently graphically described by Dr. Latham and by Metschinkoff, should so speedily result in favor of the almost incredibly rapidly-multiplying bacilli.

Treatment.—Be the pathogenesis of tuberculosis what it may, there can, I think, be no question of the fact that the disease is most generally developed at puberty in patients of an otherwise evidently strumous diathesis, and that its primary predisposing cause is generally traceable to malnutrition, general or local, in such cases. Under these circumstances, and bearing in mind the facts ascertained by recent investigations just referred to, it is obvious that our primary therapeutic efforts should be directed towards endeavoring so to enrich or improve the condition of the circulating fluid as to increase its capability of resisting and destroying the micro-organisms by which tuberculosis is developed, and that for this purpose we must seek to rectify any existing error of nutrition that may result from defective nutriment as well as from impaired powers of digestion and assimilation. These indications should be borne practically in view in the selection, for such patients, of a dietary not only easy of digestion and assimilation, but also specially rich in those elements needed to strengthen the constitution against the inroads of the prolific micro-organisms by which tuberculosis is developed. The arrangement of that dietary must, however, be so largely controlled by the circumstances of each case as to render it impossible to lay down any general directions on this point. I may, however, observe that some of the special requirements in this respect of strumo-tubercular youth are to a large extent supplied by the food-medicines with which modern polypharmacy has now armed us for the struggle with tubercular disease, wherein pharmaceutical remedies must be assigned a place entirely subsidiary to hygienic as well as dietetic management, by articles such as cod-liver oil, maltine, and the various preparations of malt. Among the many other remedies of a somewhat similar class which are specially available in these cases, none are more generally serviceable than the officinal syrups of iodide and phosphate of iron, or the many valuable combinations of this with other salts, such as Parrish's, or Squire's, or Dusart's syrup, or that which I have found specially useful in the chronic wasting disorders of tuberculous or strumous youth,—namely, Fellows's syrup of the hypophosphites of iron, lime, and potash,—with other tonics. In this connection I may again observe that as a food-medicine in such cases there is nothing more generally beneficial than Irish moss. This species of algæ (the *Chondrus crispus*), of which the sea-shores of Ireland furnish an inexhaustible supply, was recommended upwards of half a century ago by Dr. Todhunter, of Dublin, as an anti-strumous remedy. But at the present day the value of Irish moss in this way, and as an article of diet, appears generally ignored or unknown; and hence I take this opportunity of bearing my testimony to its utility as an abundant, cheap, easily-prepared, palatable, and generally serviceable article of food for those suffering from any of the chronic diseases which are connected with the scrofulous diathesis during puberty.

In the way of medicine, iodine is still the only drug for which anything like a specific property can be claimed in these cases; and the reason that more benefit is not generally derived from its employment is probably that it is now seldom given in the metallic form and in the long-continued minute doses originally advised by Lugol, in whose hands such benefit was obtained from its use.

The hygienic and climatic treatment of the chronic strumo-tubercular disorders of puberty is a subject of considerable practical importance, and, although we can only extend the benefit of favorable hygienic and climatic conditions to a comparatively small class of strumous patients, still, so great are the remedial advantages of such treatment that it obviously demands our careful consideration. On this point I may venture to speak with some confidence, having devoted much attention to the influence of change of climate and the uses of mineral waters in the treatment of chronic scrofulous and tubercular complaints during several years passed in various distant health-resorts, and repeated visits to foreign spas.

Change of climate and the use of mineral waters are especially advantageous in the treatment of the chronic tuberculous and scrofulous diseases of early life, in which no brief course of treatment, however judicious, can be expected to counteract the constitutional effect of years of disease.

The mineral waters most generally serviceable in the chronic disorders of strumous youth are the iodated and bromated saline springs, of which Wildegg in Switzerland is that from which I have found the greatest benefit derived.

The simple chalybeate mineral waters are very generally useful in the management of most of the strumo-tubercular diseases of early life. Waters of this class, containing the carbonate of the protoxide of iron in the most easily assimilated form, with excess of carbonic acid gas, are powerfully tonic and stimulating, increasing directly the number of red corpuscles and the amount of hæmoglobin, both of which are so much diminished in these cases.

The thermal arseniated waters are also especially beneficial in the treatment of the strumous disorders of puberty, as well as in the anomalous hysteric affections of chlorotic girls at this age.

Cold sulphurous waters are also serviceable in some forms of these chronic affections. But the thermal springs of this class, although occasionally prescribed with much benefit in cases of scrofulous complaints (being powerfully stimulating in their action on the vascular and cerebrospinal nervous system), always require great caution in their use, and are especially contra-indicated in cases of active tuberculous disease.

In change of climate we have another, and, as I believe, a most effectual, remedy for chronic tubercular disease. There are no cases in which the beneficial influence of change of climate may be so confidently hoped for as in youth of either sex predisposed to consumption by the scrofulous diathesis. In such cases the constitution, being yet unformed, may be expected to receive and retain whatever impression a pure, bracing, or mild air can produce, and thus they may be enabled to pass safely the critical period intervening between childhood and puberty. The climates suitable for this predisposition to phthisis should be dry, moderately warm, and bracing,—dryness of atmosphere being the essential condition.

At present the health-resorts most in vogue for this class of patients, and in many cases very judiciously selected for them, are the cold, dry, tonic climates of Alpine districts, such as Davos-Platz, in the elevated tableland of the Upper Engadine, and other similarly situated mountain sanitaria, of which there are several in the United States as well as in the Old World, —the Adirondack Mountains in New York, Asheville in North Carolina, and numerous elevated regions of Colorado and the Rocky Mountains.

The primary effect of a cold, dry climate and of a pure mountain-atmosphere is unquestionably tonic and bracing in the case of a still moderately strong albeit strumous youth, in whom a tendency to tuberculous disease exists, and has been fostered by life in the impure, variable, and humid atmosphere of any of our overcrowded centres of population. When such a patient is removed from one of these hot-beds of tuberculosis to a dry, cold climate, the respiration becomes more energetic, more oxygen being required, and more carbonic acid exhaled, to supply which increased food, especially of a fatty character, is consumed. Thus the blood is directly enriched, the proportion of hæmoglobin and red corpuscles, the oxygen-carriers, being augmented, and the general nutrition of the system is improved. But, on the other hand, those whose constitutions have been longer and more seriously affected by the scrofulous cachexia or its local manifestations must be injured by exposure to the cold rarefied atmosphere of an Alpine height. In such a climate, if the patient's vital energies had been previously lowered by chronic disease, the blood repelled from the surface of the body by cold must be driven back on the internal organs; and if active pulmonary disease be developed, then the diminished pressure of the atmosphere in these Alpine health-resorts, and the sudden and frequent variations in the electrical condition and temperature, as well as the pressure of the air to which such places are necessarily subject, must affect the balance of the circulation, and produce more or less pulmonary irritation, if not congestion or hæmoptysis.

Hence, from extensive personal experience of the effects of change of climate, I would venture to deprecate the too general adoption of mountain sanitaria in all cases of consumption which is now coming into fashion; nor indeed does it need any such experience to show that change of climate, like all other remedies, must be prescribed on rational principles and with due regard to the special condition of each individual patient.

In selecting a winter residence for children suffering from, or predisposed to, any chronic tuberculous disease, primary consideration should be given to the comparative facilities and inducements for open-air exercise which are afforded by different health-resorts. As a general rule, consumptive children are disinclined or unable to undergo any fatigue, and love to hang over the fire, whence their friends fear to disturb them, "lest they might catch fresh cold." But how mistaken this view is needs no argument: for such children free exposure to pure fresh air and sunlight are all-essential. Hence, in choosing a health-resort in these cases, preference must be given to whatever place the climate of which will permit, with safety, of the maximum exposure to the open air, and the situation of which will afford the greatest inducements and opportunities for out-door exercise.

General Hygiene and Culture of Puberty.—It would be difficult to overestimate the practical importance of the physico-moral management and training of "the spring-time of life," as this epoch is aptly termed. For within its limits must be implanted the fructifying seeds of health, mental, moral, and physical, by which alone the future well-being of the individual may be assured; or else, on the other hand, will be then sown the no less potent morbific germs by whose development the sanitary integrity of mind or body must eventually be impaired or destroyed.

The pathological influence on the course of puberty of some of the latter agencies was well described by the late Dr. James Johnson, of London, in his "Economy of Health." As, however, that work is long out of print, and has been largely used without acknowledgment by some recent writers, I may here briefly recapitulate a few observations which appear specially applicable to the circumstances by which the health of puberty is most frequently thus modified.

"It is," remarks the writer just named, "in this stage of rapid development, corporal and mental, that the greatest difficulty is experienced in preserving the *physique* within the bounds of health and confining the *morale* within the limits of virtue. How many minds are wrecked, how many constitutions ruined, during the third septennial! At so early a period of life, when passions so much predominate over principles, it is hardly to be expected that the force of precept can be so efficient a preventive as the fear of bodily suffering. If the youth of both sexes could see through the vista of future years, and there behold the catalogue of afflictions and sufferings inseparable attendants on time and humanity, they would pause ere they added to the number by originating maladies at a period when Nature is endeavoring to fortify the material fabric against the influence of those that must necessarily assail us in the progress of life! Yet it is in this very epoch that some of the most deadly seeds of vice and disease are implanted in our spiritual and corporeal constitutions,—seeds which not merely 'grow with our growth and strengthen with our strength,' but acquire vigor from our weakness and obtain victory in our decay. This melancholy reflection is applicable to all classes and both sexes. The sedentary and insalutary avocations to which young people of both sexes in the middle and lower classes of society are confined, between the ages of fourteen and twenty-one, occasion dreadful havoc in health and no small deterioration of morals. The drudgery, scanty clothing, bad food, and exposure to the elements, of our laboring or factory population, as well as the still greater miseries of the too numerous unemployed poor in these countries, are but little more injurious to health and life than the sedentary habits, unsanitary surroundings, and depressing passions of the various species of artisans, mechanics, and shopkeepers in the classes immediately above them. The infinite variety of new avocations among these grades has given rise to a corresponding infinity of physical and moral maladies, of which our forefathers were ignorant, and for which it requires much ingenuity at present to invent significant names. The incalculable numbers of young females confined to sedentary avocations from morning till night, and too often from night till morning, become not only unhealthy themselves, but afterwards consign debility and disease to their unfortunate offspring. It is thus that infirmities of body and mind are acquired, multiplied, transmitted from parent to progeny, and consequently perpetuated in society. He would be blind indeed who did not perceive the outward working of these causes in our own day. Nations are only aggregations of individuals, and whatever be the influence, whether good or evil, that operates on a considerable number of the population, that influence will radiate from ten thousand centres, and diffuse its effects, sooner or later, over the whole surface of the community."

In viewing the ascending links of society, there is no great cause for gratulation. The youth of both sexes, doomed to the counter, the desk, and the school-room, are little elevated, in point of salubrity, above their humbler contemporaries. It is during puberty that the destiny of youth is fixed for all the various professions and pursuits, into the training for which the young are now too often prematurely forced by the increasing exigencies of the struggle for existence, wealth, or distinction, in all densely-crowded centres of population. What wonder, then, that under such circumstances the intellectual advantages thus secured are too dearly purchased at the expense of health? The physical stamina, as well as the mental powers, are too frequently thus so overstrained in this fierce competition that both thereby become prematurely exhausted, and if not permanently at least temporarily debilitated and incapacitated for their ordinary functions. These results are, moreover, very commonly consequent on errors in the mental or physical training of children in the period immediately antecedent to puberty, the results of which, being manifest at this epoch, must be here referred to.

The Mental Training or Education of youth and early puberty is a question always of great importance, but of special interest at the present time. We are all, of course, agreed as to the duty of suitably educating the young so as to fit them for the daily increasing requirements and competition of modern life, but as to the extent to which this should be carried in early childhood there is, unfortunately, a great discrepancy between the doctrinaires of the Education Department and the views of those who have any knowledge of the laws of nature or who as physicians have to deal in disease with the consequences of their violation. The red-tape officialism of the former is often supreme over medical experience. And hence, whilst children, before the age of puberty, are thereby overworked into disease or death, the physician must still raise his protesting voice.

The first years of life should be mainly occupied by moral and physical training, and during this period the amount of mental cultivation which a child's brain is capable of receiving with permanent advantage is much less than is commonly believed. No greater physiological mistake is possible than the prevailing idea of attempting any considerable degree of mental culture until the sufficient development of the physical stamina and moral faculties is accomplished. The organ of the mind is as much a part of the body as the hand, and ere either can function properly its vital force must be developed and maintained by nutrition. Hence arises a very important practical question in connection with compulsory elementary education. A large proportion of those who must come within the provisions of the law in most large cities are ill-fed children of the poorest classes, and are those with whom for the past sixteen years I have daily had to deal in the Hospital for Sick Children. As a matter of fact, I may accordingly observe that children thus debilitated by privation are necessarily as much incapacitated for any mental strain as they are for the accomplishment of any feat of physical strength, and that it is as inhuman, injudicious, and impolitic to expect the former as it would be to look for the latter from children so circumstanced.

If, therefore, the state, for reasons of public policy, determines that all children shall be compulsorily educated from their earliest years, it should certainly afford the means by which this may be least injuriously and most effectually carried out, by providing sufficient food as well as education for every pauper child compelled to attend school.

Among the results of over-pressure in schools referred to in Sir Crichton Browne's admirable report on this subject, are cerebral diseases in all forms, —viz., cephalitis, cerebritis, meningitis, as well as headache, sleeplessness, neuroses of every kind, and other evidences of cerebro-nervous disorders.

It would be difficult to overestimate the pathological consequences of thus directing all the available energies of the system to the brain during early youth, to the irreparable injury of the over-stimulated cerebral organization, and at the expense of the other functions and organs of the body. Time, however, does not permit of my dwelling on the ill effects of mental over-pressure brought under my own observation, nor of any reference here to the many most painful scenes of misery thus occasioned, with which long and sad experience has made me but too familiar. I now allude to this subject merely with the view of pointing out the imminence of the danger and the importance of its avoidance.

The deterioration of the physical and mental stamina, thus observable, is, as I may repeat, mainly due to the fact that a large part of the first years of life, which should be primarily devoted to religious or moral as well as physical training, is now given up to the development of the mental powers. The child is too early compelled to attend some school where the immature brain is forced into abnormal and disastrous activity.[1]

Ill Results of Sexual Precocity during Puberty.—In no particular are the pathological effects of the killing pace at which the race of life is nowadays too often run, from its start to its untimely finish, more apparent than in the premature break-down of constitution consequent on the abnormally precocious indulgence or abuse of the sexual instincts or appetites. To these abuses, on which (as they will be better considered in other portions of this work) it will not be here necessary to dwell in any detail, is unquestionably due a large and increasing proportion of many of the maladies by which the course of after-life is embittered or its duration shortened. I do not now refer to the specific diseases which at the earliest period of puberty, as at any other epoch, may follow sexual vice, or which may even be manifested long before the normal date of the evolution of the sexual functions, as I have recently seen illustrated in one disgusting instance of primary syphilis in a boy only ten and a half years of age, now in hospital, under the care of my colleague, Mr. Chance.

---

[1] The system of kindergarten now so much in vogue in this country, and the wonderful strides which have been made in physical training, even in our elementary schools, will undoubtedly have the effect of reducing the death-rate from cerebral disease in childhood, and will aid in giving our youth of both sexes sound minds in sound bodies.—EDITOR.

What I particularly here refer to are those remote or secondary constitutional effects of sexual precocity which must be familiar to every experienced physician who in almost daily practice may encounter the wretched, cachectic, and mentally as well as physically debilitated victims of early erotic excesses and abuses. To these causes must, I fear, be largely ascribed the failure of physical stamina, as well as that nervous hyperæsthetic condition and lamentable lack of mental power and determination of character too often noticeable among youth of the present day, and which clearly mark the Nemesis of a wide-spread epidemic of precocious sensuality. The means by which this epidemic may be best mitigated is, however, a subject which cannot be adequately here considered, as its treatment is beyond the reach of the physician, and belongs rather to the domain of the moral teacher or the minister of religion.

Consequences of Abuse of Alcohol and Tobacco during Puberty.—Another phase of the results of the too common untimely abridgment of early puberty by precocious indulgence in the habits and vices of adult life is exemplified by the painful exhibitions of juvenile drunkenness daily witnessed, especially among the neglected street Arabs, who during and even before the first stage of puberty are forced into the thoroughfares of our great cities, there to eke out a living as best they may, and the pathological consequences of whose acquired or inherited alcoholism are brought under clinical observation in the form of gastric and hepatic disorders, and especially cirrhosis of the liver, as well as in the protean forms of cerebro-spinal disease, and the various neuroses which are so frequently noticed in hospitals for children.

In the *British Medical Journal* and elsewhere I have reported several instances of juvenile alcoholism that came under my care in the Children's Hospital, and latterly some deaths from this cause have occurred among mere lads. In the majority of cases of juvenile alcoholism this tendency appears inherited, and is most marked in those whose mothers were inebriates,—intemperance in women also bearing in other ways on the diseases treated in hospitals for children, where its effects are strikingly evinced by the moral and physical deterioration of the offspring of the drunken, and by their special predisposition to strumous, tubercular, and other constitutional taints.

The evil thus resulting from the prevailing intemperance of the young as well as the old should induce us to warn those whom our counsel may influence against that custom of giving alcoholic stimulants to children, which is so general in its practice among all classes and so calamitous in its results. Even in those exceptional cases in which such stimulants may be necessary, we should never sanction their administration save under the guise and in the definite doses of other remedial agents; and my experience in hospital and private practice, at home and abroad, has amply confirmed the view expressed in a work of mine published many years since, that it is physiologically wrong, as well as morally unjustifiable, ever to allow a healthy youth to taste alcohol in any form.

With regard to the effects of the abuse of tobacco during early puberty, of which we see so many instances, especially among the neglected children of the poor, I may refer to an observation I long before made on the stunted and prematurely aged appearance of children in Portugal, where smoking is indulged in from the earliest possible age. There, in the streets of Lisbon, I have often seen with astonishment boys obviously much under the age of puberty gravely sucking a strong cigar with apparently the same gusto which our less precocious progeny derive from the forbidden delights of the sugar-stick. There can be no doubt that the influence of the nicotine thus absorbed must be most injurious at this age; and this is evident in the physical aspect of the youths referred to.[1]

Special Hygiene and Culture of Female Puberty: its Practical Importance.—In a previous section we have described the special functional disorders incident to female puberty, and must now refer to certain other causes of those various nervous and constitutional ailments that are prevalent during this period. Of these causes some, although of a moral rather than of a physical character, are yet so intimately connected with the production and course of the complaints referred to as to demand a brief notice.

Among the subjects thus included in this connection are the influences on female health in puberty of the moral, mental, and physical training or education during or immediately before this period; secondly, the ill effects of the customary modes of dress and habits and occupations of girls at this time; thirdly, the results of premature or abnormal stimulation of the sexual system, whether by too early marital life or in any other way at that age. As was observed by Dr. Johnson, female life at any period, *cæteris paribus*, is fully as good, in respect to probable duration, as that of the male,—perhaps even a little better. In this point of view woman has a longer senectitude than man. More men are annually born than women, and consequently more must die. It is in the period of puberty that the seeds of female diseases are chiefly sown, or at least that the soil is specially prepared for their reception and growth. The predisposition to infirmities and disorders of various kinds is affected by acts of omission and commission,—in the first class being included the deficiency of healthy exercise of the body in the open air, and of intellectual exercise in judicious studies. The ill results of these are perhaps most apparent among girls of the upper classes of society.

---

[1] Dr. More Madden on "Change of Climate in the Treatment of Chronic Disease," 3d edit., London, 1874.

The increasing exigencies of modern life, and the desire to render girls accomplished at all hazards, have originated a system of forced mental training, which greatly increases the irritability of the brain, whilst at the same time sedentary employments are followed frequently as amusements, to the exclusion of active exercise out of doors. The slow but powerful influences of music, dancing, vivid colors, and odors, on the nervous system, but especially on the reproductive system, are quite overlooked. Many hours of severe application are occupied in the acquisition of pieces of music which are forgotten as soon as possible after marriage, when music would be least hurtful, or rather most useful. Dr. J. Johnson very justly asks, "Is it probable that so potent an excitant as music can be daily applied for many hours to the sensitive system of female youth with impunity?" The same writer points out that "the stimulus of music is of a very subtle and diffusible nature, and the excitement which it produces in the nervous system is of a peculiar character, and one by no means generally understood." Any excessive exposure to this potent stimulation is therefore liable to be productive of some of the various hyperæsthetic morbid conditions of mind and body so prevalent during the period of female puberty.

The excessive attention given to music in female education is, moreover, indirectly hurtful by not leaving sufficient time for other and more serviceable employments of mind and body, by which the former may be strengthened against the vicissitudes of fortune and the moral crosses to which female life is doomed, nor for healthful physical exercise, by which the material fabric may be fortified against the thousand causes of disease continually assailing it. The consequence of all this is, that the young female too often returns from school to her home an hysterical, wayward, capricious girl, imbecile in mind, habits, and pursuits, prone to hysteric paroxysms upon any excessive mental excitement. This, I may add, appears very liable to be superinduced by the pernicious novels of the erotic and sensational school, which are the popular literature of young females, and by which the impressionable mind of girlhood is perverted, the passions stimulated, and the foundations laid for the future development of various morbid conditions of mind and body, and more especially erotomania and nymphomania.

I shall not now refer to the latter of these disorders. As to the former, I shall only repeat that it may be regarded as an exaggeration of the peculiar sentimentality which is generally inherent in female youth, and which is usually so evanescent as to require little if any medical attention. In some instances, however, this excess of natural sentiment is of graver consequences, the mind becoming so occupied by its predominant illusion as to impair, more or less completely and permanently, the exercise of the rational faculties, and not alone produce mental derangement but also react injuriously on the general health and more especially on the utero-ovarian functions of the love-sick girl. Instances of this kind are familiar to nearly every practitioner. There are few among us who have not been consulted by some anxious mother, alarmed by symptoms of mental dejection and nervous or mental functional disturbances, for which no physical cause can be discovered, arising from cardiac causes beyond stethoscopic diagnosis. This condition is, in its inception, entirely distinct from erotomania, but if allowed to develop unchecked may in some instances ultimately result in the latter.

If neglected mental training is of so much account in the causation of hysterical disorders, on the other hand to the excessive and misdirected application of the female mind may in many instances be traced the origin of the morbid nervous conditions already discussed.

The robust unmarried female in easy circumstances may escape many of these evils, but after the age of eighteen the reproductive organs are fully, probably largely, developed, and strong passions, indolence, and luxury fail not to produce their effects on the system, and to develop some form of hysteria.

The transition from girlhood in those of the more affluent classes of society is generally accompanied by a complete revolution in the patient's habits and mode of life, which now become changed from the early hours and open-air exercise and simpler fare of the girls' school to the too luxurious living, late hours, constrained movements, and unsanitary dress of the *débutante* in the world of fashion.

The Influence of Dress on the physical health of female youth is unquestionable, and this subject has accordingly been fully discussed by countless authorities, although the practical result of all that has been thus written has apparently been *nil.* The two cardinal points to be here borne in mind with reference to female clothing are, first, that the material should be such as may serve to retain the necessary animal warmth, and, secondly, that its form be so arranged as to occasion neither undue visceral compression nor any interference with impeded muscular action. Hence, whatever little influence the physician may exercise in this matter should be employed to induce his clients at this epoch to adopt underclothing of flannel or merino, or Jaeger's undyed woollen fabric, as well as to persuade them to eschew those dearly-prized compressing corsets, elastic garters, and tightly-fitting high-heeled boots, by which young ladies seek to reduce their natural proportions, however robust, and at whatever cost of comfort or health, within the limits prescribed as "the pink of fashion and the mould of form." This advice we should give whenever the occasion offers, seldom as our counsel may be followed, for in such matters fashion and the *modiste* will probably continue to the end of the chapter to reign triumphant over common sense and the doctor.

The injurious consequences of the absurd modes prevalent in the dress of female youth are exemplified in the effects of tight lacing on the pulmonary functions, for the normal accomplishment of which free expansion of the chest and unimpeded action of all the muscles connected with respiration are so essential. The results of errors of this kind are most apparent at the period of puberty, when the young lady exchanges the comparatively easy garb of girlhood for that imposed by the requirements of fashionable life. And these errors reach their extreme in the attire of the ball-room or theatre, or what, on the *lucus a non lucendo* principle, is now regarded as *full* dress. "At these assemblies," as Dr. Barlow has well observed, "the tightly-laced stays, the exposed chest, and thin draperies furnish a combination of influences, the combined effects of which no constitution could withstand; while to these is yet to be added that of respiring for hours a heated and vitiated atmosphere, and, after this, of passing, when relaxed and exhausted, into the cold currents of a frosty night air. So far from wondering that many suffer from these egregious imprudences, our surprise should be that any escape; and instead of the inherent delicacy so often imputed to the constitution of females as explanatory of their peculiar ailments, we have ample proof, in their powers of resisting such noxious influences, that they possess conservative energies not inferior to those of the most robust male. Were men to be so laced, so imperfectly exercised, so inadequately clothed, so suffocated, so exposed, their superiority of bodily vigor would soon cease to have any existence."

Defect of clothing, though most signal in the chest and shoulders, is not confined to the upper part of the body. The feet require warmth, which subservience to fashion prevents. They cannot be compressed but at the cost of much suffering, some distortion, and the infliction of positive disease. Fashion also permits the legs to be covered with only the thinnest materials. Thus the capillary circulation of the feet, rendered sufficiently languid by the general weakness, becomes further impeded by the pressure of tight shoes and the debilitating effects of cold. The crippled state, too, thus occasioned, is a further obstacle to efficient exercise, and so adds to the general debility.

Occupation in Relation to Health of Female Puberty.—Formerly the period of puberty in women was less subject than the same epoch in the opposite sex to some of the predisposing causes of reflex cerebro-nervous disorders other than those connected with their utero-ovarian health. This, however, is hardly the case at the present day, in which women are not only liable to those special functional derangements productive of nervous disturbances, but moreover in too many cases are also now exposed to all the accidental causes of cerebro-nervous disorders to which formerly only the ruder sex was subject.

General Treatment of Nervous Disorders of Female Puberty.—It would be beyond the scope of this article to discuss in any detail even the general principles which should guide us in the treatment of the protean forms of nervous disorder incident to female puberty, and to some of the causes of which we have just referred. I may, however, notice a few points which appear to me applicable to the management of the ordinary phases of hysterical disease at this period. I would, in the first place, observe that, under these circumstances, any special local uterine treatment should be avoided as far as possible, and is seldom either necessary or expedient in these cases, unless in those exceptional instances where the hysterical symptoms are the reflex effects of some uterine displacement.

Foremost among the remedies by which we may hope to allay the perverted molecular activity of the nerve-centres in the hysterical disorders of puberty are the various special nerve-sedatives, more especially the different bromides and the valerianates of quinine and zinc. Mere hypnotics are of little value, and narcotics, more particularly opium and its alkaloids, are worse than useless, for this purpose. The curative effects of change of climate and the utility of chalybeate and other mineral and thermal waters, though obvious in all chronic diseases, are in none so essential as in the nervous and hysterical disorders of puberty. In such cases, by the very journey to a foreign and distant health-resort the patient is afforded the benefit of change of climate, occupation, and mode of living. The new scenes and variety of places suggest new thoughts, by which the attention of the hysterical girl is diverted from her morbid fancies and exaggerated sensations, until at length, by ceasing to dwell on her self-created complaints, they gradually may cease to trouble her.

It may be observed that no cases so much demand the exercise of the highest qualities of the physician as those now under consideration. In such instances the practitioner must, above all, rise above any narrow gynæcological or other specialism. He should, of course, seek to remove any local disease or to restore any disordered function of which the hysterical disturbance may be the result. But, as already remarked, in doing this he should also carefully avoid the imminent possibility of increasing whatever vaginal, uterine, or ovarian hyperæsthesia may be present by any topical examination or treatment that is not absolutely and obviously indispensable.

In dealing with those suffering from any of the hysterical conditions of puberty now referred to, the physician should strive to act upon the moral as well as the physical constitution of his patients. He must, therefore, insist on healthy occupation of mind and body, and fit the latter for this by the appropriate remedies called for by the special requirements of each case. If the nervous derangement is consequent on disordered menstruation, this condition must, if possible, be rectified. If it results from premature or undue stimulation of the sexual organs, he must point out distinctly the physical and moral evils consequent on such abuses.

In conclusion, it only remains for me to add that in the foregoing attempt to describe the many-sided medical aspects of the epoch of puberty (concerning some of which my views have already elsewhere been published) my object has been accuracy rather than originality. Hence I shall rest content if adjudged to have been in any wise successful in the humble task of welding together in a homogeneous form the *dejecta fragmenta* of special knowledge on the subjects included in this article, from whatever sources these might be collected, and supplemented, as far as possible, by the light of my own experience.

PART II.

FEVERS AND MIASMATIC DISEASES.

FEVER.

GENERAL CONSIDERATIONS AND TREATMENT.—SIMPLE CONTINUED FEVER.—THERMIC FEVER.

BY WILLIAM PASTEUR, M.D., F.R.C.P.

Definition and Nature.—The term "fever" is used in its widest sense as denoting a complex of symptoms or group of symptoms of which heightened temperature is the most striking and the most constant.

Disorder, then,—in the direction of increase,—of the body-heat, is the essential condition of fever.

In health the maintenance of a normal temperature involves three co-operant factors,—a source of heat, channels for the discharge of heat, and a regulating mechanism which shall maintain a stable balance between heat-production and heat-loss. Owing to the integrity of this mechanism, any variations in heat-production are immediately compensated by concurrent variations in heat-discharge, so that the temperature of the body, as a whole, is not appreciably affected.

In fever, heat-production and heat-loss are both increased, and it has also been demonstrated that the febrile rise of temperature cannot satisfactorily be accounted for by increased production alone, or by temporary diminution of heat-loss alone (retention-theory of fever). It has been shown experimentally that during fever the variations of the two processes are no longer interdependent,—that the regulating mechanism is out of gear. In consequence of this the rates of heat-production and heat-loss vary irregularly, so that the elevation of temperature cannot be regarded as a true measure either of increase of the former or of diminution of the latter. Under circumstances determining a diminution of heat-loss, a high temperature may coexist with a low rate of heat-production; and conversely, if heat is being rapidly parted with, there may be a considerable increase of heat-production without any commensurate elevation of temperature. So that we must conclude that although the clinical thermometer affords unmistakable evidence of some derangement of the heat-maintaining apparatus, it throws little or no light on the nature or direction of the disturbance. It is true that in the adult the height of the temperature is to some extent an index of the severity of the disease; but I shall have occasion to point out that in children, for various reasons, no such reliance can be placed on this indication.

In fever, as in health, the source of heat is the same, at least in kind. It is stated with admirable clearness by Professor Forster: "We may at once affirm that the heat of the body is generated by the oxidation not of any particular substance, but of the tissues at large. Wherever metabolism of protoplasm is going on, heat is being set free. . . . In growth and in repair, in the deposition of new material, in the transformation of lifeless pabulum into living tissue, in the constructive metabolism of the body, heat may be undoubtedly to a certain extent absorbed and rendered latent; the energy of the construction may be in part, at least, supplied by the heat present. But all this, and more than this,—namely, the heat present in a potential form in the substances so built up into the tissue,—is lost to the tissue during its destructive metabolism: so that the whole metabolism, the whole cycle of changes from the lifeless pabulum, through the living tissue, back to the lifeless products of vital action, is eminently a source of heat."

Thus it is in increased destructive metabolism that we must look for the origin of fever-heat, and its chief seat is the muscles. In health it is estimated that they yield four-fifths of the whole body-heat, and in fever their relative contribution is probably larger. Both clinical and pathological observations bear witness to the profound manner in which they are affected. Among these may be mentioned the characteristic pains and weakness, the marked wasting, often so striking in the fever of children, and the associated increase of the salts of potash, of urea, and of other nitrogenous substances in the urine.

Recent experiments on animals show good ground for believing that the muscles possess a heat-generating function which is to some extent independent of their motor function and under the control of a twofold nervous mechanism,—the one part exciting thermogenesis, accompanied by destructive metabolism, the other staying thermogenesis and subserving constructive metabolism. "The processes which issue in motion on the one hand and in thermogenesis on the other are of course associated with chemical movements in the muscle, with metabolisms whose terminal steps are the accretion of oxygen and the excretion of carbonic acid and water. . . . The 'contractile stuff' in the muscle is not the same as the 'thermogenic stuff.' Both of them are stored in the muscle: so far as function goes, they *are* the muscle. The store of each can be exhausted by repeated stimulation, but in some cases the thermogenic store sooner than the other. Both can be upbuilt again by the circulating blood, but in some cases the contractile store sooner than the thermogenic. Both the metabolisms are affected by cold, but the thermogenic much sooner and much more intensely than the contractile. We know little of the exact nature of the chemical changes involved in either form of metabolism. Oxygen is taken up in each and carbonic acid is discharged, but the processes passed through between these terminal stages are much more complex than simple oxidations. The evidence rather goes to show that it is the living substance as a whole, contractile stuff and thermogenic stuff, which is continually being decomposed and as continually being recomposed by the blood. The net balance shows only gain of carbonic acid and loss of oxygen, but the nitrogenous parts also of the working substance have in the process undergone partial destruction and equivalent reconstruction. This is in health; but if the reconstructive part of the process is inadequate or absent, the balance of accounts will give evidence of a nitrogenous residuum which is morbid. The muscle-substance will appear to be itself consumed; it will no longer be what I may call merely the circulating medium of consumption. The cast-out nitrogenized molecules of the muscle are not really excretionary in the sense that carbonic acid is excretionary. They have merely failed of that immediate upbuilding into muscle-substance again which is characteristic of the healthy metabolism. This incessant upbuilding of the muscle-substance which is a necessary consequence of the fact that no increase of urea or other nitrogenous matter is produced in the ordinary processes of muscular metabolism must require the expenditure of a certain amount of energy. If in any measure the upbuilding is checked or abolished, so much energy is of course unexpended." (MacAlister.)

This whole line of investigation is most suggestive, but, so long as the interpretation of the results is not perfectly clear, we should be very guarded in drawing far-reaching conclusions.

As regards the amount of heat contributed by the abdominal viscera, we may safely assume that, in so far as it depends on processes connected with the metabolism of foods, it is diminished in fever.

The channels for the dissipation of heat are the skin,—by radiation, conduction, and evaporation,—and the lungs, by evaporation and warming of the expired air. As sweating is not common in the fever of children, evaporation necessarily plays a less important *rôle* than in the fever of adults. The pungency of skin which is often met with may be in part due to this peculiarity. It is usually associated with a preternaturally dry skin and contracted cutaneous vessels, conditions which tend to prevent a rapid loss of heat and therefore favor elevation of the surface-temperature. We are all familiar with the remarkable way in which the aspect and feel of the skin may vary during an attack of simple fever. In less than an hour a pale, dry, pungent skin may become flushed and moist. A febrile blush so intense as to arouse suspicions of an acute exanthem may vanish altogether in a few minutes. Phenomena such as these not only suggest very pointedly that heat-discharge is largely under the control of the nervous system,—acting through the vaso-motor nerves,—but also point with equal emphasis to the profound disturbance of that control. It is probable that about sixty per cent. of the heat leaving the body does so by radiation. The laws of this loss have recently been investigated with striking results. When a portion of skin is uncovered it naturally becomes cooler, but the cooling does not take place according to physical laws. It is found that radiation increases steadily as the temperature falls, until a certain limit is reached; that it varies directly with the activity of the processes of nutrition and metabolism, and is therefore more active in children than in adults; and, further, there is some ground for believing that it is subject to nervous control. These considerations have a practical bearing. If I read them aright, they plead strongly in favor of light clothing and light covering and of cool well-ventilated sick-rooms.

The nature of the relations of the central nervous system to thermogenesis and thermolysis is still very imperfectly understood. Our knowledge in this direction is chiefly based upon experimentation on animals. Careful experiments have demonstrated the presence at the anterior part of the caudate nucleus, near its median convexity, of a tract which, whenever it is stimulated by puncture with a fine needle or by an electric current, gives rise to an increase in the body-temperature which persists for some time and is attended by an increase in the amount of the oxygen absorbed and of the carbonic acid given off by the animal. It has been shown by calorimetric experiments that this pyrexia is not due to vaso-motor disturbance causing retention of heat, but that there is an increase in heat-production. At the same time the pulse- and respiration-rates are raised and the elimination of urea is increased. It appears, therefore, that puncture of the caudate nucleus produces in certain animals a pyrexia which possesses all the essential properties of fever regarded as abnormal elevation of temperature. The value of this experiment in support of the neurotic origin of fever can hardly be overestimated. Some valuable evidence is also forthcoming on the clinical side. In this connection it will suffice to recall the fact that there are on record well-attested cases of cerebral tumor, pontine hemorrhage, softening and hemorrhage in or about the basal ganglia, injuries of the spinal cord, tumors of the spinal meninges and others, in which very high temperatures have been observed,—temperatures for which no other cause was discoverable than the nervous lesion with which the patient was affected.

In this very brief reference to the nature of pyrexia, attention has been directed to the close relation of the nervous system with heat-production and heat-regulation. The interpretation of many facts is still doubtful. But we may accept as beyond question that "there exist in the body chemical processes resulting chiefly in the production of heat; that these processes are under direct control of the nervous system and possibly of special thermal nerves; and, lastly, that there are regions in the central nervous system which are in some way connected with these nerves and through them control the chemical processes resulting in heat-production." (Welch.)

The nervous theory of fever certainly seems to offer the best working hypothesis yet advanced to explain the various phenomena of the disorder. Viewed in this light, the disturbance of the regulating mechanism alone will give rise to irregular risings and fallings of temperature as the independent variations of production and loss are concurrent or the reverse, without wasting, failure of nutrition, or other signs of increased thermogenesis,—thermal ataxia. If both the regulating and heat-producing mechanisms are disturbed, we have true fever, with excessive thermogenesis and wasting. The temperature rises, and after a while the discharging mechanism is set going, and if not itself disordered presently overtakes the work thrown upon it. This is what occurs in ordinary fever. If, however, the thermolytic mechanism is not capable of being stimulated to increased activity by the antecedent excessive thermogenesis, the temperature rises to a dangerous or even fatal height. This is hyperpyrexia. (MacAlister.)

Causes of Fever.—To formulate a classification of the causes of fever which shall be at once simple and comprehensive is, in the present state of our knowledge of the etiology of the various fevers, almost impossible. Experimental investigations on ferments and putrid intoxicants have thrown much light on the nature of inflammatory fever. The discovery and investigation of the so-called cadaveric alkaloids have revealed to us an important group of fever-producing agents. But the nature of the relationship of many of the acute specific febrile diseases to the invasion and multiplication within the body of pathogenic micro-organisms still remains to be solved. In common with ferments and putrid intoxicants, the poisons of the majority of these diseases exert a pyrogenic effect after being received into the circulation. Their precise mode of action is still matter for speculation. It is supposed by some that they act on the nervous system, directly or indirectly; by others, that they lead to increased heat-production by a direct action on the blood and tissues; but even in such an event, as already pointed out, we must assume that the equilibrium of the thermal nervous system is in some way disturbed. It is possible that all pyrogenic substances act by producing a common change in the body.

The first group of fever-producing agents comprises substances which, if not actually identical with physiological ferments, are readily produced by them, independently of the action of bacteria. Some of them are normally present in small amount in the body, others may be produced in the disintegration of extravasated blood or by the abnormal disintegration of tissue, and if absorbed in sufficient quantity or under suitable conditions are held to be the cause of the febrile states known as ferment intoxications. As probable members of this group may be cited cases of the so-called aseptic fever which follow extensive injuries or lacerations in spite of rigorous antiseptic precautions, and the febrile reaction attending subcutaneous injuries and extravasations, especially fractures of the large bones. It is probable that the pyrexia which accompanies certain forms of anæmia and possibly some obscure varieties of simple fever also belong to this category.

The second group comprises substances which are the product of micro-organisms not in themselves pathogenic; that is, not capable of further multiplication when inoculated in pure cultivation into the body. These saprophytic bacteria are very widely distributed both in and out of the body, and may exert their influence in several ways. Their presence in foul wounds leads to the formation of chemical substances which, when absorbed into the circulation, give rise to pyrexia with toxic symptoms,—putrid intoxication. Under this head are included the febrile states which subside after the thorough cleansing of a foul wound, and the whole class of septic fevers which result from the absorption of poisonous substances produced in necrotic or disintegrating tissues or exudations or extravasated blood by the action of purely saprophytic bacteria. Of this nature are probably also the secondary fever of variola and, in part at least, the fever of typhoid after the end of the second week.

Fermentative and putrefactive bacteria are normally present in the alimentary canal, and it is probable that under certain circumstances the products of their activity may, by their absorption, give rise to febrile attacks, which are, however, for the most part of a milder type than those just mentioned. Here the essential morbid conditions appear to be due partly to the character of the ingesta, partly to alterations in the digestive juices. Of this order are doubtless a large number of the transient febrile attacks of children which occur in association with various disorders of digestion. Dentition fever is often cited as a type of pyrexia due to reflex neurosis; but even here evidence is not wanting to show that other causes may be operative. In support of this may be mentioned the occasional enlargement of the glands at the angle of the jaw; the not infrequent association of middle-ear catarrh, indicating an extension of inflammation along the Eustachian tube; and the presence in some instances of a considerable degree of stomatitis.

Of far graver significance are the putrid intoxications which result from the ingestion of substances which have undergone outside the body putrefaction or changes which lead to the formation of ptomaines. These substances have been separated from putrefying materials in states of tolerable purity. Some of them appear to be harmless, but the introduction of others into the circulation is attended by pyrexia with toxic symptoms. They may be absent in advanced decomposition, and in general the most virulent ptomaines are formed in the earliest stages of putrefaction.

There are also differences according to the kind of bacteria present, according to the nature of the substances decomposed, and according to various other circumstances, such as the presence of oxygen, the temperature, etc. Of this nature are some of the cases of poisoning which have been caused by eating unsound meat, fish, cheese, etc. The absorption of poisons of a similar kind, not necessarily the product of pathogenic bacteria, would afford a rational explanation of the symptoms of profound intoxication met with in many cases of diphtheria and hospital sore throat, as well as of the constitutional disturbance which is not uncommon in scarlet fever towards the end of the third week, in association with the onset of nephritis. This view would receive support from the presence in such cases of some local morbid condition, whether inflammatory or necrotic, and would explain the success claimed for local antiseptic measures in preventing or moderating the severity of the symptoms, at least in the two first-named diseases.

In the third group are included, for convenience of classification, the poisons of the acute specific febrile diseases. Of them we know that they are specific,—*i.e.*, that the diseases which they produce, and of which pyrexia is a constant concomitant, never pass the one into the other; that they prevail epidemically or endemically; that they are in large proportion infectious or contagious; that they may gain admission to the body by various routes, some by inoculation, some by the respiratory mucous membrane, some by the gastro-intestinal tract. The nature of many of these poisons is still uncertain.

That micro-organisms are present in the blood and tissues in several of these diseases is universally admitted, and in the case of a very few the evidence adduced in support of a causal relationship may be received as adequate. Such are erysipelas, pyæmia, and diphtheria. But in regard to others competent observers are by no means agreed. Thus, in several quarters the belief is held that the micro-organisms observed in relapsing and malarial fevers are epiphenomena, appearing as a consequence rather than as a cause of the morbid processes constituting the fever; and similar objections have been raised in regard to several others. So that until more unequivocal proof is forthcoming in regard to the nature of their poisons, it would be, to say the least, premature to include all these affections under one generalization, as depending on the invasion and multiplication of pathogenic bacteria within the body.

That there are causes of pyrexia other than the presence of pyrogenic substances in the blood seems beyond question. A passing reference has already been made to the direct effect of certain lesions of the central nervous system on the body-temperature, as well as to the nature of hyperpyrexia. And although reasons have been given for believing that many cases of dentition fever are of inflammatory origin, instances of this and other affections are not wanting in which the older doctrine of peripheral irritation still affords the simplest and apparently the most rational explanation, especially when we consider the instability of temperature-regulation in children.

The rise of temperature which may accompany a fit of crying, a convulsion, or excitement of any kind during convalescence from acute disease, or in weakly children, would also seem to find a place here. Increased muscular activity may be in part responsible in the two first-named, but in all there is a reasonable suspicion of some direct disturbing nervous influence.

It has been maintained that the process of growth may cause pyrexia. The evidence brought forward in support of this belief is, at the best, of a somewhat flimsy order and altogether insufficient. It cannot be doubted, however, that during the period of active growth heat-regulation is highly unstable and the temperature consequently more labile than at other periods of life.

Effects of Fever.—Opinion has varied considerably at different times in regard to the effects of fever on the body. Formerly the view was very generally held that the disorders of function and the morbid tissue-changes which attend fever were entirely due to elevation of temperature; and this opinion found support in experiments made at the time, but under conditions which have since been shown to have somewhat vitiated the conclusions drawn from them. At the present time there is a decided reaction against this view, a reaction which in some quarters goes to the extent not only of denying that there is danger in moderate pyrexia, but also of asserting that a moderate degree of fever should not be checked. Among the causes which have led to this change of opinion the failure of antipyretic treatment to curtail or even to control certain fevers undoubtedly holds a prominent place. Besides this, a careful reconsideration of the experimental evidence in the light of fresh experiments has drawn attention to the important *rôle* played in fever by other factors, such as infection or intoxication. The increased frequency of the respiratory act, the heightened pulse-rate, and possibly constipation, are probably direct effects of the high temperature, whilst the variations in arterial tension and the disorders of the alimentary and nervous systems are in large part dependent on infection or some factor of fever other than high temperature. The muscular pains and weakness are to be looked upon as the expression of abnormal innervation and nutritive changes accompanying thermogenesis, whilst a part only of the increased nitrogenous disintegration of fever is to be ascribed to pyrexia.

With regard to the granular or fatty degeneration of organs, and of the heart in particular, which is generally held to constitute one of the chief dangers to life in prolonged fever, high temperature is probably an important factor, but is certainly not the only one. Similar changes are of not infrequent occurrence in some forms of anæmia, and may be induced by a variety of poisons, with little or no pyrexia. And, further, granular degeneration is more often found after acute specific fevers than in other febrile diseases. In other words, there are strong reasons for suspecting that it bears a close relation to the kind and degree of the infection in any given case.

Whatever may ultimately prove to be the exact share of pyrexia in the causation of the phenomena now under consideration, it appears highly probable that during fever many causes are at work, which may tend to lessen the resisting power of the individual to the injurious effects of prolonged high temperature.

The case of hyperpyrexia is different. Here, without doubt, the main source of danger is the high temperature, which seems to be in great part due to an excessive disturbance of the thermal nervous mechanisms, amounting to almost complete paralysis. Such, no doubt, is the nature of the excessive rise which may usher in death in some diseases,—an indication of commencing dissolution in the higher, more recently evolved, and therefore less stable nervous mechanisms.

Significance of Heightened Temperature in Children.—The temperature of children in health is characterized by a relative instability, which renders it liable to disturbance by a variety of causes, many of them of the most trivial nature. This is to be accounted for partly by the undeveloped state of the nervous system, partly by its state of active growth. A mere nothing will send an infant into a high fever; a very little restores it again to health. But as the child grows its temperature becomes less liable to disturbance, until with years it gradually acquires the stability which distinguishes the temperature of adults. The nervous mechanisms which subserve heat-regulation are, during infancy and childhood, undergoing a progressive evolution towards relative stability. Being at this period of life among the least organized, least automatic mechanisms, they are readily thrown out of gear.

The readiness with which physiological tissue-activity in a child gives place to pathological activity, in the presence of disturbing causes, is also a reason why fever is a frequent concomitant of disease in children. As examples of this tendency may be mentioned acute lymphadenitis, that common cause of fever, and the proneness of inflammation to issue in suppuration.

In the same way instability is the key-note to the peculiarities of the febrile temperature of children. It contrasts with the pyrexia of adults, less on account of any differences in range and height, than because of its striking tendency to present sudden and temporary remissions. It may rise and fall several times in the course of the twenty-four hours. Periods of high and low temperature may alternate in the most uneven and irregular manner. The type or pattern of the chart may vary almost from day to day. The height of the temperature bears little or no relation to the severity of the disease. A trivial cause may send the temperature to 104° F., or even higher, without apparent discomfort to the patient, whereas a fatal case of pneumonia may run its course without the temperature exceeding 102° F. Enough has been said to show that temperature-observations cannot afford the same indications as to diagnosis and prognosis as we are accustomed to derive from them in the fevers of adults. For example, during the second week of typhoid, in striking contrast with the sustained high temperature of adults, the daily remissions may be so marked as to afford no assistance whatever in distinguishing the case from one of tuberculosis, a disease from which the differential diagnosis is often so difficult in the early stages. Nor, assuming the diagnosis of typhoid to be established, would the occurrence of large remissions towards the end of the second week justify us in prognosticating an early defervescence. It is in meningitis and other forms of nervous disease that we encounter irregular and ambiguous temperatures *par excellence.* These cases appear to conform to no rule. In tubercular meningitis low temperatures are perhaps, on the whole, more frequent. Cases of this disease are on record in which the association of repeated convulsion with high temperature-range seems to suggest a causal relation between them. The well-established fact that convulsion *qua* convulsion may cause a rise of several degrees lends some color to the supposition. But that this association is by no means constant is strikingly illustrated by the case of a boy, aged four, who died of tubercular tumor of the cerebellum, with meningitis, after a prolonged illness. The prominent symptoms were repeated convulsion, extreme wasting, optic neuritis, and frequent and prolonged vomiting, yet the temperature never exceeded 99° F. It may fairly be asked whether the height of the temperature in an attack of convulsions possesses any prognostic value. Any rise of temperature due to the convulsion itself speedily subsides on its cessation; and, speaking generally, the higher the temperature the greater the probability that the convulsion is essential rather than symptomatic.

One other point deserves mention. It has been found that the highest and lowest temperatures may occur at almost any time in the twenty-four hours. Consequently, if a chart is to represent with any faithfulness the daily wanderings of the temperature, the observations must be recorded at regular and comparatively short intervals. A morning and evening record may yield a fairly steady temperature when in reality there has been a daily excursion of several degrees.

In young children and infants it is best to take the temperature in the rectum or in the groin. In older children it may be taken in the mouth or axilla, if due precautions are observed. The rectal temperature is about .7° F. higher than that in the axilla and .5° F. higher than that in the mouth.

The following criteria have been laid down on the significance of pyrexia in children: "The pyrexia is good, *cæteris paribus*, which is lower in the morning than in the evening; which is equable or with but slight variations from day to day; which has a single rise and a single fall in the twenty-four hours; and whose lowest morning level approaches the normal line. The pyrexia is bad, *cæteris paribus*, which is highest in the morning; which ascends from evening through the small hours; which has two or more rises or falls for one day and night; which either maintains its level above 103° F. pretty equably for many hours together, or else is very variable from day to day and conformable to no pattern. Add to this—what was early known, yet what of itself would suffice to save the thermometer from ever falling into neglect—that the temperature-register gives the first warning of impending mischief after injuries and surgical operations; that it supplies our sole means of watching and of measuring hyperpyrexia; and that in conjunction with other signs (by no means by itself) it helps to distinguish certain fevers and to estimate their progress and severity. These latter, however, are but occasional uses." (Sturges.)

Stages and Types of Pyrexia.—Three stages are generally recognized,—the initial stage, that of rising temperature; the fastigium, or stage of sustained high temperature; and defervescence, during which the temperature returns to the normal level. The duration and pattern of each stage vary considerably in different diseases and in the same disease according to circumstances. A rapid and continuous rise is the rule in scarlet fever and ague, whilst in measles and typhoid fever the rise is more gradual and often broken by a series of remissions. A severe convulsion at the onset may intensify the initial rise; severe vomiting may delay or even lessen it. The temperature of children during the acme or fastigium is chiefly noticeable on account of the frequency of remissions. Crisis is the more common mode of defervescence. The actual crisis may be preceded by one or more sharp and deep remissions; this is sometimes seen in acute pneumonia. Typhoid fever affords a good example of defervescence by lysis. After defervescence the temperature is often subnormal for a few days, and is characterized during convalescence by its greater instability and liability to disturbance by causes which would make little or no impression on the temperature of a healthy child.

In the terms continued, remittent, and intermittent, which are still in common use, we have a survival of the nomenclature of an age in which fever was regarded as a disease—a morbid entity—presenting different types. In the present time these terms are used to qualify the pattern of a pyrexia, rather than as a basis of classification. They are too well known to need special description. The remittent type is especially common in the fevers of children. Hectic fever is the name given to the remittent or intermittent fever which occurs in some wasting diseases, more especially when these are accompanied by chronic suppuration with profuse discharge of pus. It is often present in pulmonary and in abdominal tuberculosis, with or without ulceration. In the earlier stages there may be intermissions during the day with febrile disturbances towards evening. As the disease progresses, the fever assumes a remittent type, with exacerbations at night and perhaps in the morning. The rise of temperature may be preceded by chilliness and end in a profuse sweat, especially about the head and shoulders.

Symptoms of Fever.—Prodromal symptoms may be present, but are often absent or pass unnoticed. They are peevishness or apathy, distaste for food, languor, and sometimes headache in older children. The onset is often quite sudden.

An initial rigor is very rare, even in septicæmia, acute necrosis, and ague. It is generally absent in erysipelas and in the eruptive fevers and pneumonia. Older children occasionally complain of chilliness. Dusky pallor of the face and lips, with cold extremities and burning heat of the body, is not uncommon at the onset of a sharp attack of fever. We have perhaps here the true homologue of the rigor of adults.

Much has been both written and taught concerning the convulsive onset of the fevers of children. Without any desire to detract from the importance of this symptom, I would suggest that its occurrence is perhaps less frequent than one might suppose from the amount of attention and description it has received. Discussing the causes of convulsion in children, Hughlings-Jackson makes the following reference to that now under consideration: "It scarcely comes in my way to do more in this paper than urge the recognition of these rarer possibilities in the crowd of the more probable causes." Convulsion as an initial symptom may occur in almost any acute febrile disease, but is, on the whole, more common in the eruptive fevers. It does not materially affect the ultimate prognosis. We must not lose sight of the fact that children are liable to convulsion under many different circumstances: such are rickets, states of exhaustion, dentition, diarrhœa, and organic disease of the brain, besides the convulsive seizures termed essential. All these possible causes have to be reckoned with when we are called upon to pronounce an opinion on a case of convulsion. If the temperature is high during the convulsion and continues to rise as the seizure passes off, we may suspect the onset of some acute fever. An inquiry into the family or personal history will often throw light on the cause. A clue may be found on examining the chest, which should always be done as a matter of routine. Albuminuria immediately following a convulsion is of little diagnostic value at the onset of a fever. It may be due to the direct effects of the convulsion.

The question may arise whether a convulsion indicates the onset of acute cerebral disease,—especially tubercular meningitis. This is only likely to occur where convulsion is the first symptom for which we are consulted. Tubercular meningitis is a disease of gradual and insidious onset, and very rarely begins with convulsions. The *status epilepticus* must be carefully distinguished from tubercular meningitis. In this condition, which results from a quick succession of fits, the temperature sometimes attains a considerable height, 106° F., and the urine may be albuminous. Of far more serious import are convulsions occurring during the eruptive stage of scarlet fever, measles, and variola. For information on this point the reader is referred to the special articles on these diseases. In the later stages of fever, when the patient is much exhausted, there is again manifested a tendency to convulsions, which occasionally usher in the fatal event.

Vomiting is a very frequent early symptom. It may take place after a meal or without relation to the ingestion of food. When severe and repeated, it is apt to induce collapse. The association of repeated vomiting with headache and drowsiness will raise suspicions of meningeal trouble. This grouping of symptoms, however, is sometimes seen in simple continued fever, and has been known to precede the crisis of acute pneumonia. Here is a case in point. A girl, four years of age, after ailing for about ten days, was seized with severe occipital headache and repeated vomiting. In a few hours she became cold and collapsed. When I saw her for the first time on the following morning the expression was pinched and anxious, and she complained continually of the pain at the back of the head. The pupils were dilated, sluggish, and unequal, and there was a slight convergent strabismus. The temperature was 102° F. The abdomen was natural, the *tache cérébrale* being well marked; the bowels were confined. She was ordered effervescing mixture and a grain of calomel. She vomited eight times during the day. Towards evening the headache was worse, the temperature 104° F., and the child more drowsy. During the night, however, the temperature began to fall rapidly; on the following morning all bad symptoms had disappeared, and the patient made a rapid recovery.

The skin is usually dry and hot, sometimes harsh,—especially in tubercular disease. Sweating is decidedly rarer than in adults. The perspiration of acute rheumatism is often limited to the palms and soles, that of rickets to the head. Pyæmia may run its course almost without a sweat. Except at the crisis of a fever, the occurrence of profuse sweats is an unfavorable symptom. They are sometimes met with in lung-affections attended by marked cyanosis,—broncho-pneumonia, for instance,—recalling the analogous condition of skin in the suffocative bronchitis of old people. Sweats are apt to occur in chronic tubercular disease with suppuration and in the later stages of tubercular affections of the chest and abdomen. As a general rule, however, phthisical children do not sweat in excess. Occasionally there is an eruption of sudamina on the chest which may lead to branny desquamation. Miliaria rubra probably never develops except where poultices or hot fomentations have been employed. Bed-sores seldom occur.

The aspect of the skin is very variable. Some children are pale at the onset of fever, others look hot and flushed. The two conditions may alternate in the same patient. When the skin is unusually flushed, the condition is sometimes designated as the "febrile blush." In some cases it presents a close, though superficial, resemblance to the rash of scarlet fever, and has more than once led to a mistaken diagnosis, even at the hands of competent observers. Not a few cases are admitted each year into fever-hospitals as scarlatinal which ultimately prove to be cases of simple continued fever or develop into typhoid. The febrile blush is generally an early phenomenon. Its duration is very variable. Lasting in some cases but an hour or two, in others it persists, with varying intensity, for several days. Sometimes it is followed by branny desquamation. A high temperature is by no means necessary to its occurrence. The febrile blush is usually well developed on the face, neck, and upper part of the chest. It also affects the dependent parts of the body, the back, buttocks, and backs of the arms and legs. It is usually faint on the lower abdomen and inner aspect of the thighs. When fully developed it consists of a bright reddish-pink blush, uniformly diffused beneath the surface and fading momentarily on pressure. It is very evanescent and apt to shift from place to place. Exposure of the chest often causes it to disappear entirely in a few minutes, to return again as soon as the clothing is replaced. In some cases it occurs in large irregular patches with ill-defined borders. The distinction from the rash of scarlet fever is not likely to offer any difficulties, except in cases where the blush is unusually intense and persistent. The chief points of difference are the following. On the face the febrile blush is often well marked and reaches to the margin of the lips. The rash of scarlet fever is generally faint on the face and leaves untouched a zone of skin around the mouth. The blush of fever is usually faint or absent in the groin and on the inner aspect of the thighs, parts on which the scarlatinal eruption is generally well marked. The blush lies beneath the surface of a perfectly smooth skin. The rash of scarlet fever is punctiform and not necessarily uniformly diffused. The blush is less persistent than the rash, and more susceptible to external influences.

Labial herpes is seen in many kinds of fevers, but is not necessarily an early sign. It is relatively common in pneumonia, not rare in acute tonsillitis, febrile gastric disorders, and febricula, and may occur in the eruptive fevers. Its presence practically excludes the diagnosis of typhoid fever.

The lips dry quickly and become cracked. Children are very apt to pick and cause them to bleed and sometimes to become swollen. This picking at the lips, or at other parts of the body, is often a sign of nervous prostration.

The tongue does not present with any constancy, or in the same degree, the varieties of aspect which characterize it in certain febrile diseases of adults, and is therefore of little value in helping the diagnosis. An exception may be made in favor of scarlet fever. Slight furring on the dorsum with redness of the tip and edges is the rule, but the tongue may remain clean and dry throughout. A dry tongue may become brown, but it rarely cracks to any great extent, even in typhoid fever. The thick creamy fur of rheumatic fever is hardly ever seen. Some injection of the fauces is common at the onset of an acute fever. The appearance presented differs in degree only from a mild scarlatinal throat.

The digestive functions are almost invariably impaired. The salivary and pancreatic secretions are much diminished, giving rise to dryness of mouth, great thirst, distaste for food, and difficulty in assimilating starchy foods. The secretion of bile is probably also lessened : the stools are often pale and offensive. There is usually constipation, but this is a rule to which there are many exceptions. Diarrhœa is rare at the onset of fever, but may accompany the crisis.

The pulse of children in fever does not exhibit any marked peculiarities. Its clinical significance is relatively small, in great measure owing to the difficulty generally experienced in making a satisfactory examination, on account of the smallness of the arteries and the restlessness or intolerance of the little patient. In infants undue pulsation of the fontanel will suggest excited vascular action. The frequency of the pulse is always increased. It is often full and bounding in the earlier stages, but tends to become smaller and weaker as exhaustion increases, and in the later stages, when death is threatening, is often running or thready and impossible to count.

Increase in the frequency of the respiratory act is a constant and important concomitant of fever. The respirations may rise to forty per minute and the alæ nasi be set in action, even in the absence of any pulmonary complication. More than this, rhonchi and scattered râles may be heard over the lungs during the exacerbations of a simple catarrhal fever, which disappear entirely when the temperature remits. This accession of pulmonary signs is occasionally very marked during the hot stage of ague, and may render the diagnosis from pneumonia or broncho-pneumonia somewhat difficult. It is to be observed, however, that whilst catarrhal fevers may be accompanied by very definite pulmonary signs, the simple bronchitis of children is often attended by high temperatures, even though no pneumonia is present.

The disturbances of the sensorium vary greatly in different cases. The reason of this is to be sought partly in individual differences of temperament and resisting power, partly in the nature and degree of the intoxication. Many a case of simple fever runs its course without causing any appreciable impairment of health. In others, cerebral symptoms—vomiting, drowsiness, headache, etc.—predominate to such an extent as to justify the recognition of a cerebral type of simple fever. The profound and rapid prostration, with or without coma or convulsions, often seen in the malignant forms of the eruptive fevers and typhoid, illustrates strikingly the direct effects of the fever-poison on the nervous system. Restlessness, irritability, and drowsiness are common symptoms. Uneasy sleep and sleeplessness are also of frequent occurrence, and, although in themselves insignificant symptoms, demand close attention and proper treatment. A few hours of peaceful sleep will often do more to restore the strength of a fevered child than anything else. Children under five do not generally complain of headache. Altogether, this symptom is far less common than in adults. It is not rare, however, in the early stages of typhoid, and may occur in simple continued fever. The occasional occurrence of sickness during the course of a fever has no special significance. Severe and repeated vomiting, on the other hand, is a grave symptom, requiring prompt treatment, and liable to do much harm by increasing prostration.

Delirium is relatively uncommon in children, and the key-note to this peculiarity probably lies in the incomplete state of their mental evolution. Talking during sleep, however, is common enough, and a tendency to ramble in their talk, when awake, is not rare. If prostration is very great, a condition strictly analogous to coma-vigil may be present. Deepening stupor, subsultus tendinum, tendency to convulsion, and picking at the body or the bedclothes are all unfavorable symptoms.

Hyperpyrexia is rare, except as the immediate precursor of death, when it may be regarded as one of the earliest stages in the process of dying. This form is not amenable to treatment. Rheumatic hyperpyrexia is very rare indeed. Several cases of genuine hyperpyrexia have occurred during the treatment of acute renal disease by a variety of the continuous hot wet pack which is applied in the following manner. "A blanket is soaked in boiling water and then wrung out. The patient is placed in this just as hot as can comfortably be borne, which is usually at a temperature of 130° F., and completely enveloped in it as far as the neck. A mackintosh is then wrapped round this so as to exclude, as far as possible, atmospheric influences, and finally a thick blanket, doubled, is laid over the patient thus enveloped. The pack is changed hourly, and the child is out of the pack just so long as it takes to remove the disused one and replace it by another." These unfortunate accidents have rendered great service, not only in drawing attention to the dangers of this mode of treatment, but also because they are instances of hyperpyrexia artificially induced by placing the human body under abnormal conditions which are accurately known. These are, an envelope of moist heat far above that usually encountered by the body in temperate climates, and the simultaneous and almost complete elimination of the most important cooling agent, the skin. There may be profuse sweating, but this is of little avail, as, under the existing conditions, evaporation and radiation cannot take place to any extent. Under such conditions, assuming that regulation remains effective and thermogenesis can be held in sufficient check, there need be no hyperpyrexia, in spite of the almost complete closure of the most important channels of heat-loss. The temperature may rise at first, owing to the sudden check on thermolysis, but in time a balance between production and loss will again be struck. This is precisely what takes place in some cases. The temperature rises two or three degrees and gradually falls again. But once allow the central mechanisms inhibiting thermogenesis to become exhausted or temporarily paralyzed by over-stimulation, heat-production will have unbridled sway, and the result will be hyperpyrexia. These clinical experiments, as they may fairly be termed, are in reality a faithful reproduction of the conditions under which heat-stroke sometimes occurs in tropical countries, and the symptoms exhibited in the two conditions are almost identical. Of these the more important are extreme restlessness or delirium, with ashy pallor or lividity of the face and a dry tongue, associated with very rapid shallow sniffing breathing and extreme rapidity and feebleness of pulse.

The changes in the urine do not call for any lengthy notice. The total quantity is reduced and the specific gravity raised. On cooling, the urine generally deposits a sediment of white or yellowish-white lithates. This amorphous precipitate of mixed urates dissolves readily on heating. At times, especially when there has been temporary retention of urine, hedgehog or acicular crystals of urate of soda may be deposited within the bladder, and are voided with the first portion of the urine subsequently passed.

During convalescence it is common to find a cloud of white phosphates precipitate by heat, which dissolves at once on the addition of a drop of acetic acid. At this period the urine may also contain uric-acid and oxalate-of-lime crystals.

The occurrence of temporary albuminuria is by no means rare. The quantity of albumen present is always small, rarely exceeding a large trace, and disappears rapidly with convalescence. The occurrence of small hyaline casts has no special significance, but the presence of granular or cellular casts points to some structural disease of the kidneys.

The effect of fever on the body is generally well marked in children. Wasting is general, muscles and cellular tissues chiefly, but probably all tissues in varying degree. Emaciation often takes place with startling rapidity. A fever of twenty-four hours' duration may make a noticeable change, especially in plump, fat children.

Treatment.—In dealing with fever, two lines of treatment are open to us. The first is to remove or destroy the fever-poison. The other—our only resource in cases where the poison is out of reach—is to place our patient under the most favorable conditions and treat injurious symptoms and complications as they arise.

The child should be placed in an airy, well-ventilated room with an equable temperature, between 60° and 65° F. The clothing and bed-covering should be light, but adequate,—our object being to allow radiation and evaporation free play, without exposing the patient to the variations of the external temperature, which are apt to produce slight shivers. Linen clothing is to be preferred to flannel during the active stages of fever, except when there is much sweating. It is more pleasant to wear, is not so apt to irritate the skin, and can be more easily and effectually cleaned. During convalescence a flannel vest may be worn with advantage. Soiled body- and bed-linen should always be immediately changed and removed from the sick-room.

The diet should be bland and mostly liquid, and the food is best given in small quantities at short intervals. During fever the processes of digestion are always much impaired, and we should be careful to avoid overloading the stomach in our anxiety to sustain the strength of the patient. Milk is to be given as a food, and not as a drink. Cold water, in plenty, or barley-water flavored with lemon, may be taken to slake thirst. If pure milk is not easily digested, it may be diluted with plain barley-water or with solution of gelatin or gum acacia. Beef tea and mutton broth are generally well borne, and should always form part of the diet of children over eighteen months old. If they should disagree, chicken broth or veal tea will be available. If the stomach become intolerant, pancreatized milk or beef tea may be tried by the mouth, or, if this should fail, in the form of enemas or nutrient suppositories.

Constipation should be relieved, preferably by means of enemas (soap-and-water or glycerin).

Diuretic and diaphoretic salines may be given, with plenty of water. They tend to promote free action of the skin and kidneys, and facilitate the removal from the tissues of the waste products of fever.

Fevers due to some disorder of digestion generally yield at once to a purge or an emetic; and, as many of the simple fevers of children are of this nature, castor oil and calomel have acquired great repute in their treatment. The pyrexia which accompanies certain specific diseases will often yield to drugs which exert a specific action on the disease. As examples may be mentioned quinine and salicylates in the treatment of malaria and acute rheumatism. Fevers depending on purely local causes, in accessible situations, are also readily amenable to suitable treatment. Such are the putrid fevers of foul wounds, of acute abscess, ulcerative stomatitis, and others. Admittedly these classes of fevers represent but a small fraction of the whole number and do not include those which are most often dangerous to life.

When the cause of the fever is beyond our reach, we must treat symptoms. Of these, pyrexia is not only one of the most important, but also the one that we are best able to cope with. It is well to bear in mind, however, that children are in general very susceptible to the action of antipyretics, and that unless due care is exercised in the selection of suitable cases and appropriate methods this form of treatment will often disappoint expectation. The occurrence of collapse constitutes one of the chief risks in the employment of antipyretics. To guard against this danger, they should always be used tentatively at the outset and their effect carefully watched. Thus, after a bath or a dose of antifebrin the temperature should be taken at least twice within an hour, in the mouth or the rectum according to the age of the patient. Any symptoms of collapse should at once be met by the exhibition of stimulants and warm applications to the surface. When cold applications cause much distress to the patient, they are of doubtful benefit, and should be discontinued, unless the reduction of the temperature is imperative, as in hyperpyrexia. In certain states the external application of cold is contra-indicated. It rarely does good in the condition described above as the possible homologue of the rigor of adults, in which the extremities are cold and bluish and the trunk burning hot. In such a case cold is likely to aggravate the cyanosis and further depress the patient, whereas a warm bath and a little alcohol will often quickly improve the general condition. In some cases of cerebral disease where the employment of ice-bags or cold-water coils to the head has been a source of discomfort and irritation, much benefit has attended the substitution of hot fomentations.

Although it is obviously impossible to lay down hard-and-fast rules for their use, antipyretics may legitimately be employed (1) in cases of sustained high temperature above 103° F., (2) in all cases of hyperpyrexia, and (3) whenever the rise of temperature is accompanied by aggravation of other symptoms, such as restlessness, want of sleep, drowsiness, delirium, or rapidity and weakness of cardiac action. These indications are more than ever imperative when the patient's strength has already been taxed by prolonged fever and in children of weak physique. The means at our disposal are (1) drugs, and (2) the external application of cold.

The drugs on which most reliance can be placed are quinine, in full doses, salicylates, and the class of antipyretics now in vogue,—viz., antipyrin, antifebrin, and phenacetine. The last-named drugs must be given with due caution. In full doses they may depress the heart to an alarming degree; and this constitutes a serious objection to their use when there is much prostration.

Antipyrin has now been extensively used for some time, and its mode of action carefully investigated. It increases skin-radiation, lessens heat-production, diminishes nitrogenous waste by checking destructive metabolism, and frequently, but not always, increases perspiration, while it generally slows the heart and slightly increases the tension of the radial pulse. It may fairly claim, therefore, to be a true antipyretic, and not merely a refrigerant. It has met with marked success in the treatment of fever. Its effect on the temperature is, unfortunately, very transitory, so that it may be necessary to repeat the dose hourly to keep the pyrexia under control. At times it causes gastric irritation and troublesome vomiting, and occasionally diarrhœa. This tendency may be obviated to some extent by adding one or two drops of tincture of opium; and it is a good plan to give a little alcohol at the same time, to prevent depression. The administration of antipyrin is sometimes followed by the eruption of a measles-like papular erythema, which appears first on the arms and dependent parts, and may spread to the remainder of the body. This rash is of no serious import, and quickly disappears on discontinuing the drug. In rare instances the drug has caused symptoms of acute irritant poisoning.

Antifebrin (acetanilide) possesses several advantages over antipyrin, and is gradually superseding it in the treatment of fever. Its mode of action is probably the same. As an antipyretic it is more rapid and powerful in its action, and its effect on the temperature is rather more permanent. Serious collapse is liable to occur when it is given incautiously, and it may cause rigors. It is rarely followed by sickness, and does not produce a rash. It should be given tentatively at first, half to one grain for a child of two years and about two grains for a child of five. The dose required will vary according to the stage and nature of the fever. Full doses are generally needed in the early stages of fever, especially in scarlet fever, in which the drug has proved of considerable value. The pyrexia of typhoid fever, as a rule, yields readily to all antipyretics.

Phenacetine, so far as we know, acts in the same manner as antifebrin, and is said to possess the same advantages.

One other drug deserves mention. Aconite is of conspicuous value in catarrhal fevers the result of chill, and in acute tonsillitis. If given in the earliest stages, the general malaise, sense of chilliness or burning heat, pains, etc., rapidly disappear and give place to a feeling of comfort. The skin becomes moist and may sweat profusely, and the pulse is lowered in frequency. The drug should be given in small doses at frequent intervals, half to one drop of the tincture every quarter of an hour for a child of five years, until the desired effect is obtained. Much care is required in its administration, owing to its powerful depressant action on the heart.

The means of reducing excessive temperatures by the external application of cold include, among others, sponging, the compress or ice-bag, the wet pack, bathing, and affusion.

The mode of action of cold is not exactly known, but probably differs considerably from that of the drugs just considered. It is highly probable that its good effects are attributable to some stimulant action on the central nervous system, as well as to the abstraction of heat from the surface and the probable diminution of heat-production. Certain it is that this mode of treatment is far more effective in rousing the depressed sensorium and combating prostration than the antipyretic drugs. As a rule, it induces a refreshing sleep, whereas this desirable result only occasionally follows the administration of antipyrin and its allies. This mode of treatment is always to be preferred when there is much prostration. In a general way, children do not stand cold well, and tepid applications should always be given a trial before using cold.

Sponging.—The face, trunk, and limbs are sponged over for ten or fifteen minutes with tepid water (80° F.), or with cold water (50° F. or lower). The surface is then rapidly dried and the covering replaced. A sheet and a single blanket will generally suffice. If tepid water does not bring about the desired effect, a graduated bath is likely to answer better than sponging with cold water.

Compress.—This consists in the application to the body or limbs of cloths wrung out in cold or iced water. They require to be very frequently changed, and should be discontinued when the temperature has fallen below 100° F. Like sponging, compresses demand constant attention on the part of the nurse, and may interfere with sleep. They are chiefly useful in typhoid fever when there is much abdominal distention and it is not advisable to disturb the patient.

Bathing.—The bath is the most powerful antipyretic agent we possess, and almost the only one that has achieved success in the treatment of hyperpyrexia. Its chief value in the treatment of children, however, is due to its combined stimulating and sedative effect on the nervous system. To allay restlessness and general malaise, a warm bath (95° F.) will often accomplish all that is needed, and is usually followed by some lowering of temperature. The effect of a bath at 80° F. is more powerful and lasting, but less agreeable to the patient. When reduction of temperature is the primary object, a graduated bath is, on the whole, the most convenient plan. The child is placed in a bath at 90° F. which is rapidly lowered in temperature by the addition of cold or iced water. The bath should be of short duration and given in the presence of the medical attendant. The rectal temperature should be taken at intervals of a few minutes, and the child removed from the bath when it has reached 100° F., as the after-fall may be considerable. It is often advisable to give a small dose of alcohol both before and after the bath. The occurrence of shivering or of blueness of the lips and extremities is an indication for immediate removal from the bath, in ordinary cases. In the treatment of hyperpyrexia, however, every consideration, in reason, is subordinate to the paramount necessity of bringing down the temperature.

A combination of the warm bath with cold affusion to the neck and shoulders is occasionally of the greatest service in the treatment of bronchitis and collapse of lung. The following is a good example of the class of case in question. A child of three years, who had been suffering from a severe attack of bronchitis for ten days, was admitted into hospital in the following condition. Temperature, 103° F. Eyes suffused, face puffed and livid. Respirations over sixty per minute, short, shallow, and ineffectual. Marked inspiratory recession of lower ribs and interspaces and above clavicles. On auscultation, loud rhonchi and bronchitic râles all over lungs, with weak breathing at the bases. After taking a teaspoonful of brandy in hot water, the child was placed in a warm bath and water at 70° F. dashed over the neck and shoulders. Each affusion caused the patient to take a deep inspiration, and the relief to the congested pulmonary circulation was immediately apparent. The face soon regained a more normal color and expression, the breathing became deeper and slower and the inspiratory recession much diminished. A second bath was needed a few hours later, but after this recovery was uninterrupted.

Cold Wet Pack.—The patient is wrapped in a sheet wrung out of cold or iced water and covered over with a thick blanket. The pack should not be continued longer than from ten to fifteen minutes.

The water-bed or water-pillow may be utilized to reduce the temperature by allowing water at a suitable temperature to circulate through it; but it cannot be recommended as a convenient or satisfactory method of carrying out this object.

Ice-bags or iced-water-coils are applied to the head for the purpose of allaying meningeal inflammation and reducing temperature. Their utility in the latter respect, which alone concerns us here, is undoubted, but, as a rule, children do not tolerate them, and other and more efficient means have been mentioned.

Some of the indications for the use of stimulants have been incidentally mentioned. In them we possess a most powerful means of combating prostration and the exhaustion of prolonged fever. Besides the general condition and aspect of the patient, the state of the tongue and that of the pulse afford the safest indications for their employment. When they cannot be given by the mouth, alcohol or ether may be administered by subcutaneous injection, or in the form of enema with a sufficiency of water or beef tea.

SIMPLE CONTINUED FEVER.

Synonyme.—Febricula.

Definition.—A fever of short duration which is not characterized by the presence of any definite local lesion or preceded by any known invariable antecedent. It is, in truth, a morbid genus without essential attributes, and made up, in large part, of aberrant varieties of other species. An initial diagnosis of febricula has often to be set aside in favor of pneumonia, typhoid, tonsillitis, or some other acute febrile disease; and the converse happens with equal frequency. Such facts bear strong testimony to the indeterminate character of this affection.

Simple fevers are very common in childhood, and their early recognition is of great practical importance. They may be roughly grouped under the following heads:

1. Abortive or incomplete forms of the specific continued fevers, typhus, typhoid, and relapsing fever. Cases of irregular type may occur at any time, but are more frequent during the epidemic prevalence of these diseases.
2. Cases of scarlet fever, modified variola, and more rarely measles and erysipelas, in which the eruption is either absent or unnoticed.
3. In rare instances, anomalous forms of intermittent fever.
4. Fevers due to the effects of some localized inflammation, in which the local signs are transient, ill developed, or beyond the reach of observation. Cases of this kind occur in connection with lymphadenitis, tonsillitis, and acute catarrhal affections of the alimentary and respiratory mucous membranes.
5. The whole group of fevers caused by disorders of digestion, attended by the absorption of pyrogenic substances.
6. Fevers depending on some disturbance or exhaustion of the nervous system as the consequence of exposure to heat or of some peripheral nerve-irritation.

The only symptom common to the whole class is pyrexia, and in a considerable number of the cases it constitutes the whole disease. The access is generally sudden, but may be gradual. The temperature often attains a considerable height, 104° to 105° F. The initial rise may be ushered in by any one of the symptoms considered in an earlier part of the article. Vomiting is common; convulsion, on the whole, is rare. Some or all of the clinical symptoms of fever, before described, may be present in varying degree. The febrile blush is often particularly well marked. The pulse and respirations are always increased in frequency. Restlessness, wakefulness, and slight delirium are not infrequent. Constipation is the rule, with furred tongue and disinclination for food. Thirst is nearly always excessive. The urine is usually scanty and high-colored. In some cases gastric symptoms preponderate, the tongue being thickly coated, vomiting frequent, thirst unquenchable, and the bowels difficult to move. In others the respiratory organs bear the brunt of the attack; the breathing is quick and somewhat labored, the alæ nasi acting strongly, the face a little dusky, whilst numerous râles and rhonchi are audible all over the chest; meanwhile, the tongue may remain almost clean and digestion be but little impaired. A cerebral type has already been referred to, in which headache, repeated vomiting, intolerance of light, and irritability or tendency to delirium are prominent symptoms. The temperature generally falls by crisis at the end of two or three, it may be five or six, days, and convalescence is always rapid. The diagnosis rests chiefly on the exclusion of other acute fevers.

Typhoid fever, pneumonia, tonsillitis, scarlet fever, and meningitis are the diseases from which the diagnosis is most difficult in the early stages. The occurrence of a sharp attack of fever in a perfectly healthy child is in favor of febricula. The prognosis is always favorable. In the way of treatment, rest in bed for a day or two, with liquid diet, is all that is required in most cases. Cooling drinks and diaphoretic salines are agreeable to the patient, and harmless. In suitable cases a purge or an emetic is the best treatment. Antipyretic treatment is not called for, and may be injurious. It must, however, be borne in mind that what appears to be simple fever at the outset may prove to be some severe or highly infectious disease. And, further, it would seem that certain forms of febricula are infectious. It is a common experience to find all the children in a household sicken one after another with a fever of short duration accompanied sometimes by bronchial catarrh and at other times by marked gastric disturbance. A knowledge of these facts should make us doubly cautious in dealing with cases of this nature.

THERMIC FEVER.[1]

Synonymes.—Sunstroke, Heat-stroke, Insolation.

This condition is always caused by exposure to excessive heat of some kind. A moist heat is more likely to produce it than a hot dry air. Hence cases of this disease are more often met with in damp, low-lying districts, and near the sea-coast, than on high dry table-lands. Exposure to the direct rays of the sun is not necessary: many cases have been reported as occurring towards evening or during the night.

Among predisposing causes, bodily fatigue or exhaustion and race are the principal. In hot countries Europeans always suffer more than natives. Bodily fatigue is less likely to operate as a predisposing cause in children than in adults. It is highly probable that excessive heat is a factor in the rise of the death-rate among children from enteritis during the hot months. It has been pointed out that these cases, when the pyrexia is high and the cerebral symptoms marked, are often relieved by the application of cold, either in the form of affusion or by means of baths.

---

[1] See remarks on thermic fever under Yellow Fever.

The symptoms vary within wide limits. Mild cases of thermic fever may be indistinguishable from cases of simple continued fever. In severe sunstroke or heat-stroke the more constant symptoms are a varying degree of coma, with or without delirium; rapid sniffing breathing, or labored respiration, with stertor; ashy pallor of the face or livid suffusion; intense burning heat of skin, and great rapidity of pulse. Great restlessness, subsultus tendinum, and partial or general convulsions are of frequent occurrence. A peculiar odor of the skin and the breath has also been described. The urine is generally retained, but the contents of the bowel may be evacuated involuntarily.

The cause of the rise of temperature in heat-stroke has already been discussed in treating of hyperpyrexia, and need not be recapitulated here. In the case of sunstroke the paralysis of the thermal mechanisms would seem to be due to some direct effect of the heat-rays on the central nervous system.

After death putrefactive changes occur rapidly. The most obvious post-mortem signs are those of general venous engorgement. The coagulability of the blood is often impaired. Petechiæ or small extravasations have been found in various parts of the nervous system.

The diagnosis of heat-stroke is not usually attended with difficulty, in well-marked cases. In the absence of any definite history of causation, the distinction from meningitis may not be easy. I am acquainted with one such case.

Mild cases of thermic fever are most satisfactorily treated with cold or graduated baths. In severe cases with unequivocal hyperpyrexia, immediate treatment is of paramount importance. The temperature *must* be reduced without delay. Cold affusion, cold baths, and rubbing the surface with ice are the most powerful means at our command. The thermometer, in the mouth or the rectum, is the only safe guide as to the effect of treatment. Great care must be used to avoid and prevent collapse.

In cases attended with coma, if the lowering of the temperature is not accompanied by a return to consciousness, the prognosis is almost hopeless. The tendency to relapse is sometimes very marked. In all cases the temperature should be closely watched after the fall, and antipyretic measures adopted on the first indication of a fresh rise. In such an event, cold packing, quinine, or antifebrin might be tried.

ENTERIC OR TYPHOID FEVER.

BY J. C. WILSON, M.D.

Definition.—An acute infectious disease due to a specific cause. It is characterized by gastro-intestinal catarrh, febrile movement of continued type, varying in duration from ten to twenty days, marked prostration, rapid wasting, mild nervous symptoms, and a scanty eruption of isolated, slightly elevated, rose-colored spots disappearing on pressure and developed in successive crops. After death, constant lesions of the solitary and agminate glands of the ileum, with enlargement of the mesenteric glands and of the spleen, are found. Enteric fever in infancy and childhood does not conform closely to the type of the affection in adult life.

Synonymes.—Infantile remittent fever; Nervous fever; Slow nervous fever; Infantile hectic fever; Gastric fever; Acute mesenteric fever; Entero-mesenteric fever; Intestinal fever; Pythogenic fever; Dothiénentérie; Typhus abdominalis; Ileo-typhus.

The names by which this fever has been described at various periods and by different authors are derived from its supposed relationship to typhus, its mode of prevalence, its remittent character, its long duration, its supposed nervous origin, the occurrence of septic or putrid symptoms, its hectic phenomena, the presence of symptoms denoting disturbances of the stomach and liver, the intestinal symptoms, the morbid anatomy, and its mode of origin.

I have not considered it necessary, however, to enumerate all these names, seeing that by far the greater part of them have for obvious reasons fallen out of use. The term "abdominal typhus" and its equivalents in general use in Germany and elsewhere upon the Continent are open to the objection that they suggest a relationship with typhus which is now known not to exist. They are, in fact, due to the opinion formerly generally entertained that there existed between the two affections an essential pathological relationship,—that they were, in fact, two varieties of a single species of fever. This opinion is no longer tenable. "Typhoid," suggested by Louis in 1829, is open to the same objection, since the labors of pathology during the past half-century have shown with increasing clearness not that the fever in question is like typhus, but that it is unlike it. This term has, however, the sanction of very general acceptance in France and among [illegible] physicians. The strongest objection to its use lies in its [illegible] an adjective to designate a condition or group of [illegible] appear in the course of any severe acute disease,—a use [illegible] confusion of thought and vagueness of de[illegible] enteric fever" proposed by the late Prof. George [illegible] the advantage of designating at the same time the con[illegible] by a now accepted usage of the word fever in [illegible] the infectious nature of the disease. It was [illegible] Nomenclature of Diseases" in 1869. The term "infantile [illegible] used, has an historical importance as embody[illegible] pathology of this disease among children an error at [illegible]

History.—[illegible] this article does not include any extended [illegible] the growth of knowledge concerning the disease under [illegible] interested are referred to my work upon "The Con[illegible] the elaborate classical treatise of Dr. Murchison.[2] It [illegible] fever has come down to us from a remote antiquity. [illegible] separated from the general group of the fevers as a [illegible] the present century. The fact that the fevers [illegible] until about the end of the first quarter of [illegible] than passing interest to the history of the [illegible] Louis, Chomel, and others, in France; of Aber[illegible] Bright, and Jenner, in England; of Hildenbrand, in [illegible] of Gerhard, Pennock, Shattuck, and Bartlett, in [illegible] by the work of these physicians that during [illegible] present century enteric fever, the great fever of the [illegible] was distinctly separated from all other forms of [illegible] of its pathology placed upon a sure basis of [illegible] of these observers, however, were directed almost [illegible] as it shows itself in adult life, the opinion prior [illegible] held that infancy and childhood enjoyed an im[illegible] Rilliet[3] and Taupin,[4] who published at about the same [illegible] descriptions of typhoid fever as it appears in childhood, [illegible] shown that this view was erroneous, and that [illegible] cases of fever among children previously described [illegible] were in fact instances of enteric fever.

[illegible] fever is due to the entrance into a susceptible [illegible] infecting principle.

[illegible]—These are, on the one hand, all conditions [illegible] and accumulation of the infecting principle,

---

[1] [illegible] C. Wilson, M.D., 1881.

[2] [illegible] Britain, by Charles Murchison, M.D., LL.D., F.R.S., [illegible]

[3] [illegible] Enfants. Thèse, 1840.

[4] [illegible] Nov.–Déc. 1839, Janv. 1840.

and, on the other hand, those conditions which increase the susceptibility of the individual to the cause of this particular fever and the liability of his exposure to it. The etiological considerations relating to enteric fever are equally applicable to childhood and to adult life.

The geographical distribution of enteric fever is wide. This disease has been observed in all countries and in every climate. It is endemic in the British Isles, in almost all parts of Continental Europe, and in North America. Hirsch[1] has reached the conclusion that its general prevalence in Europe and America dates no further back than the second and third decades of the present century,—that is, from the period at which typhus (*der Petechialtyphus*) became everywhere less common and in many regions disappeared altogether. In America it prevails as the common fever from Hudson's Bay to the Gulf of Mexico. In new and sparsely-settled districts, where the land is being gradually brought under cultivation, the malarial fevers occur; after a time, as populations increase, the malarial diseases and enteric fever prevail side by side; finally, when the land has been generally taken up, drained and tilled for some generations, and villages and cities abound, the malarial diseases, true agues and remittents, impress communities but faintly or disappear altogether, while enteric fever becomes common and asserts itself as the predominant endemic fever in proportion to the neglect of the sanitary measures by which alone it can be kept in check in populous localities.

Climate, not of itself, but indirectly, as determining the mode of life in communities, has a manifest influence upon the extent of the prevalence of enteric fever.

Enteric fever is by no means confined to temperate climates; it is far from uncommon in tropical and subtropical countries.

The season of the year is a predisposing cause of great importance.

Hirsch found that 519 epidemics of typhoid fever were distributed among the seasons as follows: in the spring, 29; in the summer, 132; in the autumn, 168; and in the winter, 140. Of 116 circumscribed epidemics occurring in France between 1841 and 1846 recorded by De Claubrey, 20 began in the first quarter of the year, 21 in the second, 39 in the third, and 36 in the fourth. The number of cases in localities where the disease is endemic is usually greatest from August to November, decreasing in December, and is lowest from February to May, again increasing in June. This fever is so much more common in the latter part of the year that it has received in some districts of the United States the popular name of "autumnal" or "fall fever."

The state of the weather as regards dryness and moisture exerts a remarkable influence upon the prevalence of enteric fever. Hot and dry summers favor the development of the disease, cold and wet summers check it. This statement is supported by the concurrent testimony of observers in all countries. Dryness of the atmosphere alone does not, however, lead to an increase of enteric fever. In cities and other localities possessed of a system of undergound drainage, warm damp weather often leads to an outbreak of the disease, while heavy rainfalls by flushing the drains remove the causes to which its origin and spread are chiefly due. On the other hand, outbreaks of enteric fever may be traced to the influence of abundant rains in washing the germs of the disease into the water used for drinking-purposes, particularly where the water-supply is derived in part from tilled and therefore manured fields.

---

[1] Handbuch der historisch-geographischen Pathologie, Erlangen, 1860.

Pettenkofer and his pupils have sought to establish a direct relation between the prevalence of enteric fever and the height of the deeper springs of water. When the water rises, the number of cases of enteric fever decreases; when the water sinks, the number of fever-cases increases. This relation holds true for Munich, Berlin, and some other places. It has not, however, been satisfactorily explained. The observation corresponds with the statement above made that enteric fever is much more frequent after hot and dry summers than after cold and wet ones. These observers seek to explain the varying prevalence of enteric fever in connection with the changes in the ground-water by the assumption that the ground-soil is the chief place of development for the schizomycetic fungus. When the water-level sinks, the layers of earth, containing moist organic substances and exposed to the air, undergo changes which lead to the development of the fever-poison; when, on the contrary, the water rises, these layers of earth are again covered and the development of the germs is arrested.

The views of Pettenkofer lack confirmation and have not been generally accepted.

Age is of great importance among the predisposing causes of enteric fever. This affection is pre-eminently a disease of adolescence and early adult life. The period of greatest susceptibility lies between the ages of fifteen and thirty, and the liability diminishes progressively both above and below these limits. Cases in the first year of life are exceedingly rare, but from this period through infancy and childhood the liability is fully established. In 1864, Murchison showed at the London Pathological Society the intestines of an infant six months old who had been attacked at the same time with her mother. The explanation of the fact that the proportion of cases occurring in infancy is smaller than that of childhood and adolescence is to be sought in the increased exposure to the infecting principle at the later periods.

On the other hand, enteric fever is not common in advanced life, though well-authenticated cases in persons seventy, eighty, and even ninety years of age have been reported. The infrequency of the attack in the later periods of life is doubtless to be accounted for in part by the fact that many persons, having already passed through the disease, enjoy an acquired immunity.

Sex in childhood exerts no influence whatever as a predisposing cause.

Statistics that have been from time to time adduced to show that the disease is much more frequent in boys than in girls embody the fallacy arising from a failure to appreciate the fact that beyond the age of infancy girls are much less exposed, under ordinary circumstances, to the infection than boys. The latter in their out-door sports, bathing, swimming, and the like, are not only in frequent danger of inhaling the concentrated emanations from sewers and drains, but also subject to the liability of drinking water directly defiled by sewage.

The mode of life is also without influence. Enteric fever is as common in the houses of the affluent as in the most crowded and destitute localities.

It was at one time thought that some sort of relationship existed between enteric fever and variola, and that the former was more prevalent in communities protected by general vaccination than those less fortunate in this respect. This opinion is now known to be devoid of foundation. The suggestion of Harley that scarlatina and enteric fever are different manifestations of the same poison, but that enteric fever is an abdominal scarlatina, is likewise untenable. The two diseases are essentially different in their causes, course, symptoms, duration, and lesions.

Habitual exposure to the poison of enteric fever in small amounts appears to confer an immunity from the disease. Instances are recorded where successive visitors at the same house at intervals of months or even years have been seized shortly after their arrival with enteric fever or intestinal catarrh from which the ordinary inhabitants were exempt. Persons changing their residence from one part of a city to another have not infrequently been taken with enteric fever, and persons coming from the country into cities very frequently become the subjects of the disease. The French observers strongly insist upon recent residence as a predisposing cause.

2. *The Exciting Cause.*—It may now be regarded as settled that the cause of typhoid fever is a specific, organized, pathogenic germ.

Numerous observers—Eberth, Klebs, Koch—found bacilli in Peyer's patches, the mesenteric glands, and the spleen, from cases of this disease. Eberth gave the name "bacillus typhosus" to an organism constantly found in the affected organs; but a species described by Koch and extensively studied by Gaffky[1] appears to be the only one constantly present in this disease which is not known to occur under other circumstances.

The last observer found this organism in the mesenteric glands, liver, spleen, and kidneys of twenty-six of a series of twenty-eight cases of enteric fever which he investigated. The subjects in which these bacteria were found had died in the earlier stages of the disease. Gaffky was unable to determine the presence of the typhoid bacilli in the blood or in the intestinal contents.

Pfeifer,[2] however, claims to have discovered them both in the intestinal

---

[1] Mitth. a. d. Kaiserl. Gesund-Amt, Bd. ii., 1884.

[2] Deutsche Medicin. Wochenschr., 1885, No. 29.

The doctrine, so ably defended by Murchison and his followers, that the specific cause of this disease may be generated *de novo* in sewage without the presence of enteric excreta, is no longer tenable. There is no proof whatever that enteric fever can, in the absence of the specific pathogenic germ above described, be produced by the products of decay or decomposition, by tainted food, or by the action of other bacteria; nor is there any reason to believe that typhoid bacilli can be developed from other micro-organisms.

When introduced into the human body, this germ is capable, under favorable circumstances, of indefinitely reproducing itself. It is eliminated with the fæcal discharges. It retains its activity when it has found its way into favorable situations for an indefinite period after it has passed out of the body, the requirements to this end being decomposing animal matter, especially fæcal discharges and moisture; therefore cesspools, sewers, drains, dung-heaps, and wet manured soils favor its prolonged existence. It is capable of indefinite multiplication in these favorable situations. It remains suspended in and may be conveyed by water and milk. These fluids become the means of conveyance for the enteric fever germ to the interior of the organism. It is probable that it may also, under certain conditions, float in the atmosphere and thus occasionally find its way into the body by means of the inspired air.

This germ retains its power of growth and reproduction within wide ranges of temperature. Prudden[1] found it capable of growth after having been frozen in ice for one hundred and three days and after having been heated to a temperature of 132.8° F. He also found that it retained its vitality after repeated alternate freezing and thawing. The investigations of Seitz, Wolfhügel, and others show that it grows abundantly in milk.

The fact that the infecting principle of enteric fever retains its vitality and is capable of multiplication in water of various temperatures has been fully established by the great number of carefully-studied outbreaks in the past. Among others, the well-known epidemic of North Boston in 1843, described by Dr. Flint,[2] the epidemic at Lausen in the canton of Basel, Switzerland, in 1872, and the extensive outbreak at Plymouth, Pennsylvania, in 1885, have attracted especial attention. .

It remained, however, for the science of bacteriology to demonstrate the presence of typhoid bacilli in drinking-water as the actual visible cause of some recent outbreaks. Arloing and Morat[3] found this germ in four out of six specimens of drinking-water taken from various sources of supply at a school in Cluny during a local epidemic of enteric fever, comprising one hundred and nineteen cases, with twelve deaths, in a general household of two hundred and thirty-five persons.

---

[1] Medical Record, ix., 1887.

[2] Treatise on the Principles and Practice of Medicine, Phila., 1867.

[3] Annales d'Hygiène publique, Nov. 1888.

A similar result followed the investigations of Rodet, described by Bondet,[1] in regard to the water-supply of Ville-sous-Charmoux, the scene of a limited outbreak of the disease in the spring of 1887.

Finally, Vaughan and Novy[2] have demonstrated the typhoid bacillus, by means of potato-cultures and by physiological experiment, in the drinking-water at Iron Mountain, Michigan, at the time of the prevalence of a severe epidemic.

Our knowledge of the part played by milk as a means of transmission and culture-medium of the typhoid bacillus rests upon similar facts.

Ballard[3] in 1871 called attention to the danger of infection by milk, in a valuable report concerning a local epidemic which he investigated, with reference to its cause, as health officer of Islington. This outbreak was apparently due to the use of water defiled by direct communication with drains and probably by the backing up of the contents of the drains into the water-tank, for the purpose of washing the milk-cans.

Since that date a large number of local epidemics have been traced to infection by milk. In such cases the probable cause is not a disease in the cows, but an admixture of defiled water with the milk, either intentionally, or as a result of the use of such water for the purpose of cleansing milk-cans.

Seitz[4] and Wolfhügel and Riedel[5] found that the typhoid bacillus grows abundantly in milk.

Vaughan and Novy obtained a poisonous extract, hereafter to be mentioned, both from milk and from a meat preparation which had been inoculated with water containing typhoid bacillus.

Philipowicz[6] was the first to demonstrate by culture the existence of the typhoid bacillus in the blood of patients. He obtained blood from the spleen by capillary puncture under antiseptic precautions, and succeeded in cultivating the bacillus from it. His observations were confirmed by Lucatello, Chantemesse and Widal, and other observers.

Neuhauss[7] found the bacilli in the blood drawn from the points of eruption in nine cases out of fifteen, and succeeded in reproducing them by cultures.

Efforts to obtain cultures from the blood of epistaxis and uterine hemorrhages during the course of the fever have thus far failed.

Wyssokowitsch[8] injected pure cultures of the typhoid bacillus into the veins of a rabbit. The animal in eighteen hours was killed, and investigations into the distribution of the bacilli were made by plate-cultures upon gelatin. The plates inoculated with the blood of the heart remained sterile; those inoculated with the blood of the liver produced twelve colonies; those with the marrow of the bone, two hundred colonies; finally, the inoculations made from the spleen produced two hundred and forty-two colonies.

---

[1] Lyon Méd., 25 Déc., 1887.

[2] Experimental Studies on the Causation of Typhoid Fever, with Special Reference to the Outbreak at Iron Mountain, Mich., Medical News, January 28, 1888.

[3] On a Localized Outbreak of Typhoid Fever in Islington, London, 1871.

[4] Archiv für Hygiene, Bd. vii.

[5] Arbeiten aus dem Kaiserl. Gesundheitsamte zu Berlin, 1886.

[6] Ueber diagnostiche Verwerthung der Milzpunction bei Typhus abdominalis, Wien. Med. Blatt, 1886.

[7] Nachweis der Typhusbacillen am Lebenden, Berl. Klin. Wochenschr., 1886.

[8] Ueber die Schicksale der im Blut injicirten Mikroorganismen im Körper der Warmblätter, Zeitschr. f. Hyg., 1886.

These experiments indicate that the typhoid bacillus does not remain in the blood, but tends to collect in particular organs, especially the liver, the marrow, and the spleen.

The researches of Chantemesse and Widal and Neuhauss seem to establish the fact that the microbe in question is able to pass by way of the placenta from the blood of the mother to that of the fœtus.

The period of incubation varies within wide limits. Its precise determination in any given case is by no means so simple a matter as would at first sight appear. In an outbreak which occurred at Guildford, England, in 1867, the contaminated water which was the cause of the infection was supplied on a single day, the 17th of August. A large number of cases came under observation on the 3d and 4th of September,—a period of incubation apparently covering seventeen or eighteen days. The possibility that the water may have been partaken of a day or two later than the 17th of August is obvious, and the fact is not to be overlooked that there are few cases which come under observation upon the first appearance of the disease; so that in this case, which is one of the most definite upon record, the apparent is longer than the actual period of incubation, and longer by a number of days not possible to determine.

On the other hand, there are facts tending to show that this period may be as short as two or four or eight days. A case has recently occurred under my observation in the wards of the Philadelphia Hospital in which the first rise in temperature took place on the fourth day after the return of the patient from twenty-four hours' leave of absence from the wards. This patient had been long resident in the hospital, in which there was not at that time, nor had been for many weeks previously, a single case of enteric fever.

That enteric fever is contagious in the ordinary sense may well be doubted, although from time to time cases arise which appear to be explicable upon no other hypothesis than that of direct communication.

The occurrence of house-epidemics is to be explained by infection at the same time or in quick succession of a number of individuals from the same source; of cases developing in patients occupying beds adjacent to that of an enteric fever case in the wards of a hospital, by the conveyance of infecting material contained in the fæcal discharges from one patient to another through the neglect or carelessness of the attendants.

In the Children's Hospital at Basel[1] there were treated during fifteen years two hundred and ninety-three cases of enteric fever without special separation from the other inmates of the hospital; during this period there occurred eight cases—namely, 2.46 per cent.—of house-infection, viz.: in the year 1872, three cases; in the year 1873, one case; in the year 1875, one case; in the year 1878, one case; and in the year 1883, two cases. In one instance a child suffering from hip-joint disease that had been in the wards two months, no case of enteric fever having been during that time in the hospital, developed the disease: concerning the source of the infection in this case, no theory could be advanced. In four other instances there was at the time of the house-infection a case of enteric fever in the same ward. In two instances there were at the time of the development of the disease cases of enteric fever in the hospital, though not in the same ward. Enteric fever developed in two instances of patients suffering from spondylitis; twice in patients suffering from chronic inflammation of the knee-joint; once in hip-joint disease; once in a case of nephritis; and once in a case of multiple osteitis. It is worthy of note that patients suffering from diseases of the bones occupied the first rank among the victims of the typhoid infection. Of the eight cases developed in the hospital, a single one—namely, the patient suffering from nephritis—terminated fatally. The enteric fever cases manifested during their convalescence a special liability to other infectious disease, among which diphtheria occupied the first rank. Of the two hundred and ninety-three cases of enteric fever, twenty-six, or 8.8 per cent., died; among these twenty-six fatal cases the cause of death in seven was diphtheria or scarlet fever developed at the end of the attack or during the convalescence. The elimination of these seven cases reduces the enteric fever mortality to 6.4 per cent.

---

[1] E. Hagenbach-Burckhardt, Jahrbuch für Kinderheilkunde, N. F., xxiv., 1886.

Pathology.—The bacilli or spores, being swallowed, gain entrance to the organism by means of the intestine. This may be assumed to be the case even in those instances where the germs reach the organism by means of the inspired air, as it is probable that they are engaged in the mucus of the mouth and then swallowed. If not destroyed in the stomach, the bacilli retain their vitality, pass on into the alkaline contents of the intestine, and here find conditions favorable to their further development.

In cases examined post mortem in the earliest stages of the disease, the lesions are mainly confined to the lymphatic tissues of the intestine. The bacilli penetrate into the solitary follicles and Peyer's patches and there multiply and form colonies. From these colonies they migrate by way of the lymphatic vessels to the mesenteric ganglia, and by way of the radicles of the superior mesenteric vein to the liver, to be finally distributed by the blood-current to the spleen and other organs.

A knowledge of the causative influence of the typhoid bacilli in the production of enteric fever and of the mode of distribution of these germs in the various organs fails to account adequately for the symptoms of the disease. When, however, it came to be known as a result of the discoveries of Hoffa,[1] Brieger,[2] Vaughan,[3] and others within the last few years, that in the infectious diseases the special pathogenic micro-organisms produce the definite chemical poisons called ptomaines, the relation between these germs and the symptomatology of the diseases which they cause became obvious.

In 1885, Brieger isolated from pure cultures of the typhoid bacillus a toxic ptomaine. This substance injected into guinea-pigs produced a slight flow of saliva, accelerated respiration, dilatation of the pupils, profuse diarrhœa, paralysis, and death in the course of from twenty-four to forty-eight hours. Upon post-mortem examination, the heart was found to be in systole, the lungs deeply congested, and the intestines contracted and pale.

Brieger considers this substance, to which he has given the name "typho-toxine," to be the special poison of enteric fever.

In 1887, Vaughan and Novy obtained from pure cultures of the typhoid bacillus, derived from the drinking-water which had been the supply of a large number of persons who had the disease, a syrupy extract, which injected under the skin of cats caused an elevation of temperature from 2° to 4.5° F. above the normal. Similar results were obtained by Sirotinin,[4] Beumer, and Peiper[5] by inoculating animals with sterilized cultures of the germ. These observers found that the severity of the symptoms varied in proportion to the amount of the culture injected.

In view of these facts, Vaughan and Novy have suggested a new definition for the infectious disease: "An infectious disease arises when a specific pathogenic micro-organism, having gained admittance to the body, and having found the conditions favorable, grows and multiplies, and in so doing elaborates a chemical poison which induces its characteristic effects."

It is probable that certain of the intestinal symptoms of enteric fever are due to the direct action of the typhoid bacillus; but the constitutional symptoms, including the fever, must be explained by the continuous action of a chemical poison produced by the growth and multiplication of these organisms within the body; especially is this true of the nervous and vasomotor phenomena, the feeble circulation, dicrotism, relaxed capillaries, flushed face, dilated pupils, and delirium.

Pathological Anatomy.—Enteric fever differs from the other continued fevers, with the exception of cerebro-spinal fever, in the invariable presence of special anatomical lesions. These lesions are so characteristic that an examination of the body after death will in all cases make known the nature of the disease, even when the symptoms have been or were unknown.

It is important, however, to bear in mind that the lesions of the intestine and of the mesenteric glands do not constitute the disease, but that the chemical poison produced by its specific cause is taken up by the fluids of the body and gives rise to general disturbances, which are present in all fully-developed cases, and which manifest themselves at a very early period in the attack. The more important symptoms of enteric fever are directly attributable to the general process, and not to the special lesions.

---

[1] Die Natur des Milzbrandsgiftes, Wiesbaden, 1886.

[2] L. Brieger, Ueber Ptomaine, Berlin, 1885–1886.

[3] Ptomaines and Leucomaines, 1888.

[4] Zeitschr. für Hygiene, i.

[5] Idem.

The anatomical lesions, therefore, fall naturally into two groups.

The first embraces those arising from the local action of the typhoid bacilli and the concentrated ptomaine which they produce, and includes changes in the lymphatic system of the intestinal canal.

The second group includes lesions which are not the direct result of the local action of the bacilli, but are due to constitutional infection. They consist of degenerative changes involving the tissues of the various organs, and are to be found generally manifested throughout the body, and particularly in the liver, the kidneys, the voluntary muscles, the heart, the salivary glands, and the pancreas.

The marked disturbances in the function of the nervous system indicate profound nutritive derangement, the nature of which is at present unknown.

The changes of the second group are not peculiar to enteric fever; they occur in other acute febrile diseases, and must be ascribed to the action of the various special toxic principles to which the phenomena of such diseases are due. These anatomical changes attain their fullest development in enteric fever, however, for the reason that in this disease the organism is continuously subjected to the action of these toxic principles for a prolonged period.

As the following description is intended to present to the reader not only the conditions which obtain in ordinary cases, but also those which are occasional and exceptional, it is based upon the well-recognized lesions of adult life taken as a type. This course is necessary for the reason that while the anatomical changes in childhood are, as a rule, less fully developed, this is a rule to which there are not infrequent important exceptions.

The same course will be followed in the description of the clinical phenomena of the disease.

Cadaveric rigidity is usually marked and of long duration. Emaciation is often extreme. The integuments and the dependent parts of the body are apt to be more or less discolored, but the deep livid discoloration of typhus is rare. Except where death has taken place in consequence of pulmonary complications, the face is seldom livid. The characteristic rash of enteric fever is not often observed on the body, even in those cases where the spots have been numerous immediately before death. Sudamina and other accidental eruptions persist.

The lesions in childhood are, as a rule, less extensive and conspicuous than in adults, just as the disease itself is less intense. They have been also less thoroughly studied, by reason of the favorable course of the disease in childhood and the consequent low rate of mortality. Exceptionally, however, the local changes attain their full development in childhood.

The Digestive Tract.—The pharyngeal mucous membrane is usually normal; occasionally, however, it exhibits signs of recent inflammation, and sometimes distinct points of ulceration. The pharynx may also be the seat of diphtheritic exudation. Occasionally the œsophagus shows evidences of ulcerative processes similar to those met with in the pharynx. These ulcers are massed at the cardiac extremity of the œsophagus, and vary from simple excoriations to deep lesions implicating the muscular coat. The foregoing changes are not found when death occurs earlier than the third week of the disease, and are extremely rare in childhood.

Inflammation of the mucous membrane of the stomach is common in typhoid as in other acute febrile diseases.

The duodenum usually presents no anatomical changes. Sometimes it exhibits the evidence of increased vascularity, with slight enlargement of the mucous follicles. Ulceration does not occur.

The jejunum and the upper part of the ileum may be distended with gas; the lower portion of the ileum is usually collapsed.

The tympany which belongs to the disease is chiefly due to the presence of gas in the colon. Invagination of the intestine, unaccompanied by the evidences of inflammation, is occasionally met with at one or more points.

The constant and therefore characteristic lesion is an affection of the solitary and agminate glands in the lower part of the ileum. The lymphatic follicles of the cæcum are usually involved, and not infrequently those of the colon also.

First Stage.—The earliest change observed is a swelling of the glands, with surrounding hyperæmia. The Peyer's patches project above the surface of the surrounding mucous membrane in the form of flattened, oval plaques with a reticulated or irregularly mammillated surface and elevated margins. The solitary follicles, which are not constantly implicated, form when affected discrete, shot-like projections varying in diameter from one-eighth to one-fourth of an inch. These changes are always progressively more advanced in the lower part of the ileum, reaching their full development in the neighborhood of the ileo-cæcal valve. This glandular swelling is due to extensive hyperplasia of the lymphatic elements. The cellular infiltration extends downward into the submucous tissue, but at the borders of the patches is more or less abruptly limited. It attains its maximum about the end of the first week.

It is probable, for reasons to be hereafter mentioned in connection with the discussion of the subject of the temperature-range, that the glandular lesion in a considerable proportion of the cases in childhood at this time undergoes resolution without ulceration.

Second Stage.—The infiltrated lymphatic tissue now undergoes necrosis. The mass changes to a dirty-yellow color and becomes more opaque, and the lymphatic follicles, with the epithelium covering them and some of the surrounding tissue, break down at scattered points, so as to form an irregular or ragged ulcerated surface, or *en masse* into one large slough. This slough, stained a deep yellow or brown by the intestinal contents, remains for a time attached. It is separated by an abrupt line of demarcation from the deeply-congested surrounding tissue. It is then gradually cast off, either in a single piece or in fragments. This process occupies another week, so that the separation of the slough takes place at the end of the second or early in the third week. In the greater number of instances of enteric fever in childhood the sloughs are superficial and of limited extent, and the ensuing ulceration undergoes prompt cicatrization.

Third Stage.—The ulcer thus formed, as a rule in adults, but exceptionally in childhood, has a smooth floor, and abrupt and to a certain extent overhanging edges. The bevelled margins seen in tubercular ulcers of the intestine do not occur.

The ulcer when deep involves the greater part, usually the whole, of the Peyer's patch. Its base is usually formed by the muscular coat, but occasionally this is also destroyed, so that the peritoneum alone forms its floor. The process in the solitary glands is the same. The ulcerative process may involve a small artery and give rise, upon the separation of the slough, to a more or less copious or even fatal hemorrhage. Or perforation of the wall of the intestine may occur from an implication of the serous coat, permitting, unless immediate adhesions by plastic lymph to neighboring parts occur, extravasation of the intestinal contents, with the production of purulent peritonitis.

The process above described, when confined to the agminate glands, produces oval lesions, the longer axis of which corresponds to the direction of the bowel, and the position of which is opposite to the mesenteric attachment; when the solitary follicles are involved, the lesions are small, circular, and irregularly scattered.

The hyperplasia of the lymph-elements is due to the growth and multiplication of the typhoid bacilli; their necrosis and the subsequent ulceration, in part to the bacilli themselves and in part to the irritating effects of the concentrated ptomaine produced at the place of its formation.[1]

Cases have occasionally been reported in which irregular ulceration has extended laterally from the Peyer's patch, transversely involving a considerable extent of the bowel. This form of ulceration is, as a rule, superficial; exceptionally, however, it is deep and may ultimately give rise to a perforation of the intestine. It is without doubt due to an accidental local infection of an entirely different nature, and usually much prolongs the intestinal symptoms and constitutional disturbances of the period of convalescence.

Cicatrization in adults does not begin until after the third week, and probably occupies a period of two or three weeks before it is completed. The ulcers heal by granulation with restoration of the epithelium; the lymphatic structures, however, are permanently destroyed. The resulting scar is thin, transparent, and flexible, and does not lead to puckering of the tissue of the bowel nor to constriction.

---

[1] Vaughan and Novy, op. cit., pp. 93–107.

The mesenteric vessels connected with the affected portions of intestine are distended and hyperæmic.

The mesenteric glands undergo changes histologically the same as those which take place in the lymphatic structures of the intestine. In the early stages of the disease they are highly vascular and enlarged, as a result of lymphatic hyperplasia; later they become pale and undergo necrotic changes. The ordinary termination of the process, however, is in complete resolution.

If, however, the softening be considerable, resorption does not always take place, but the softened material undergoes cheesy metamorphosis and ultimately becomes calcareous. Sometimes the softening results in the formation of pseudo-abscesses, which may burst into the peritoneal cavity and give rise to general peritonitis. The other lymphatic glands, particularly those in the fissure of the liver, and the retro-peritoneal and bronchial glands, are occasionally found enlarged.

Fagge[1] expresses a strong feeling of doubt as to the correctness of the generally-accepted statement that caseation or the deposition of calcareous salts forms part of the ordinary retrograde process.

The lymphatic follicles at the root of the tongue and in the tonsils undergo changes analogous to those described, giving rise to enlargements, which appear early in the course of the disease and usually disappear without further change, although in some cases softening, rupture, and subsequent ulceration result.

The changes in the spleen are analogous to those which take place in the lymphatic follicles of the intestine and in the mesenteric glands. The organ is enlarged, tense, and hyperæmic. In the early periods of the disease it is of moderate consistence; later its tissue is soft, pulpy, or often diffluent. On section, its substance is of a brownish-red color. Hemorrhagic infarcts are often met with. The enlarged and softened spleen in the later stages of enteric fever is liable to be ruptured by mechanical force in palpation, and in some instances it has undergone spontaneous rupture. Enlargement of the spleen is rarely absent in the young.

The second group of anatomical changes comprises parenchymatous degenerations of the various organs of the body. These changes are to be ascribed in part to the action of the toxic infecting principle, and in part to the intensity and long duration of the febrile movement. Though reaching their highest development in fatal cases of enteric fever, they are not confined to that disease, nor are they characteristic of it.

The liver is occasionally hyperæmic, but, as a rule, it is normal in appearance; exceptionally it is pale. Its tissue is often softened, and upon microscopical examination the cells are found to be granular and their nuclei indistinct or no longer to be seen. The amount of bile is usually diminished, and in the later periods of the disease it is thin and almost colorless.

---

[1] The Principles and Practice of Medicine, 1886.

The kidneys also show parenchymatous degeneration. The epithelium becomes granular, the contour of the cells indistinct, and the nuclei disappear. These changes affect first the cortex, later the pyramids. In many cases they are but little marked. They are usually associated with albuminuria: Liebermeister[1] states, however, that he has repeatedly noted the absence of albuminuria throughout the whole course of the disease where at the autopsy advanced degeneration of the kidneys was discovered.

Endocarditis and pericarditis are rare. As Strumpell[2] has pointed out, the slight mitral endocarditis sometimes found at the autopsy has little clinical significance. On the other hand, the myocardium shares in the general atrophy which accompanies the disease. The heart becomes relaxed and flabby. In addition to this simple atrophy, the myocardium undergoes parenchymatous or fatty degeneration. This change, according to Payne,[3] consists of three processes: 1, interstitial inflammation; 2, degeneration of the muscular fibres; 3, regeneration of the same.

The first two must be regarded as simultaneous effects of the typhoid poison acting on different tissues, the last as a subsequent process.

1st. The interstitial inflammation shows itself in the form of a small-celled infiltration in the connective tissue of the muscle. Rindfleisch called attention to the fact that the cells are somewhat larger than ordinary lymph- or pus-corpuscles, and suggests an analogy between this process and the lymphatic hyperplasia of the intestines and glands above described.

2d. Many muscular fibres undergo a peculiar form of degeneration. The muscle-substance, after passing through a stage of cloudy swelling, loses its striation and becomes translucent or vitreous. This change is perhaps less frequent in the heart than in the voluntary muscles. In the former, minute granules are deposited in the muscular tissue.

3d. The degenerative fibres are slowly absorbed, but in the mean while there are always seen along with them other immature or newly-formed fibres, indicating a regeneration of the muscular tissue. This new formation apparently takes place within the sarcolemma from the persistent nuclei.

The whole of the destroyed tissue is not always restored, so that a gap partly filled by fibrous tissue remains. In its higher degrees, this degeneration gives rise to changes in the muscular tissue of the heart that are easily recognizable by the unaided eye.

The heart is soft and of a pale-yellow or yellowish-gray color, strongly in contrast with the bright-red hue of the voluntary muscles. Its tissue is easily torn, and the organ thrown upon the table settles down into a formless mass.

Cardiac thrombi are sometimes present, and may cause embolism of the lungs, spleen, kidneys, and other organs.

---

[1] Ziemssen, Cyclopædia of Medicine, vol. i.

[2] Practice of Medicine, Amer. ed., 1887.

[3] Manual of General Pathology, 1888.

Fatty degeneration of the minute arteries of the brain, kidneys, and other organs was demonstrated by Hoffmann, who also called attention to the frequency with which thickening and opacity of the inner coat of the larger vessels, and particularly of the pulmonary arteries, occur. The blood is dark-colored, and there are numerous small, soft coagula. If death take place in the latest stage of the disease or during convalescence, the vessels are frequently nearly empty, the blood thin and watery, and the tissues œdematous.

Changes in the voluntary muscles, similar to those already described as occurring in the muscular tissue of the heart, are of very frequent occurrence. They were originally described by Zenker,[1] who distinguished two forms: first, a granular degeneration which in its highest degree does not differ from ordinary fatty degeneration; second, a waxy or fibrous degeneration by which the muscle-substance is converted into a glazy, colorless mass. The former is more frequent, but the two forms are often associated, sometimes one and sometimes the other predominating. The swollen and vitreous fibres break up into polygonal masses, probably through the contraction of neighboring fibres which are not affected. Transverse ruptures of large bundles sometimes occur, producing hemorrhage or pseudo-abscesses in the substance of the muscular masses.

The muscles most commonly affected are the recti abdominis, the adductors of the thigh, the pectoral muscles, the diaphragm, and the tongue. All the voluntary muscles may, however, be affected to some extent. These changes are most marked usually after two, three, and four weeks. They are not peculiar to enteric fever, but occur in other severe febrile diseases.

The salivary glands and pancreas are frequently found to have undergone parenchymatous degeneration analogous to that which occurs in the other glandular structures of the body.

The organs of respiration show no anatomical changes peculiar to enteric fever.

The epiglottis is congested, sometimes ulcerated or œdematous, or, if diphtheria complicates the case, it may be the seat of diphtheritic exudation.

The larynx may be the seat of more or less extensive ulceration.

The trachea is usually normal in appearance, or somewhat congested.

In the bronchial tubes those changes are met with which underlie the various forms of bronchial catarrh occurring in other diseases.

The lungs almost constantly present changes referable to the enfeeblement of the circulation and the blunted condition of the nervous system. Hypostasis is very frequent; it is limited to the more dependent portions of the lungs. More or less extensive lobular pneumonia of the nature of the so-called inhalation pneumonia is often present. Pulmonary œdema is common. The bronchitis sometimes takes on a putrid character, and the

---

[1] Ueber die Veränderungen der willkürlichen Muskeln im Typhus abdominalis, Leipzig, 1864.

transformed into genuine gan-
only as a complication, but in
case, under circumstances which
localization. For this reason,
to the group of cases thus charac-

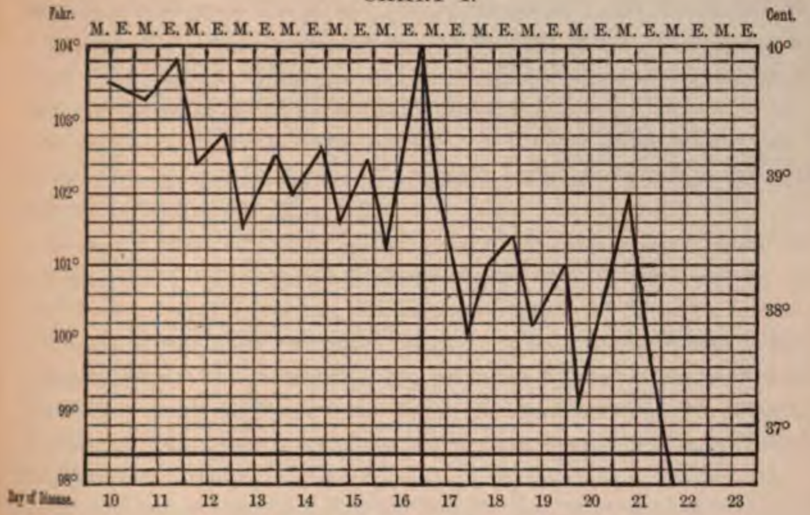
most instances no coarse anatomi-
symptoms during life. More or less
the inner surface of the cranium
periods of the disease. Increased
of the pia mater, and injection of
are frequently observed. Later in
is found œdematous, sometimes
moderate distention of the ventricles,
In rare cases, large cerebral hemor-
been found.

enteric fever in childhood is not only,
the well-marked sequence of phe-
of the sickness in the later periods
children the distinct periods into which
may be more or less successfully divided
development and the successive prominence
able, as a general rule, to divide satisfac-
the two distinct and well-defined stadia
known, the first stadium corresponds to
due to special infection; and it is in this
able to recognize in the blood and tissues
The fever is of subcontinuous type.
hand, corresponds to that period of the
formation and separation of the intestinal
The fever, instead of being subcontinuous,
presents the characteristics of surgical or hectic
to an infective process analogous to that
and not to any specific action of the

stadium is imperfectly developed, short in
—modifications of the course of the fever
in close accord with the fact that in the former
lesions frequently undergo resolution wholly
commonly present some superficial sloughing
exceptionally reach the high grade of development
adult life.

clear, we may consider a corresponding state of
course of the temperature presented by cases of scarla-
disease runs its course without complications, the temper-

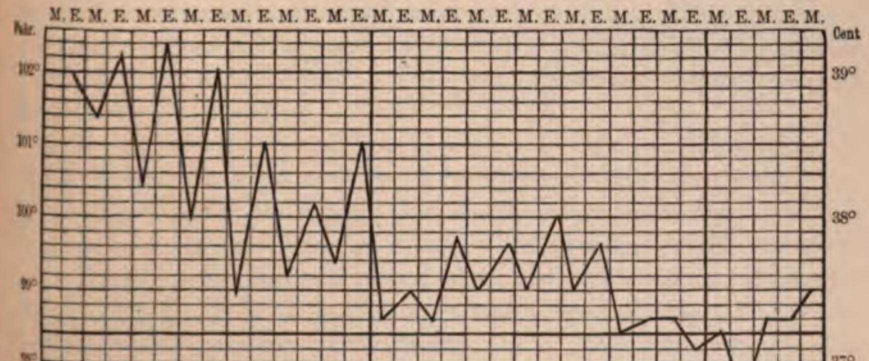
CHART I.



M. K., aged eight years. Enteric fever.

J. C. Wilson, M.D.

CHART III.



G. W., aged nine years. Enteric fever.

J. C. Wilson, M.D.

ature-curve presents a single stadium, and terminates in the course of eight or ten days, usually by a somewhat prolonged critical defervescence. When, on the other hand, it is complicated by secondary infection and an inflammatory implication of the parotid gland, the middle ear, or the lymphatics of the neck, the febrile movement of the primary infection is succeeded by a hectic fever and suppuration, and a second febrile stadium shows itself, which may indefinitely prolong the sickness.

In typhus, a fever without constant or distinct local lesions, we have a single febrile stadium due to the specific infection, which usually terminates by the tenth or twelfth day.

A consideration of the foregoing facts enables us to understand at the same time certain of the modifications of the temperature-curve of enteric fever as seen in childhood and the shorter duration of the disease.

Another usual modification of the temperature in childhood consists in the greater extent of the morning remissions and the evening exacerbations. This finds a ready explanation in the labile tendencies of the temperature of childhood under all circumstances, and is to some extent a trait of every febrile disease in early life.

The corresponding instability of the nutritive processes serves to explain the rapid wasting of children suffering from enteric fever, and has been invoked by Kaulich,[1] though upon insufficient grounds, to explain the somewhat rapid evolution, and consequent relatively shorter duration, of the disease.

The well-known tendency of delirium to take its form from the development and mental habit of the individual in sickness of every kind will serve to explain the fact that in childhood apathy, somnolence, and stupor are much more common than active or even wandering delirium. Other physiological conditions peculiar to the earlier periods of life and capable of analogous modifications of pathological processes will readily suggest themselves to the reader.

It is convenient to sketch first in a general way the clinical course of the disease and afterwards consider in detail the particular symptoms.

A stage of prodromes usually precedes the onset of the fever, which is so insidiously developed that it is impossible to designate the day of its commencement. Patients are noticed to be easily fatigued and indisposed to play; they complain of feeling badly, and of headache, especially frontal headache, which is usually worse towards night. They often complain also of pain and soreness in the muscles. Sleep is restless and broken. The bowels are, as a rule, constipated. The expression is dull, the appetite poor, the tongue coated. This period is of uncertain but brief duration; it gradually merges into the declared disease. Slight irregular chills or repeated attacks of chilliness may mark the beginning of the fever.

Less commonly the disease is preceded by an attack resembling intermit-

---

[1] Jahrb. für Kinderheilk., Bd. xvii., 1881.

between the tenth and twentieth days, and the child enters upon convalescence pallid, feeble, and emaciated out of proportion to the symptoms of his sickness. Thirst diminishes; appetite returns; the mental activity quickens, and the restoration to health is more rapid than in adult life.

Such is the clinical picture of enteric fever as commonly seen in childhood. Not rarely the symptoms are much milder than this account would indicate, often so mild that it is difficult to restrain the patient in bed or to convince his attendants of the actual character of the disorder. On the other hand, cases are encountered marked by the intensity of the morbid processes which characterize the worst cases of adult life. In such instances the disease may be prolonged to the end of the fourth week or beyond it, and the attack may terminate in death, the phenomena under these circumstances being analogous to those seen in adult life,—subject always to the special modifications, as regards the fever, the nervous symptoms, and the nutritive derangements, which have been already pointed out.

Relapses occur in a certain proportion of the cases in childhood and infancy. What this proportion may be it is impossible to state definitely. They appear to be much less common than in adult life.

The relapse constitutes a true second attack of the specific fever. It is due to re-infection, probably from some source within the organism itself, and is attended by the characteristic phenomena of the disease. It is, as a rule, however, of more abrupt onset and shorter duration than the primary attack. It is commonly separated from the latter by an interval of some days, during which the temperature-range is subnormal or normal. (See Chart II.)

It is to be noted that during this period, in cases in which relapse occurs, the spleen remains enlarged.

The relapse is not invariably thus separated from the primary attack, but may occur during its course. It is then termed an intercurrent relapse; and it is to intercurrent relapse that cases of unusual prolongation, in the absence of complications, are to be ascribed. Two or more relapses may occur.

Relapses not infrequently occur after primary attacks of moderate severity; less commonly after grave cases. The opinion at one time held that relapses were much more frequent after primary attacks treated by strict antipyretic methods than after those treated upon the expectant plan does not seem to be well founded.

An attack of enteric fever appears in the great majority of instances to confer immunity against subsequent attacks. To this rule, however, there are exceptions, and numerous cases are recounted in which a second or even a third well-authenticated attack of the disease has occurred in the same individual. It is to the immunity often acquired in infancy or childhood that the insusceptibility of many adults to the disease is to be attributed. A considerable proportion of individuals in local epidemics, where the exposure to the infectious principle must be universal, usually escape the disease.

Very mild cases doubtless frequently escape recognition. The little patients, though far from well, continue to play about with their companions. These cases do not correspond to the "walking cases" of adult life. The intestinal lesions being superficial, the attack is not likely to terminate in hemorrhage from the bowels or in perforation.

ANALYSIS OF THE PRINCIPAL SYMPTOMS.

The Temperature.—The typical typhoid temperature-range of Wunderlich[1] is rarely seen in childhood. Nor do we often meet with charts showing the regular progressive zigzag rise of the first five days, which is of diagnostic importance in adults.

Owing to the difficulties of diagnosis at the beginning of the attack, which are much greater in childhood than in adult life, and to the insidious development of the disease, accurate temperature-observations of the earlier periods are not often taken. Even when cases develop in the wards of hospitals or in the families of well-to-do people and thus come comparatively early under the observation of the physician, the morbid phenomena are not usually such as to lead immediately to the anticipation of a serious disease, nor are there such obvious evidences of fever as to suggest the systematic taking and recording of the temperature.

In those rare instances when the fever begins suddenly with a chill followed by a rise in temperature of several degrees and sweating, there remains the possibility of a previous insidiously-developing febrile movement which has been overlooked. In these cases the rise in temperature following the chill is abrupt, often reaching 103.5° to 104° F. Much more common is a gradual rise in temperature, the curve taking the form of an irregular zigzag and the evening exacerbation exceeding the remission of the following morning by about one degree Fahrenheit, so that the fastigium is reached in the later days of the first week. Here the initial chill is absent, but there is often a slight sense of chilliness or transient shivering during the early days at the time of the evening exacerbation. The fastigium having been reached, the type of the fever becomes subcontinuous, the difference between the evening and the morning temperature being about a degree to a degree and a half.

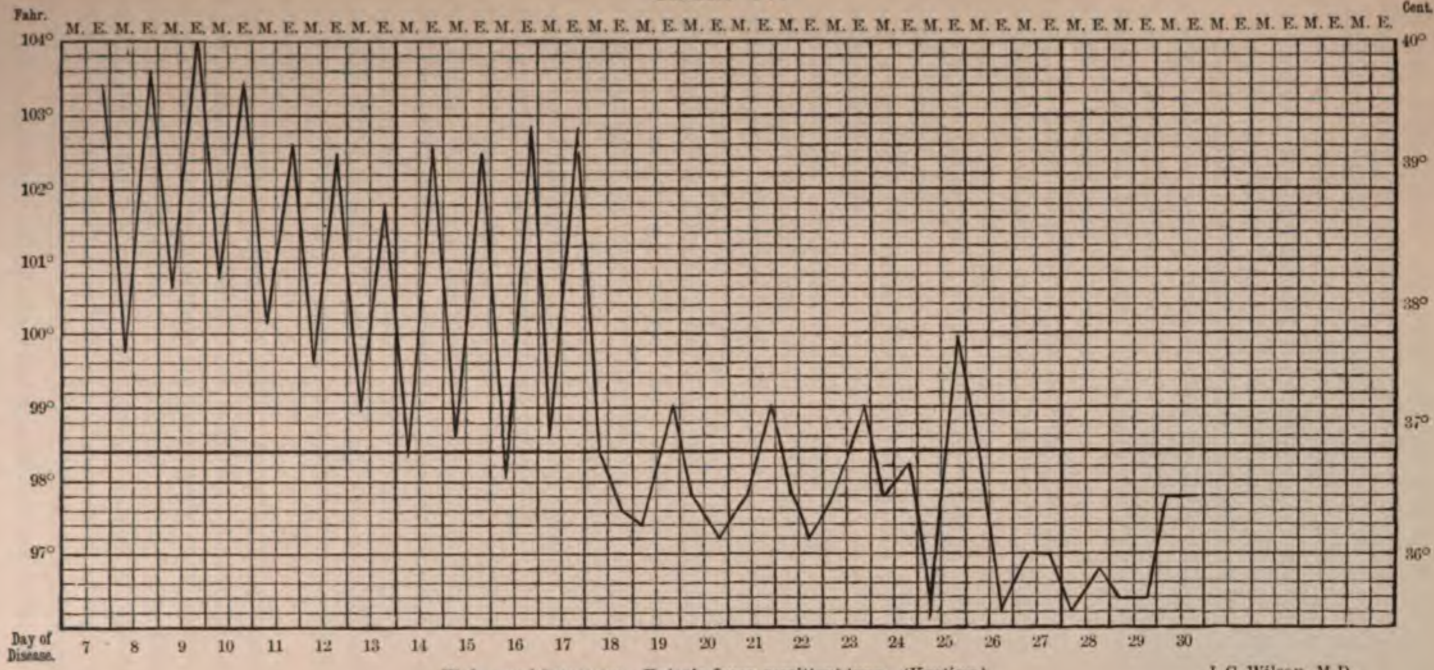
At some time between the middle of the second and the end of the third week, or exceptionally even later than this, the type of the febrile movement gradually becomes distinctly remittent or even intermittent, the remissions and exacerbations being gradually increased. The defervescence thus assumes the form of a rapid or gradual lysis, the fever terminating when the evening temperature falls to normal.

In a considerable proportion of the cases the defervescence takes place by a rapid lysis, without the intervention of distinct remissions or intermissions, the fall being of an irregular zigzag not unlike that which marks the

---

[1] On the Temperature of Diseases, Sydenham Society's Transactions, 1881.

CHART IV.



W. L., aged four years. Enteric fever, remittent type. (Keating.)

J. C. Wilson, M.D.

access. (See Chart III.) Less frequently—and this is especially the case where the duration of the fever is relatively short—the defervescence takes place with considerable rapidity, in itself suggestive of crisis. This is the well-known mode of termination in the abortive cases seen in adult life.

In the early days of convalescence the morning temperature, and at times the evening temperature also, fall to subnormal ranges. (See Chart IV.) During convalescence the temperature is exceedingly unstable. Transient perturbations, with a rise of three or four degrees, to which the term recrudescence of fever has been applied, are brought about by slight causes, among which are errors in diet, especially the eating of meat, constipation, undue muscular effort, and mental excitement.

The temperature-range alike during the fever and during convalescence is liable to modifications in consequence of complications. Abundant blood-loss from epistaxis or from intestinal hemorrhage is apt to be followed by a considerable fall of temperature. This fall is not permanent unless it occur during the defervescence.

The temperature of the disease in childhood reacts promptly and decidedly to antipyretic treatment.

Of eighty cases in which Henoch was able to determine accurately the duration of the fever, in seven it terminated in between seven and nine days; in four, in ten days; in eight, in eleven days; in one, in twelve days; in seventeen, in between thirteen and fifteen days; in five, in between sixteen and seventeen days; in eleven, in between eighteen and nineteen days; in fifteen, in between twenty and twenty-three days; in six, in between twenty-eight and thirty days; in one, in thirty-five days; in one, in forty-two days; and in four, in between forty-eight and forty-nine days.

The occurrence of complications or of intercurrent relapses in the cases of unusually long duration in the above list is not noted.

The height of the fever in the fastigium is variable. In many cases it does not exceed 102.5°–103° F.; exceptionally it reaches 104° or even 105°. The average difference between the morning and the evening temperature is, as a rule, about a degree or a degree and a half. There is occasionally observed a slight exacerbation occurring early in the afternoon, followed by a correspondingly slight remission. The maximum temperature is reached in the evening.

Where two temperature-observations only are made in the course of twenty-four hours, the most convenient time is between seven and eight in the morning and between seven and eight in the evening. In grave cases, and where the temperature is either very high or shows a tendency to sudden changes of considerable extent, observations should be taken much more frequently. A temperature of inverse type has been occasionally noted.

Hyperpyrexia, the temperature reaching 105.8° F. and rapidly rising to 108°–110° F., does not occur in uncomplicated enteric fever in childhood.

The Circulatory System.—In mild cases the action of the heart is but little disturbed. In those of moderate severity it is progressively diminished until the impulse and the first sound are much enfeebled. These changes are less marked, as a rule, in infancy and childhood than in cases of corresponding severity in adult life.

Pericarditis is rare, and must be looked upon, when it occurs, as a complication. A faint systolic apex-murmur is occasionally met with in consequence of the general relaxation of the wall of the heart. Actual endocarditis of a mild grade is probably of more frequent occurrence than the descriptions of the text-books would indicate. Certain it is that a slight mitral endocarditis is occasionally encountered upon post-mortem examination.

The degenerative changes in the muscle of the heart previously described play a large part in the enfeeblement of the circulation: that they are its sole cause, however, is doubtful. It is probable that the loss of cardiac force is largely due to functional disturbances of the nervous system.

It is not uncommon to find later in life in those who have suffered from enteric fever evidences of impaired integrity of the myocardium. There is reason to believe that in a considerable proportion of the cases of enteric fever endocarditis of the branches of the coronary vessels leads to permanent degenerative changes in limited areas of the heart-muscle.

Sudden death in the absence of adequate anatomical lesions, such as occasionally occurs in adult life, does not take place in enteric fever in childhood.

The pulse in children corresponds in a general way to the height of the temperature. The exceptions to this rule are very frequent, a pulse of 120, 108, or even 96, being occasionally met with where the temperature ranges as high as 103° or 104° F.

On the other hand, the pulse may be exceedingly rapid, reaching a frequency of 150 or 180, in cases that terminate in recovery.

It is sometimes irregular both in rhythm and in force. Dicrotism is much more rare in childhood than in adult life; but we must, in this connection, again insist upon the fact that the phenomena of enteric fever in the later years of childhood closely correspond to those of the disease in adults.

The peculiarities of the pulse are in infancy, by reason of the narrowness of the arteries, difficult to recognize, beyond that it is always small and compressible. Extreme irregularity of the pulse is a grave symptom.

During convalescence the pulse is apt to be more rapid than in health, and to show marked variations in frequency. It is, on the other hand, not rarely slower than normal.

The œdema of the ankles and legs often seen in the early days of convalescence must be ascribed to the weakness of the heart and imperfect circulation of the blood.

Collapse is rare in childhood. It occurs only in the graver cases, and may result, as in adult life, from various accidents, among which are intestinal hemorrhage, the shock following perforation, or even sudden copious diarrhœa or violent vomiting.

The Respiratory System.—The frequency of the respiration, in the absence of pulmonary complications, varies with the intensity of the febrile movement. It rises with the pulse; but in cases characterized by an unusually slow pulse there is no corresponding slowness of the breathing.

A certain amount of bronchial catarrh is so frequent that it merits consideration as a phenomenon of the disease rather than as a complication. Cough is much more common in children than in adults, although the corresponding physical signs are in many cases not present,—a fact to be accounted for by the superficial respiration of childhood and the impaired muscular tone of the disease. For the same reason, the respiratory murmur is ordinarily much enfeebled. Upon deep inspiration, such as accompanies crying or cough, mucous râles are distinctly heard. In other cases dry and moist râles are to be heard over all parts of the chest.

In a majority of the cases the bronchitis is of moderate intensity, especially if the patient early receives proper care and treatment; in severe cases, however, there is frequently developed broncho-pneumonia, which manifests itself by notable enfeeblement of the respiratory murmur of the dependent portions of the lungs and by impairment of resonance upon percussion. These changes are apt to affect both lungs, although, as a rule, the signs are somewhat more pronounced upon one side than upon the other. They are largely due to the condition of the nervous system. The patient expectorates little, is apt to swallow food the wrong way, and lies quietly upon his back.

This form of pneumonia, which is the outcome of a severe diffuse bronchitis and has been described as a "deglutition" or "inhalation" pneumonia, was formerly described under the name of hypostatic pneumonia. It occurs during the acme of the febrile movement or early in the defervescence, and frequently amounts to a complication which much prolongs the febrile process.

Hypostatic congestion also occurs in consequence of the feeble circulation; it is limited to the posterior parts and bases of the lungs.

Much more rare is a true lobar or fibrinous pneumonia. It occasionally appears as early as the second or even during the first week, and may attack the lower as well as the upper lobes. In cases where it comes on very early, the diagnosis is for a time obscure. It may sometimes occur during convalescence. It remains an open question as to whether or not this form of pneumonia, occurring in the early period of the disease, is due to a pulmonary localization, or to an independent coincident infection. The pulmonary symptoms, the physical signs, and the post-mortem appearances are not to be distinguished from those of lobar pneumonia occurring as an independent affection.

The same statement is true of pleurisy with fibrinous, serous, or purulent exudation,—a complication, however, which is rare. Henoch has noted in a girl aged four years the occurrence of gangrene of the lung in pneumonia occurring as a complication of enteric fever.

Catarrhal laryngitis with hoarseness, due to the same causes that produce the bronchitis, is occasionally encountered.

An extremely infrequent complication, which appears to be much more rare in this country than in Europe, is ulceration of the vocal cords or of the posterior wall of the larynx.

Perichondritis of the arytenoid cartilages has also been observed. This is a complication of grave import, and may lead to rapid development of the œdema of the glottis. Faucial and laryngeal diphtheria may also occur as complications.

Stenosis of the larynx may occur as a result of (1) œdematous laryngitis, (2) ulcerative laryngitis, and (3) laryngeal perichondritis. In ninety-four cases collected from various sources by Keen in which the age is regarded, six were under fifteen years of age.

Ulceration of the nasal cartilages, resulting sometimes in perforation of the septum, sometimes in a permanent deformity of the nose, also occurs.

Epistaxis is much less common in children than in adults, and is, as a rule, not severe. Forchheimer[1] noted it in five per cent. of seventy cases observed during a recent epidemic in Cincinnati. In the early period of the disease it usually consists simply of the spontaneous loss of a few drops of blood from time to time; occasionally, however, profuse epistaxis occurs among the earlier phenomena. Later in the course of the attack, trifling epistaxis is frequently induced by the patient picking at the nasal orifices. Other nasal symptoms, except a certain amount of dryness of the mucous membrane, are uncommon. It has been said that enteric fever never begins with coryza.

The Digestive System.—The symptoms indicative of disturbances of the digestive organs are much the same in childhood and in adult life. Loss of appetite during the progress of the fever is the rule; a desire for food, the exception; upon the occurrence of convalescence there is usually marked hunger. Thirst is a prominent symptom, but it is generally of moderate amount and readily gratified.

The tongue is in exceptional cases clean and moist throughout the attack; much more commonly it is red at the tip and edges and covered with a pasty, yellowish-white fur, which is apt to separate in the course of the disease, leaving the tongue smooth, bright red, and dryish. It is not usual to find in childhood the hard, dry, and brown tongue which is common in the later stages of the disease in adults. Occasionally the appearance of the tongue differs from day to day. Sordes upon the teeth and gums are not common in childhood. On the other hand, the lips are apt to become cracked and fissured, with the formation of superficial crusts. Superficial aphthous ulcerations likewise occur upon the tongue, upon the buccal mucous membrane, and at the corners of the mouth.

Vomiting, sometimes spontaneous, sometimes following the administration of food or medicine, occurs much more frequently in childhood than in grown persons. It is more common in the first week of the attack, but may occur from time to time throughout the whole course of the disease.

---

[1] Polyclinic, March, 1888.

It is much more apt to be present in severe than in mild cases; but this symptom has not the ominous significance in childhood that it has in adult life. Repeated vomiting occurring at the beginning of the attack tends to increase the resemblance between enteric fever and tubercular meningitis, and renders the diagnosis for the time being obscure.

The condition of the bowels is extremely variable. Constipation is at least as frequent as diarrhœa in the beginning of the attack. In a considerable proportion of the cases the bowels are almost normal as regards the frequency of the movements and the consistence of the dejections throughout the disease. In a limited number of cases there is a marked tendency to constipation; more frequently the constipation of the earlier period of the disease gives way to a more or less frequent diarrhœa in its later course. The number of passages may vary from one to ten, or even to fifteen or twenty, in the course of twenty-four hours. When the movements are infrequent, the dejections are often formed or of a mushy consistence, being usually of a lightish-yellow color; when diarrhœa is present, however, they usually present the well-known appearance of thick pea-soup and divide upon standing into an upper cloudy, quite liquid layer and a lower layer composed of greenish-yellow masses. They have generally an alkaline reaction, and are found upon microscopical examination to contain crystals of triple phosphate. The discharges from the bowels are sometimes of a rather bright greenish color, sometimes of the color described as *café au lait*.

The frequency of the diarrhœa is very often proportioned to the general severity of the case. This rule, however, is not absolute, and the converse of it is by no means to be assumed, since very severe cases, and often those in which there are advanced intestinal lesions, run their course without marked diarrhœa. Diarrhœa, when present, is apt to continue until defervescence is completed, and to be succeeded during the convalescence by constipation, which, when obstinate, is not infrequently a cause of transient recrudescences of the fever. On the other hand, it occasionally happens during convalescence that spontaneous diarrhœa occurs.

Except in the case of very young infants, involuntary evacuation of the bowels or bladder in the bed is unusual. Rilliet and Barthez, Henoch, and others have noted the fact that quite young children during the course of the disease, even when a certain degree of apathy and somnolence is present, frequently call the attention of the attendant at such times. Later in the course of severe cases involuntary discharges are apt to take place.

Upon pressure, the belly is often slightly sensitive, but tenderness is rarely extreme; it is more apt to be present in cases characterized by constipation. It is more difficult to determine the presence or absence of local tenderness upon pressure in the ileo-cæcal region in children than in adults.

For this reason, the symptom is less important. This is also true of borborygmi, a symptom by no means rare in simple diarrhœa. The abdomen is usually normal in contour or moderately distended; in a small proportion of the cases it is flat. Marked tympany is comparatively rare in childhood.

Abdominal pain is often wholly absent. Rather sharp attacks of colic, especially just before an action of the bowels, occasionally occur. Such attacks are also apt to be present from time to time during convalescence; they are sometimes severe and may occasion apprehension lest they be the beginning of actual peritoneal inflammation. Rilliet and Barthez have called attention to these abdominal pains, and record the case of a boy eleven years old who during the course of the disease was attacked with violent pain in the abdomen of thirty-six hours' duration, accompanied by vomiting.

Intestinal hemorrhage occurs as the result of the implication of the walls of blood-vessels in the ulcerative process. It is apt to show itself, therefore, at the time of the separation of the sloughs,—namely, comparatively late in the course of the attack. It is far less common in children than in adults,—a fact which finds its obvious explanation in the relatively slighter lesions of the former period of life. The blood escapes from the vessels into the intestine, and is voided with the stools. It varies in amount from a mere trace to a profuse hemorrhage, which may be followed by lethal collapse. When the amount is small, it is usually dark in color and the later discharges are sometimes tarry. On the other hand, copious intestinal hemorrhage is apt to present the appearance of bright, recently-drawn arterial blood. Hemorrhage from the bowels is in all cases a significant symptom. Even slight hemorrhages may be followed after some hours or days by dangerous or fatal loss of blood.

Earle, of Chicago, has furnished me with the notes of an unpublished case of fatal intestinal hemorrhage in a child twenty-two months old. Post-mortem examination revealed the characteristic lesions of enteric fever.

A case of Henoch's is instructive. Hemorrhage occurred in a girl ten years old in the course of a relapse which took place in the third week. There was first an insignificant bleeding, which was followed upon the next day by very copious hemorrhages, resulting in fatal collapse.

After every intestinal hemorrhage of considerable amount, the anæmia characteristic of the period of the disease in which this accident is liable to occur is intensified, and the signs of collapse are apt to appear. The fall of temperature amounts usually to two or three degrees Fahrenheit, and may reach normal or even subnormal ranges. Hemorrhage, even when large, does not necessarily result in a fatal issue of the case. It not infrequently exerts a favorable influence upon the cerebral symptoms, and occasionally is followed by improved intelligence, permanent reduction of fever, and other signs of beginning convalescence.

More common, but still extremely rare in childhood, is perforation of the floor of an ulcer into the peritoneal cavity. This accident is apt to occur late in the course of the attack. Henoch observed it in a boy aged eleven in the fifth week after convalescence had been apparently established. The solution of continuity in the wall of the gut may consist of a simple, irregular, longitudinal rent in the serous coat, or may present the appearance of a circular or oval opening three or four lines in diameter. The edges of such perforations are usually the seat of a deposit of plastic lymph: hence, if the gut at the point of perforation come immediately into contact with the serous wall of a neighboring coil of intestine or some other viscus, adhesions may take place and extravasation of the intestinal contents be prevented, with the result of a localized, or at all events a plastic, peritonitis. If, on the other hand, the opening communicate without such adhesions directly with the cavity of the peritoneum, extravasation of the intestinal contents is sure to occur. Slight as the amount may be, there results a purulent or even an ichorous peritonitis, with the well-known symptoms of a fulminant attack of this form of serous inflammation: abdominal pain, distention, collapse, eructations and vomiting, a small and frequent pulse, rapid fall of temperature, followed by an abrupt rise and great fluctuations, constitute the too familiar clinical phenomena of this accident.

It is proper to state that statistics of various authorities present a wide range of variation in regard to the frequency of perforation in children. Thus, among two hundred and thirty-two cases according to Barthez and Rilliet[1] it occurred in three only, while among seventy-three persons in whom this accident occurred, observed by Murchison, fourteen were under fifteen years of age.

Peritonitis may occur through direct extension of the intestinal ulceration to the serous membrane without actual perforation. Peritonitis arising in this manner may be either local or general.

I have reported an instance of this kind.[2] The patient was a puny girl, twelve years of age; her defervescence began about the middle of the third week. On the seventeenth day the morning temperature was normal, and on the twenty-first, defervescence was complete. On the twenty-third day after the beginning of the sickness, the patient suddenly complained of severe pain in the abdomen and had a chill, which was followed by vomiting, intense nausea, and thirst; the axillary temperature was 104.5° F.; the belly tense and tender; the pulse small, hard, and 140. Decubitus was dorsal with the knees drawn up. The whole abdomen was exquisitely tender upon palpation, the focus of tenderness being in the right iliac fossa. The urine contained a trace of albumin and a few hyaline casts. Recovery took place in three weeks. On two occasions in the course of the following month, however, a larger meal than usual was followed by fever, lasting two or three days, and by tenderness in the right iliac fossa.

---

[1] Traité des Maladies des Enfans, 2e éd., Paris, 1853.

[2] Philadelphia Medical Times, Dec. 11, 1886.

Strumpell has seen as a result of the peritoneal bands and false membranes thus produced complete occlusion of the intestine: the age of the patient is not stated.

The spleen is very generally enlarged: in this respect enteric fever does not differ from many of the other acute infectious diseases. The enlargement of the spleen is, however, so frequent and so considerable in this disease that it has acquired a certain sort of diagnostic importance.

The difficulty of determining with satisfactory precision the area of splenic dulness in enteric fever is often considerable in adult life, not infrequently impossible in childhood. It is a difficulty sometimes due to lesions of the left lung and pleura, sometimes to the distention of the colon by gas, and very often to the restlessness of the patient. When the enlargement is considerable, it may often be readily made out by gentle palpation, especially at the moment of deep inspiration. It is sometimes tender upon pressure. There appears to be no relation between the degree of splenic enlargement and the severity of the case. The area of splenic dulness usually begins at the lower border of the seventh rib or in the neighborhood of the sixth rib.

In a proportion of the cases the methods of physical examination fail to reveal splenic enlargement. The splenic tumor may occasionally be made out as early as the third or fourth day; in most instances, however, not until the sixth or even the ninth day. The more rapid the rise in fever, the earlier does the spleen enlarge. The enlargement of this organ begins to subside with defervescence; exceptionally the spleen remains enlarged for some days after convalescence is established.

In cases of relapse the spleen has been occasionally found enlarged during the interval between the primary attack and the relapse.

Pain in the splenic region is rare; it may result from laceration of the distended capsule. Softening infarcts may prove the starting-point of peritonitis.

The Nervous System.—The symptoms of disturbance of the nervous system are, as a rule, much less marked in infancy and childhood than in adult life. In some of the cases decided nervous symptoms are lacking throughout the whole course of the disease; in the greater number, however, manifestations of disturbance of the nervous system are present. Headache, increased towards evening, frequently occurs during the period of prodromes and the first week of the disease; it is commonly referred to the forehead or temples; it may affect the whole head; it is occasionally neuralgic in character, and may be intense; it almost always subsides about the end of the first week.

The child is usually apathetic and indifferent, even in cases of moderate severity; it lies with its eyes half closed, lightly somnolent, sometimes restless; when spoken to, turning away, or answering in monosyllables with the evidences of irritation, often, even in advanced childhood, refusing to show its tongue, or, if showing it, forgetting for a few moments to draw it in again, unless told to do so. There is dulness of hearing, sometimes amounting to marked deafness. Dilatation of the pupils is common. Sleep is restless and disturbed; not infrequently wakefulness shows itself as a distressing symptom. Dizziness is not rare.

Among the nervous symptoms must be mentioned cutaneous hyperæsthesia, which is often well marked and extensive.

Delirium is usually mild; it is more apt to occur in the evening or towards night, and is sometimes associated with night-terrors. The common form in childhood is the so-called wandering delirium; it is apt to be transient and recurrent rather than continuous. The severer forms of nervous disturbance are infrequent, and present themselves only in grave cases. In infants and young children, nocturnal delirium is sometimes accompanied or replaced by sudden, sharp, and prolonged outcries, with excitement not easy to pacify. In older children, and especially at the age of puberty, the manifestations of the disturbance of the nervous system are often fully developed, so that we encounter active delirium accompanied by efforts to leave the bed, deep stupor, and tremor of the hands and tongue. Slight twitching of the muscles of the face and of the tendons of the wrists and hands also occurs. It is to these motor disturbances that the name subsultus tendinum was given by the older writers. Plucking at the bedclothes is not common, even in the worst cases of the disease, in childhood.

Grinding with the jaws occurs as an ominous symptom. Persistent tremor of the extremities and lower jaw is apt to be associated with increased tendon reflexes and mechanical excitability of the muscles. In deep coma the muscles become lax, the movements of the eyes are no longer co-ordinated, reflex excitability is diminished, involuntary evacuation of the bowels takes place, and there is often retention of urine.

The view advocated by Griesinger and Liebermeister, and at one time universally entertained, that the nervous symptoms are due to the prolonged high temperature, is no longer generally accepted. It is much more probable that the high temperature and the nervous symptoms have a common cause in the toxic principles with which the blood is charged during the course of the disease.

A number of nervous affections develop in the course of enteric fever or after its decline.

As has been already pointed out in speaking of the anatomical lesions of the disease, actual meningitis is exceedingly rare, notwithstanding the comparative frequency of symptoms suggestive of its presence. In recent years, the occurrence of cases characterized by associated convulsions, paralysis, hyperæsthesia, and opisthotonus, with the discovery upon examination post mortem of the lesions of cerebro-spinal meningitis, has placed this affection indisputably among the complications of enteric fever.

As regards the etiology of meningitis as a complication of enteric fever, two views may be advanced: first, that of localization of the typhoid infection upon the meninges; and, second, that of mixed infection. In the present state of knowledge, this question cannot be decided. I believe it more in accordance with the natural history of enteric fever and cerebrospinal fever to explain their association on the principle of mixed or independent infection.

Neuralgia occasionally occurs; it is sometimes present in the beginning of the attack, but is much more apt to occur during convalescence. Its most common seat is in the area of distribution of the trigeminal and occipital nerves. Pains in the feet and ankles, with tenderness upon pressure and disturbances of sensibility and in some instances slight localized œdema, occasionally occur during convalescence. These pains are due to a subacute peripheral neuritis.

Among the rare nervous accidents of enteric fever in children are paraplegia, hemiplegia, paralysis of the muscles of the eye and of the larynx,[1] and paralysis of single muscles, as, for example, the serratus magnus, or of a single extremity. These accidents usually occur during convalescence. Their tendency is to recovery, which takes place, as a general rule, in the course of some weeks or months.

Aphasia is much more common in children than in adults. It is very often complete. It likewise usually appears during the period of defervescence or in the early days of convalescence, never during the height of the fever. It usually passes away gradually in the course of ten days or two weeks. As Gowers[2] has pointed out, although there is almost complete speechlessness, there is no disorder of speech or partial loss of speech, such as occurs in cases of organic disease of the brain.

Angel Money[3] states that in cases of typhoid fever the knee-jerk is exaggerated and that there is occasional distinct clonus of the quadriceps extensor. This observer found that the muscular irritability was greatly increased, and that when there was a marked degree of irritability the ankle clonus was usually present and easily elicited. I have observed that in deeply soporose cases the tendon reflexes and mechanical irritability of the muscles are distinctly diminished. In a boy nine years old under my charge at the Philadelphia Hospital, the knee-jerk, absent during the height of the fever, only slowly reappeared although convalescence was almost completed. In another case, that of an adult, in the same institution, there was complete abolition of the knee-jerk.

The electrical reactions are found by Money to be altered both quantitatively and qualitatively, the faradic excitability as well as the galvanic, but the faradic excitability is rapidly exhausted. "There is also a qualitative change in the galvanic reactions. The contraction with negative break occurs with a much feebler current than in health, and the contraction from positive may occur as early as the contraction with negative make. These facts show that we have not to do with the ordinary reaction of degeneration."

---

[1] Landouzy, Des Paralysies dans les Maladies aiguës, 1880.

[2] Diseases of the Nervous System, Amer. ed., 1888.

[3] Lancet, Nov. 7, 1885.

The cutaneous reflexes, as the plantar, cremasteric, epigastric, abdominal, scapular, and gluteal reflexes, are increased. These reflex disturbances come on usually in the second week, and gradually increase till defervescence, persisting for some days or a week or two into convalescence.

General convulsions have no place in the symptomatology of enteric fever even in childhood. When present in the early stages of the disease, they are either the manifestation of some pre-existing disorder or indicate an accidental irritation of the intestinal tract; in the later stages they may be due either to a developing meningitis or to nephritis occurring as complications.

The Urine.—Systematic examinations of the urine are much more difficult in young children than in adults. Transitory albuminuria occurs with sufficient frequency to merit consideration as a symptom rather than as a complication. The amount of albumin is usually slight, and casts are rarely present. Bouchard has pointed out the fact that the bacillus typhosus is found only in albuminous urine. Seitz found in cultures from the urinary deposit in seven cases the bacillus typhosus present in two only. In those two cases the urine was albuminous and the number of the colonies obtained was proportionate to the amount of albumin. Enteric fever differs from scarlatina in the extremely rare occurrence of acute nephritis as a sequel. Retention of urine is much less common in childhood than in adult life; nevertheless it occasionally occurs; catheterization is then necessary. Vesical catarrh, urethritis, and epididymitis are liable to result from want of proper precaution in the disinfection of the catheter. Polyuria has been observed in the course of the disease in children.

Menstruation in girls at puberty is apt to be profuse and prolonged. In a girl of fourteen years, recently under observation, menstruation occurred for the first time during the attack and continued for a fortnight. This case was one of great severity and terminated fatally.

The Skin.—The rose spots peculiar to the disease differ in no respect from the eruption as seen in adults. They appear as scattered, pale-red, slightly-elevated (papulo-erythematous) spots of an oval or irregularly circular shape, varying in diameter from one and one-half to three lines. They disappear upon pressure or when the skin is made tense by traction. They occur in crops from time to time during the disease, single spots remaining present from two to three days. They are at first distinctly marginate, but in fading their color blends with the surrounding skin, and there remains a faint brownish-yellow pigmentation, which only gradually wholly disappears. They are to be looked for upon the abdomen, the lower part of the chest anteriorly, and posteriorly between the shoulder-blades; exceptionally they are found upon the anterior and inner surface of the thighs. In three instances in children where the eruption was otherwise relatively scanty I have seen it upon the face. In a small proportion of the cases the spots are absent during the whole course of the disease; as a rule, they are not numerous; exceptionally they are abundant and are distributed to some extent upon the extremities. The appearance of the eruption is usually coincident with the occurrence of the splenic enlargement. Where the fever appears suddenly and rapidly increases in intensity the eruption may sometimes be found as early as the fourth or fifth day. In a majority of cases it is first found somewhere between the seventh and tenth days, and rarely not until the end of the second week. Upon the occurrence of defervescence no more crops appear. If relapses take place, the eruption again makes its appearance.

Sudamina occur in childhood as in adult life. They make their appearance abundantly over the lower part of the abdomen, coincidently with the free sweating which occurs in the latter period of the febrile movement.

True petechiæ rarely occur. They are of unfavorable prognostic import.

Herpes labialis is very rare.

Boils may occur, and abscesses in the integuments, the muscles, or the intramuscular connective tissue are met with infrequently. Suppuration of the lymphatic glands of the axilla or in other regions may also occur. Inflammation of the parotid gland is exceptionally met with in childhood as in adult life. Superficial bed-sores may occur in children who are not carefully nursed. Their most frequent site is over the sacrum and trochanters; occasionally they form at the elbows, heels, or occiput. The hair falls out during convalescence. The new hair is often lacking in lustre, but gradually acquires its normal appearance. The nails, both of the hands and of the feet, show transverse markings that indicate the impaired nutrition of the tissues during the attack. These markings extend across the whole width of the nail. The portion of the nail developed during the attack is duller than the rest, rough, white, and more or less thin. Similar changes occur during the course of other acute febrile diseases.

A diffuse, faint, erythematous rash is not infrequently observed in children, and even in adults with white delicate skins, during the first week. It is usually most marked over the abdomen and upon the flexor surface of the limbs. It disappears upon pressure. Its usual duration does not exceed three or four days, although Murchison states that it occasionally persists throughout the course of the fever. When more than usually intense and associated with a slight erythematous sore throat, it may lead to an error in diagnosis and be mistaken for scarlatina.

Taches bleuâtres—spots of a delicate bluish or bluish-brown tint, irregularly rounded form, three to eight lines in diameter, not raised above the surrounding surface, nor affected by pressure—are occasionally met with in enteric fever and some other diseases in adults. They do not occur in children.

Facial erysipelas occasionally occurs at the end of the attack or during convalescence in childhood as in adults; in the former it is, however, a much less serious complication. It is apt to terminate critically in the course of four or five days. Gerent[1] collected sixty-four instances out of 3910 cases, at all ages, observed by various clinicians.

---

[1] Thèse de Paris, 1883.

Suppurative otitis media, generally one-sided, is by no means an infrequent complication. It is usually of moderate intensity, and if properly cared for terminates in complete recovery during the period of convalescence from the fever.

Lesions of the Osseous System.—Inflammatory changes in the bones are relatively common in infancy and adolescence. The process implicates, according to Gelez,[1] alike the substance of the bones, the marrow, and the periosteum. Keen,[2] of Philadelphia, reported in 1878 an important series of cases of disease of the bones occurring after enteric fever. Of forty-one cases studied by Mercier,[3] the age of the patient was below twenty years in nineteen cases; between twenty and thirty years in eleven cases; between thirty and forty years in six cases; and over forty years in five cases.

The relative frequency with which the divisions of the skeleton are implicated is indicated by the following table:

Bones of the head	22 cases.
Trunk	7 cases.
Superior extremity	6 cases.
Inferior extremity	42 cases.

The symptoms are local. There is pain, at first vague, speedily becoming localized, usually severe, lancinating, aggravated at night often to such a degree as to render sleep impossible. The pain is associated with great tenderness. Localized tumefaction of the soft parts, with or without redness, speedily follows. After a time fluctuation appears, and one or more fistulous openings are formed, which discharge a small quantity of pus. These sinuses frequently close spontaneously, only to open again. The usual termination is thus in suppuration and necrosis.

Lesions of the bones are more common during the convalescence from prolonged attacks. They are due to disturbances in the blood-supply; traumatism plays only a secondary part in their causation.

Early surgical intervention is imperatively demanded.

Spontaneous dislocations are among the rare accidents of enteric fever in childhood. They are of the nature of "distention-luxations," and are probably due to subacute synovitis with gradual serous distention of the capsular ligament. They have been fully described by Keen, who collected forty-three cases at all ages, in which spontaneous dislocation occurred twenty-seven times in the hip, twice in the shoulder, and once in the knee. Fifteen of the hip cases occurred in enteric fever; a number of these were in children under fifteen years of age.

In a boy aged eleven years and eight months, the subject of rickets in infancy, but otherwise healthy and of healthy parentage, Woronichin[4] observed acute hip-joint disease developed in the early convalescence from an attack of enteric fever in which defervescence was complete upon the twenty-first day.

---

[1] Thèse de Paris, 1884.

[2] Toner Lecture, v., 1877.

[3] Revue Mensuelle de Méd. et de Chirurg., No. 3, 1879, pp. 21 et seq.

[4] Jahrbuch für Kinderheilkunde, N. F., xxiv., 1884.

Henoch[1] saw in a boy eleven years old in the third week of convalescence from enteric fever a synovitis of the left wrist-joint, and encountered in another case a synovitis of the right knee-joint.

Freundlich[2] observed among two hundred and twenty-eight cases of enteric fever acute synovitis of different joints in four cases. On the other hand, Griesinger, Barthez and Rilliet, Steiner, West, and others make no mention of diseases of the joints as sequels of enteric fever.

Complications and Sequels.—In the foregoing analysis of the special symptoms it has been necessary to allude frequently to the complications of enteric fever; to consider the complications in detail would necessitate much unnecessary repetition and unduly extend the limits of this article. A few additional remarks are, however, necessary.

It is obvious that no hard-and-fast line can be drawn between the complications of an acute febrile disease and the mere intensification of certain processes with corresponding prominence of local symptoms. I am in the habit of regarding intestinal hemorrhage, perforation, and peritonitis as complications. To the same category it seems proper to refer such results of parenchymatous degeneration as atrophy of muscles, abscess of muscles, parotitis, and nephritis. The category to which hypostasis, œdema, thrombosis, embolism, and infarction, with their results, should be referred is a matter of opinion. Whether these processes should be regarded as belonging to the primary disease or as complications of it would depend, to a great extent, upon the prominence of the morbid phenomena to which they give rise. On the other hand, pneumonia, erysipelas, phlegmonous abscesses, diphtheria, and other septic processes are obvious complications, concerning the relation of which to the original disease there can be no question.

Enteric fever during its course confers no immunity from the ordinary diseases of childhood.

Scarlatina may immediately precede, coexist with, or follow enteric fever. Taupin, Murchison, and others have recorded instances in which patients suffering from scarlet fever have developed enteric fever, or in which the enteric fever has merged into scarlatina, and other instances in which the eruptions of the two diseases have coexisted.

Measles may also develop during the course of typhoid fever. Instances of this association of the two diseases have been reported by Kesteven,[3] Barthez and Rilliet,[4] and Ringer.[5] Quite recently, Chrystie[6] has reported an interesting case of this kind. (See Chart V.) A boy in his twelfth year was doing fairly well during the third week of a well-characterized attack of enteric fever; on the sixteenth day of his attack two other children in

---

[1] Vorlesungen über Kinderkrankheiten, 1881.

[2] Deutsches Archiv für Klinische Medicin, Bd. xxxiii. Hft. 3 u. 4, 1883.

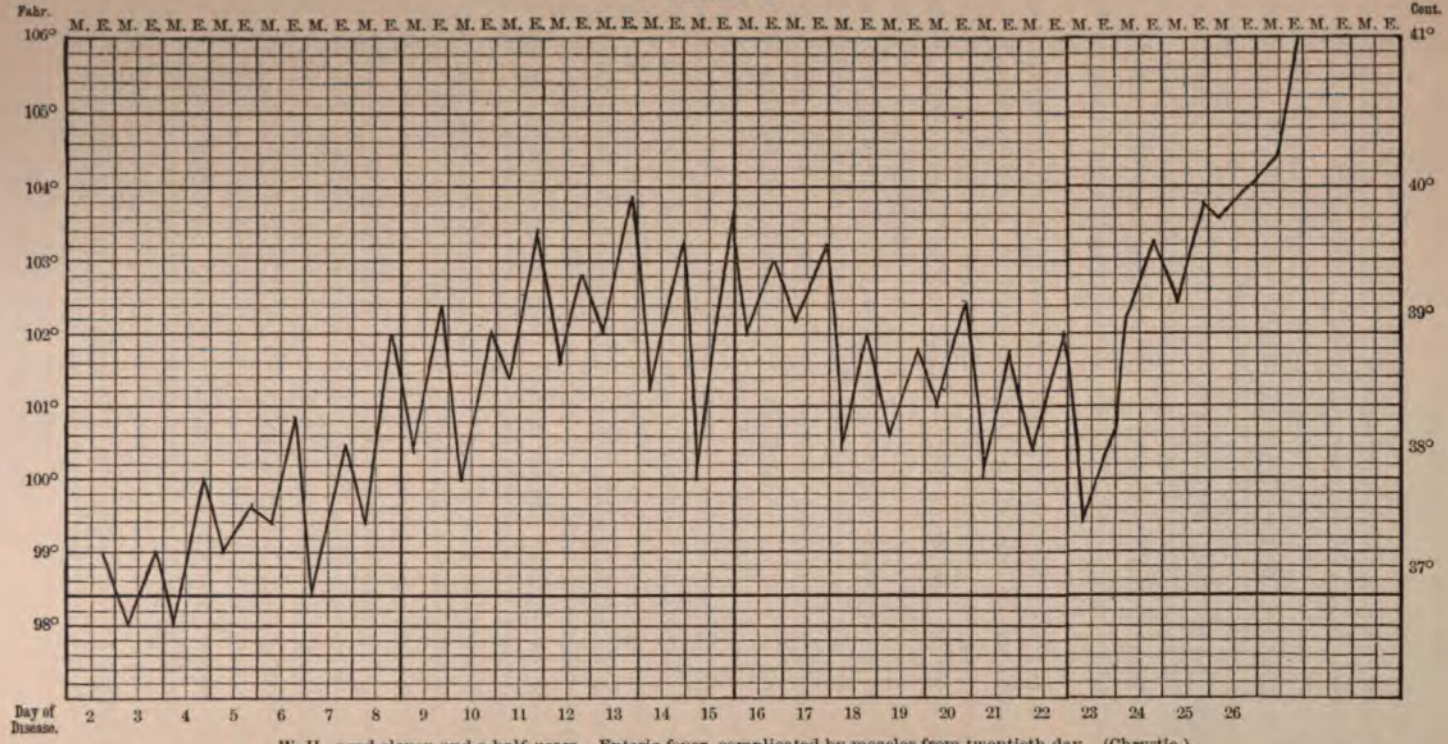
[3] Lancet, vol. i., 1866.

[4] Loc. cit.

[5] Lancet, June 30, 1888.

[6] University Medical Magazine.

CHART V.



W. H., aged eleven and a half years. Enteric fever, complicated by measles from twentieth day. (Chrystie.)

J. C. Wilson, M.D.

the house developed measles; on the twenty-third day the enteric fever patient showed the symptoms of measles; five days later his respiration became embarrassed, and he died in convulsions with a temperature of 106° F. A number of instances have been recorded in which measles has developed during the convalescence from enteric fever.

Pertussis has likewise been observed during the course of enteric fever. Gillespie[1] has reported a case of this kind.

The coexistence of diphtheria and enteric fever is much more frequent.

Tuberculous infection often occurs during or immediately after enteric fever: hence pulmonary phthisis is not a rare sequel. Tuberculous meningitis and tuberculous ulceration of the intestine are also encountered as sequels.

I have reported a case of enteric fever in a precociously developed girl at the age of puberty in which death occurred at the end of the first week from intercurrent fulminant peritonitis of pelvic origin. At the autopsy the lesions of both conditions were found. The peritonitis resulted from acute double septic salpingitis.[2]

Crural phlebitis, which was noted in fully one per cent. of the cases under Murchison's care, does not occur in childhood.

Arterial thrombosis resulting in spontaneous gangrene is among the rarest of the accidents of enteric fever in childhood.

Asch[3] reported a case of abscess of the liver occurring as a sequel of enteric fever in a previously healthy boy twelve years of age. The primary disease ran its course without notable complications. On the twenty-first day there was vomiting, which was repeated on the following morning. On the twenty-seventh day there was a violent chill, and the temperature rose to 41.1° C. (106° F.). Two days later the patient complained of pain in the region of the liver, and a tense, elastic tumor reaching to the navel was observed; there was no fluctuation; jaundice was not observed; the spleen was only moderately enlarged, and there was no distention of the superficial epigastric veins. The type of the fever was remittent; the patient sank rapidly, suffered from profuse sweating, was delirious, and died in collapse on the thirty-fifth day. Upon post-mortem examination, there were found typhoid ulceration of the intestines in the stage of cicatrization, and a liver-abscess, the result of a pylephlebitis due to suppuration of the mesenteric glands. On the upper surface of the right lobe of the liver, which was attached to the diaphragm by recent adhesions, were eight or ten small abscesses. A larger abscess occupied the substance of the right lobe. In this case there was no repetition of the chill which occurred on the twenty-seventh day.

Burder[4] has reported the case of a boy nine years old in which the liver upon post-mortem examination was found to be the seat of a number of small abscesses and there was superficial ulceration of the Peyer's patches.

---

[1] Edin. Med. Jour., May, 1870.

[2] Archives of Pediatrics, July, 1887.

[3] Berlin. Klin. Wochenschrift, 1882, 51.

[4] Lancet, 1874, ii. 552.

Diagnosis.—The diagnosis of well-developed cases of enteric fever in childhood after the first week is usually unattended with difficulty. During the first week, however, it is often impossible to form a positive diagnosis; but even then the nature of the disease may be suspected if there be febrile movement with nocturnal exacerbations each night attaining a higher temperature, and especially if there be bleeding at the nose, diarrhœa, either spontaneous or readily produced by laxatives, appreciable enlargement of the spleen, and headache.

The direct diagnosis of the developed disease rests upon the continuance of the febrile movement and the appearance of abdominal symptoms,—namely, diarrhœa, abdominal pain, moderate tympany, and enlarged spleen. If, in addition to these phenomena, lenticular rose spots appear, the diagnosis becomes certain.

Plate or potato cultures of blood drawn from the spleen, of the sediment of the urine when albuminous, or of the fæcal discharges, afford an additional means of diagnosis in obscure cases.

More useful, because more available, as a means of diagnosis, is the occurrence of well-marked typical cases in adults or children in the same house or locality. It is not easy to exaggerate the importance of the light shed upon doubtful cases by coincident or recently-preceding house and local epidemics.

The differential diagnosis from the other febrile disorders which more or less closely resemble enteric fever is, in the absence of the characteristic eruption and the abdominal symptoms, sometimes attended with considerable difficulty.

The diseases with which enteric fever in childhood is likely to be confounded may be divided into two groups: first, those which resemble it in the first week of its course; and, secondly, those which resemble it in its more advanced stages. To the first group belong simple continued fevers and the exanthematous diseases. Diarrhœa is not, however, present in these diseases, nor is their onset characterized by the occurrence of marked prodromes. Furthermore, the character of the temperature-range of all these affections differs greatly from that of enteric fever, being marked by an abrupt rise which lacks the distinct morning remissions of the typhoid and attains its maximum with greater rapidity. Moreover, simple continued fever comes to an end in less time than is required for the full development of typhoid. The exanthemata cannot be distinguished from enteric fever with absolute certainty in their pre-eruptive periods: notwithstanding this, the presence of naso-pulmonary catarrh in a doubtful case would lead us to suspect measles, or a sore throat would lead us to suspect scarlet fever, while the intensity of the febrile movement and the lumbar pains in smallpox serve to distinguish it in its early stages from enteric fever.

After the first week, enteric fever may in some instances be confounded with the following diseases: remittent fever, small-pox, influenza, entero-colitis, peritonitis, meningitis, tuberculosis, and trichinosis.

Remittent Fever.—Enteric and remittent fevers not infrequently prevail together in malarious countries, and physicians practising in such regions are familiar with the form of fever frequently designated typho-malarial, which is, in fact, enteric fever modified by malarious influences. On the other hand, severe remittent fever not infrequently presents clinical resemblances to enteric fever, particularly when complicated with marked intestinal symptoms. Thus, vomiting, diarrhœa, splenic enlargement, cerebral symptoms, and the condition known as the typhoid state may occur in both diseases. The more important points of distinction are found in the eruption and the subcontinuous or imperfectly remittent character of the temperature-range in the second week of enteric fever and its longer course.

In a considerable number of the cases formal rules for the discrimination of the two diseases are unavailing; only by prolonged study of the complexus of symptoms presented by the patient does the diagnosis become possible.

Bacteriological investigation will, however, at once and definitely determine the presence or absence of any form of malarial disease. The concurrent testimony of all observers who have carefully studied the subject serves to establish the fact that in malarial diseases, properly so called, the blood-parasites described by Laveran, Marchiafava and Celli, Golgi, Osler, Councilman, and others, are invariably present in one or another of their forms; that is, as (1) amœboid bodies in the red corpuscles; (2) pigmented bodies in the red corpuscles; (3) larger solid bodies in the interior of the red corpuscles; (4) free pigmented crescents, which crescents may sometimes be seen to develop in the interior of the red corpuscles; (5) rosette forms; (6) scattered small bodies, the result of segmentation of the rosette forms, —described with great fulness by Golgi; (7) flagellate organisms, round or ovoid or pear-shaped, with finely granular protoplasm containing pigment with flagella variable in number, one, three, or four being observed at different times; (8) small round pigmented bodies one-fourth to one-half the size of the red corpuscles.

The absence of the characteristic rash of enteric fever loses its diagnostic value in doubtful cases, in view of the fact that in a small proportion of the cases the eruption does not appear during the whole course of the disease.

Variola.—Murchison states that he has frequently known a copious eruption of lenticular spots to be mistaken for small-pox. This is an error that should under no circumstances occur. The eruptions are essentially unlike: they differ in date of appearance, in character, and in evolution. The rose rash of typhoid rarely appears earlier than the sixth or seventh day of the illness. It is only in exceptional cases present upon the face. It disappears on pressure, and undergoes little or no change from the time of its appearance until it begins to fade; that of variola appears during or after the third febrile exacerbation of the initial stage,—that is, upon the third day of the disease; it shows itself first upon the face and hairy scalp; from the beginning it is hard, shot-like, and acuminate; it undergoes characteristic and unmistakable changes with great rapidity, and leaves a more or less persistent conspicuous scar.

Influenza occasionally closely resembles enteric fever. The following symptoms are common to both affections: fever, attended by weakness, sleeplessness, delirium, sweating, sometimes diarrhœa; pulmonary catarrh, deafness, epistaxis, and dry, red tongue are likewise seen in both. The differential diagnosis rests chiefly upon the occurrence of influenza in widespread epidemics, the short duration of the attack, the atypical temperature-curve, and the absence of the eruption and the abdominal symptoms that are usually associated with the diarrhœa of enteric fever.

Enteritis and *entero-colitis* may be confounded with enteric fever. These are, however, local diseases, the fever and constitutional disturbances of which are symptomatic. The spleen is not commonly enlarged, rose spots are absent, the abdominal pain is more conspicuous and severe than that of enteric fever, and the whole attack is usually of comparatively short duration.

Peritonitis due to other causes than perforation is to be discriminated from that arising in the course of enteric fever by the antecedent history of the case. If the patient, however, does not come under observation until after the appearance of the symptoms, it may be impossible to determine whether they are due to perforation or not.

Meningitis, whether secondary or occurring in the form of epidemic or sporadic cerebro-spinal fever, presents marked points of difference from enteric fever, yet this disease has in some instances been at first mistaken for it. The differential diagnosis would be determined by the abrupt onset, the acute headache, the frequent vomiting, the constipation, the irregular temperature-curve, the rapid evolution, and the herpetic and petechial eruptions of meningitis.

Acute tuberculosis presents many points of resemblance to enteric fever.

The chief points of difference are these: in enteric fever the temperature-range is typical or more or less conformed to a definite type, whereas that of tuberculosis is extremely irregular. In enteric fever diarrhœa and some degree of tympany are common; in tuberculosis diarrhœa is rare and the abdomen is apt to be flat and often scaphoid. In enteric fever epistaxis and enlargement of the spleen occur; in tuberculous meningitis these symptoms are rare or absent altogether. The headache of enteric fever is dull, while that of tuberculous meningitis is acute and commonly associated with intolerance of light and sound. In enteric fever vomiting is much less common than in tuberculous meningitis. Convulsions, especially in the early part of the disease, are likewise rare, and the headache of enteric fever, as was pointed out by Jenner, disappears upon the occurrence of delirium, whereas in tuberculous meningitis headache and delirium may alternate from the beginning.

Trichinosis.—In trichinosis there is pyrexia with vomiting and diarrhœa. The rose spots do not occur, and epistaxis and enlargement of the spleen are rare, while, on the other hand, the severe muscular pains and tenderness due to the myositis peculiar to the disease, and the local and general œdemas which are almost constant symptoms in trichinosis, are absent in enteric fever.

Prognosis.—The death-rate among children in the first year is high, especially among the new-born. Taking all the cases together, the mortality in childhood is decidedly lower than in adults, probably not exceeding one per cent. The published statistics relating to mortality are without value, except as showing these general facts. Enteric fever, like all the acute infectious diseases, shows an extremely variable intensity in children, the severe cases, however, being the exception rather than the rule.

Among the conditions which tend to make the prognosis unfavorable are (1) the previous poor health of the patient, hereditary syphilis, local or pulmonary tuberculosis, chronic catarrhal bronchitis, previous unwholesome sanitary surroundings, improper food, or other causes tending to impair the powers of resistance of the organism; (2) the intensity of the infection as manifested by the rapid development of severe symptoms, intense pyrexia, failure of heart-power, ataxic phenomena, and the occurrence of multiple cases in the same house or in the immediate locality; (3) intestinal symptoms of a high grade, as copious diarrhœa, meteorism, abdominal pain, and the like. It is to be added that prolonged and intractable vomiting has an ominous prognostic import. Finally, (4) complications, as intestinal hemorrhage, perforation, local or general peritonitis, ulcerative endocarditis, meningitis, nephritis, diphtheria, croupous pneumonia, pleural effusions, and the acute exanthemata, arising as intercurrent or consecutive affections, render the prognosis extremely grave.

Treatment.—This division of the subject embraces the following topics, each of which demands separate consideration: 1, prophylaxis; 2, general management of the patient, and dietetics; 3, special forms of treatment, and the treatment of symptoms, complications, and sequels; 4, the management of the patient during convalescence.

Prophylaxis.—A knowledge of the cause of enteric fever and of the ways by which the disease is propagated warrants the confident belief that it may not only be greatly restricted in its prevalence, but may even be ultimately got rid of altogether.

An efficient prophylaxis is theoretically within reach; its practical results in localities in which the disease is endemic depend upon the energy and steadfastness with which measures for the destruction of the infecting principle and the prevention of its spread are carried out. What these measures are is to be directly deduced from the statements made regarding the causation of enteric fever in a previous section of this article. They belong to the subject of public hygiene, and are of sufficient importance to demand the closest attention of all local and general sanitary organizations;

they are, nevertheless, largely within the personal control of the physicians of every community. It is the highest duty of the doctor to see to it that no new case of disease arise by direct or indirect contagion from any patient under his care. In enteric fever we have to do with a disease in which this is wholly possible. Not only may the spread of the typhoid bacilli be prevented, but they may be absolutely destroyed. The efficiency of the measures of prophylaxis adopted in any given case will be proportionate to the belief in the material nature of the cause of the disease and in the possibility of at once and definitely destroying that cause by disinfection.

The danger that a house-epidemic of enteric fever may arise from a single case suffered to become a focus of infection is to be constantly borne in mind. It is not house-epidemics alone that are to be prevented, but also the spread of the disease to distant points in consequence of the pollution of running streams or other sources of water-distribution, or in cities by way of continuous sewer-systems that may convey the poison to comparatively remote localities.

The one efficient measure of prophylaxis that includes all others is the proper treatment of the dejections. The fæcal discharges of every case, and the urine when albuminous, must be promptly and thoroughly disinfected. This is to be accomplished by the action of powerful chemical agents. Chloride of lime of the best quality dissolved in pure water in the proportion of six ounces to the gallon may be used for this purpose. One quart of the solution is to be poured over each discharge, thoroughly mixed with it, and the vessel allowed to stand for an hour or more before being emptied into the privy-vault or water-closet. If the discharge be very copious, it is advisable to use even a larger amount. For the disinfection of solid fæcal matter the above solution should be of double the strength. The matter to be disinfected must be exposed to the action of the solution for four hours, and solid masses are to be broken up by agitation of the vessel. Solution of carbolic acid 1 to 20 or of sulphate of copper 1 to 25 may also be used for this purpose, but the best of all is a solution of corrosive sublimate of the strength of 1 to 500. This fluid should be colored red by the addition of potassium permanganate and kept in a glass bottle or earthen crock, for the reason that the corrosive sublimate is decomposed with the precipitation of mercury by contact with copper, lead, or tin. No stool from a case of enteric fever should be thrown into a closet without having been previously disinfected as above. Great care must be taken to prevent the contact of the discharges with the wood-work of the seat. The closet is to be fully flushed several times a day, and in the intervals of its use a quantity of carbolic-acid solution or chloride-of-lime solution should be allowed to remain in the hopper.

A privy-vault requiring disinfection may be treated with two or three pounds of corrosive sublimate dissolved in a large quantity of water and slowly poured into the vault. During an epidemic, chloride of lime should be freely sprinkled over the surface of the contents of the vault every day.

Prolonged boiling will destroy the vitality of all known disease-germs. There is, therefore, no better way of disinfecting clothing that can be washed than to subject it to the ordinary operations of the laundry. Clothing may also be disinfected by immersion for two hours in a solution of corrosive sublimate of 1 to 1000, or of sulphate of copper of 1 to 100, or of carbolic acid of 1 to 50, or of chloride of lime of 1 to 100. The bleaching properties of chloride of lime must not be overlooked. Clothing should not be allowed to accumulate in the sick-room, but should go to the laundry as promptly as can be arranged. It should at once be freely sprinkled with one of the above solutions by the nurse.

Search must in all instances be made for the original cause of infection, and measures taken to correct faulty arrangements which lead to the pollution of the drinking-water or of the air. During the prevalence of enteric fever in local or general epidemics all drinking-water and milk should be subjected to boiling; especially is it important at such times that the milk supplied to children should be sterilized by means of the Soxhlet apparatus.[1]

2. *The General Management of the Patient, and Dietetics.*—The successful treatment of enteric fever in childhood is largely dependent on the attention which is given to the general management and nursing of the patient.

In the first place, it is important to see that he is not exposed to the further action of the poison. If the original source of the infection be found upon inspection to be connected with faulty sanitary arrangements in the house or in the neighborhood, it may be necessary to remove the patient to more favorable surroundings. In hospitals, enteric fever patients are usually treated side by side with the patients suffering from other diseases. This practice is unattended with the danger of the communication of the disease, if proper precautionary measures are taken with reference to the disinfection and removal of the dejections and to the cleanliness of the patient's person and bedding.

In private practice among the well-to-do classes children commonly come under observation during the prodromic period or early in the first stage of the disease. If the fever have already declared itself, the use of the thermometer will put the physician upon his guard as to the nature of the sickness. If in cases seen during the period of prodromes the symptoms are such as to excite a suspicion as to the nature of the disease, the patient should be ordered to bed. Should the malady prove to be in fact a simple ailment, nothing is thus lost; if, on the other hand, the symptoms subsequently prove to have been those of the forming stage of enteric fever, early rest in bed cannot fail to influence favorably the subsequent course of the attack.

The strict rules in regard to absolute rest in bed, and to the use of the bed-pan and urinal, which must be enforced in adults, cannot well be carried out in the case of young children.

---

[1] München. Med. Wochenschr., Nrs. 15 u. 16, 1886.

The room should be large and well ventilated; the temperature should be maintained at 66° to 70° F. It is desirable that the apartment should be heated by an open fireplace rather than by furnace-heat. Thorough ventilation must in all cases be secured both day and night, and, whilst direct draughts are to be avoided, it must be impressed upon the attendants that fever patients are not likely to take cold.

The bed-covering should be light. The body should be sponged twice a day with water containing Labarraque's solution, aromatic vinegar, or alcohol in small quantities.

Among the minor duties of the nurse, which are not, however, of inferior importance, are the frequent changing of the position of the patient's body, moistening his mouth, cleansing his tongue, the prevention of the accumulation of sordes, and the most scrupulous care of his person in other respects. If the evacuation of the urine and fæces in bed cannot be prevented, the discharges and soiled clothing are to be changed without loss of time and immediately disinfected. In severe cases it is sometimes convenient to use two beds, the patient being occasionally lifted from one to the other.

Fluid is to be administered freely. The best drink is pure water, either of the temperature of the room or iced. Wine-and-water, milk-and-water or milk-and-seltzer, koumys, kéfir, matzoon, thin barley-water, or water commingled with jelly, may be occasionally given instead of simple water. It frequently happens that patients fail to obtain the necessary amount of drink unless it is proffered them, even when apparently fully conscious. It is important, therefore, that fluid be offered at short intervals; it is often taken with eagerness, though not asked for. The amount at each time must be moderate.

The diet must be rigidly restricted. The directions of the physician as to its kind, its quantity, and the intervals at which it is to be given must be definite and explicit. A record of the amounts given, as well as of the intervals of the administration of food, is to be kept by the attendant and submitted at each visit. Neither general directions nor general reports are sufficient. The diet throughout should be nutritious, easily digestible, and liquid. If overfed, the patient suffers from indigestion and an aggravation of the intestinal symptoms; if underfed, the derangement of nutrition will be increased and the convalescence prolonged. It is desirable to give the maximum amount of proper food that can be assimilated, and not to exceed this amount. How much this may be, can be determined only by careful study of individual cases. During the early stages of the disease, or indeed up to the middle of the second week, not only is it desirable that the diet of the patient should be very digestible, but it is also important that it should be of only moderate amount. After the middle of the second week, as much food is to be given as can be properly digested. Milk and milk foods, broths, soups, and meat juices constitute the diet. Once or twice a day in older children a little coffee, largely diluted with milk, may be administered. If the patient craves it, a small quantity of thickened gruel or arrow-root or bread-and-milk may be given once a day. Some patients do better with an occasional meal of such semi-solid food; it is, however, to be said that, as a general rule, starchy foods are objectionable.

The details of dietetics must in every case be determined in accordance with the age of the child and the previous character of the diet in very young children. To milk, unless otherwise diluted, lime-water must be added in proper proportions, in order to prevent the formation of firm curds in the stomach. To this end, it is also desirable that the milk should be administered slowly. If the milk thus treated be not well digested, it should be peptonized or sterilized.

Alcoholic stimulants form no necessary part of the routine treatment of enteric fever in children. In the early stages of the disease, their use, except to meet special indications, is probably in most cases injurious rather than beneficial. In the later stages the indications which call for their administration are twofold: first, great general prostration, as manifested by weakness of the heart's action; secondly, prominence of nervous symptoms: thus, a feeble or scarcely-perceptible cardiac impulse and a correspondingly faint or almost inaudible systolic sound call for their administration; while such evidences of nervous prostration as marked delirium, stupor, tremor, subsultus tendinum, and the like are best combated by the administration of stimulants. Alcohol is also indicated where the symptoms which attend extensive and deep intestinal ulceration, such as frequent diarrhœa, tympany, and great tenderness, are marked. It is impossible to lay down any general rule as to the amount. The quantity should be only as much as is necessary to modify the symptoms for which it has been prescribed. The character of the systolic heart-sound, the pulse, and the nervous symptoms are our best guides as to the amount and the frequency of its administration. If the urine is albuminous, alcohol is to be given with the utmost caution, and its effects upon the amount and character of the secretions must be carefully investigated at short intervals. To sum up, alcoholic stimulants are to be given according to the special indications of each individual case: they are not required at all in many cases, are useful in a few, indispensable in some. Alcohol may be administered in the form of milk punch, wine whey, port or Tokay wine, or champagne.

3. *Special Forms of Treatment, and the Treatment of Particular Symptoms and Complications.*—The management of enteric fever in childhood may be conducted in accordance with (*a*) the expectant, (*b*) the expectant symptomatic, or (*c*) the specific or etiological plan. The maxim that the patient rather than the disease is to be treated is specially applicable to this disease. The preponderance of cases of mild form, the inherent tendency to recovery, the exceptional occurrence of grave cases, and the peculiar difficulties which they present from the stand-point of therapeutics render

it difficult to formulate rules of treatment that will be applicable to all cases of the disease.

(*a*) The expectant plan.—Many of the milder cases do well without any medication whatever. Rest in bed, careful nursing, and a well-regulated dietary constitute all that is necessary for the proper management of the case. With moderate fever, absent or insignificant chest-symptoms, a good heart, and little or no evidence of serious intestinal lesions, there is no need for the administration of drugs. In such cases there is often a slight tendency to constipation. At most, then, we may commence our treatment advantageously with a purgative dose of calomel, which may be from time to time repeated until the end of the first or the middle of the second week of the attack. After this time constipation is best relieved by enemata of lukewarm water to which has been added a little salt, or of soapsuds, or, still better, by the administration of a ninety-per-cent. glycerin suppository. These measures to secure action of the bowels do not require repetition oftener than once in three days.

(*b*) The expectant symptomatic or so-called rational plan.—This is the method in general use and available for cases of average severity and for those presenting complications. This plan, like the expectant plan, makes no attempt to shorten the duration of the attack or to modify its course as determined by the intensity of the infection and the reaction of the organism to that infection. In the absence of serious symptoms or complications, it is practically the same as the expectant plan.

An effort is made, however, by the administration of quinine in small so-called tonic doses, the mineral acids, turpentine, nitrate of silver, or other medicinal agents from the beginning, to modify favorably the nervous system, the digestive apparatus, or the mucous surfaces, as the case may be, with a view of controlling the severity of the symptoms of the disease and diminishing the danger of the occurrence of complications. Whether or not these effects are, as a rule, actually accomplished, it is not easy to determine. The extreme variation in the intensity of the attack in different cases must put us on our guard against ascribing to therapeutic measures results which ought rather to be attributed to the natural history of the disease in any particular case or series of cases.

Under this plan of treatment, reasonable efforts are made to combat the more serious symptoms of the disease and the complications from the time of their appearance, as well as to anticipate their development; and it is here that we may most advantageously discuss the management of the various symptoms and complications.

The headache of the early days of the attack generally requires no special treatment: it subsides spontaneously between the end of the first and the middle of the second week. Absolute quiet, exclusion of light, local applications, sometimes cold, sometimes warm, constitute, as a rule, all that is necessary to control it. Small doses of antipyrin, gr. i–iii according to the age of the child, or of acetanilide, gr. $\frac{1}{3}$–i, or of phenacetin, gr. $\frac{1}{2}$–ii, may be administered for the relief of obstinate and distressing headache. If necessary, these doses must be repeated once or twice at intervals of an hour or an hour and a half; but the repetition will rarely be required. My preference is for antipyrin, both for the relief of headache and other nervous symptoms and for the reduction of the temperature when necessary. It is easier of administration than the others, and may be given by enema or subcutaneously when required. It does not diminish the ability of the blood-corpuscles to carry oxygen, and thus produce cyanosis, as does acetanilide. The theoretical danger that these small doses are likely temporarily to depress the action of the heart may be combated by the simultaneous administration of small amounts of alcohol.

Sleeplessness is sometimes an important symptom in the early stages of the disease. Like the headache, it usually diminishes some time during the course of the second week; on the other hand, it is occasionally persistent and exhausting. During the primary fever sodium bromide, gr. ii–x, and chloral, gr. ½–ii, yield most satisfactory results. These drugs may be used either in combination or separately. Chloral alone in moderate doses is usually adequate to overcome sleeplessness, and its administration has not in my experience been followed by cardiac depression or other unfavorable results. Sulphonal, gr. v–x, will also prove useful in the control of this symptom; finely triturated, it may be administered in milk. If other hypnotics fail, opium will sometimes secure sleep. This drug and its preparations must, however, be regarded as objectionable during the early stages of the disease, on account of its unfavorable influence upon digestion and the secretions, an influence not wholly obviated by the hypodermic use of morphia. In the later period of the disease—that is, during the secondary fever—opium becomes at once the most efficient and safest means of controlling prolonged sleeplessness and excitability. In the later stages of the disease, chloral is, by reason of its depressing influences upon the circulation, even more objectionable than is opium in the earlier stages.

Somnolence, stupor, and delirium are to be treated by stimulants and the abstraction of heat. Among the stimulants, alcohol stands first and almost alone; spirits of chloroform and camphor are of use in emergencies; ether may be administered subcutaneously, or a five-per-cent. solution of camphor in ether. Ammonium carbonate is of inferior value; it is, however, frequently used in the treatment of pulmonary complications. Pure Siberian musk is a powerful stimulant in conditions of nervous depression. Its high cost and the difficulty of obtaining it stand in the way of its general use.

If delirium be marked, or coma threaten, great benefit is often derived from local applications of cold to the head, by means either of the cold douche or of an ice-cap, the hair having been previously cut short. These applications must be transient and not too frequently repeated, otherwise they may produce depression or collapse. At the same time, warm applications to the feet and legs and sinapisms to the præcordia or epigastrium are called for. The tepid or warm bath is often followed by good results; cold baths are to be avoided.

Tremor indicates extreme prostration. Sir William Jenner has called attention to the fact that tremor, out of all proportion to the other signs of nervous prostration, is to be looked upon as a sign of deep ulceration of the intestine. A small deep slough, the separation of which is especially liable to give rise to intestinal hemorrhage or perforation, will often occasion great tremor. Tremor of this kind is to be treated with full doses of alcohol and opium, not only for their general effect upon the nervous system, but also with a view to their local effects in limiting sloughing and ulceration.

The nervous accidents and complications of enteric fever in childhood do not, as a rule, call for special treatment when they appear during the course of the disease; upon the subsidence of the fever, however, they are to be treated in accordance with general rules.

The fact that the nervous phenomena directly due to the typhoid infection often closely simulate meningitis renders the recognition of actual meningeal inflammation occurring as a complication difficult. It is apt to make its appearance late in the course of the fever or during convalescence. When recognized, it calls for prompt measures of treatment. Opium in full doses, caution being had in regard to the age of the patient, the bromides, cold applications to the head, external warmth to the trunk and extremities, even the abstracting of blood by leeches applied behind the ears or to the septum narium, may be indicated.

Dryness of the tongue and accumulation of sordes upon the teeth are to be obviated by the frequent administration of water or of pieces of ice allowed to dissolve in the mouth. The mouth should be frequently rinsed or washed with pure water or water containing small amounts of claret, tincture of myrrh, or listerine. Saturated solution of boric acid may occasionally be used. Fissures about the nostrils and upon the lips may be touched with an ointment containing twenty grains of boric acid to the ounce.

Constipation when slight requires no treatment beyond an occasional administration of small doses of calomel or castor oil or the juice of an orange. Constipation may, however, be due to the torpidity of the large intestine, the fæcal matter accumulating and the stools being hard and dry. These conditions may set up a sort of secondary diarrhœa, due to irritation of the lower bowel, attended with a feeling of local distress and tenesmus unusual in enteric fever, and are promptly relieved by the removal of the cause. Prolonged constipation is by no means to be taken as indicating moderate intestinal lesions; on the contrary, deep ulceration of a single Peyer's patch is not only occasionally associated with constipation, but by its paralyzing influence upon the intestine it may give rise to constipation. Aperients administered by the mouth are therefore after the middle of the second week to be shunned, lest by inducing active peristalsis they forcibly detach a deep slough, or otherwise mechanically give rise to perforation, where the slough extends to or implicates the serous coat of the intestine. Large enemata are also attended with danger, arising from their liability to set up energetic peristaltic movements which may extend to the lower part of the ileum. The systematic administration of laxative doses of calomel (*vide supra*) every second day during the first week may be depended upon to obviate to a great extent excessive intestinal disturbances, whether of the nature of constipation or of diarrhœa.

Diarrhœa, so long as the stools are of moderate amount and do not exceed in number three or four in the course of twenty-four hours, does not call for special treatment: if, however, the passages are copious or frequent, it becomes necessary to control them. When diarrhœa is due to errors in diet, such as the use of improper food or excessive amounts of food, particularly milk and the strong animal broths, it usually abates upon the substitution of a more suitable dietary. Diarrhœa may also arise in consequence of the patient's drinking excessive amounts of fluid, which pass through the bowel without being absorbed and stimulate the secretions of the intestinal mucous membrane. In the absence of these causes, it is to be attributed to catarrhal inflammation of the intestine. It is then best treated by disinfectant and soothing remedies,—bismuth subcarbonate or subnitrate in large doses, gr. v–xx, every four or six hours. To this may be added, if necessary, opium in doses proportionate to the age of the patient, gr. $\frac{1}{160}-\frac{1}{40}$, or Dover's powder, gr. $\frac{1}{24}-\frac{1}{2}$, or the deodorized tincture of opium, gtt. $\frac{1}{4}$–ii. Equally satisfactory is the combination of minute doses of calomel, gr. $\frac{1}{24}-\frac{1}{16}$, with Dover's powder in the doses above indicated. Opium may be advantageously administered in enemata of starch-water or in suppositories of cacao-butter, and when thus given may be combined with an equal quantity of the tincture or extract of cannabis indica. Formerly astringents, such as alum, plumbic acetate, silver nitrate, tannin, catechu, and kino, were recommended for the control of diarrhœa; more recently, bismuth salicylate, naphthalin, salol, thymol, and resorcin have been employed for the same purpose. All of these remedies are difficult of administration, and most of them impracticable in the therapeutics of childhood.

It is more satisfactory at the bedside to use one or two efficient remedies than to resort to a number of uncertain drugs. In bismuth freely given, or in opium in repeated small doses, either by the mouth or by enemata, or in these two remedies combined, will be found in almost all cases an efficient medication against excessive diarrhœa in enteric fever. If the stools be fetid or highly ammoniacal, small doses of animal charcoal in the form of an impalpable powder may be administered in the broth. Creasote and carbolic acid may also be of service.

Tympany may be due to sloughing or ulceration of the intestine sufficiently deep to cause paralysis, or to general prostration leading to deficient contraction alike of the intestinal walls and of the abdominal muscles, or to the alteration in the character of the digestive fluids, which no longer possess the antiseptic properties of health and therefore permit a speedy decomposition of the intestinal contents. Flatus accumulates in part in the small intestine, chiefly in the colon: it varies from an amount scarcely greater than that of health to great abdominal distention, interfering with the play of the diaphragm, and, by the outward pressure of the accumulated gas within the gut, adding to the danger of perforation. Indications for the treatment of this symptom are twofold: the first have reference to the loss of nerve-energy, and call for an increased stimulation; the second, to the nature of the food and the arrest of the gas-generating decomposition of the intestinal contents. Thus, alcohol is to be given, or, if already employed, the amount is to be increased. Turpentine, camphor, and minute doses of opium may be added to the treatment, and pepsin may be administered with the food. The application of compresses wrung out of cold water or of turpentine stupes is useful, as is also very gentle massage of the abdomen. Enemata of iced water or cold enemata containing turpentine are sometimes followed by good results. The careful introduction of a long rubber catheter will sometimes relieve the distended lower bowel. The puncture of the distended gut with a hypodermic needle is a hazardous procedure. Jacobi[1] states that he has seen fæces entering the abdominal cavity through the openings thus made and fatal peritonitis result. In cases systematically treated by laxative doses of calomel during the early days of the disease, troublesome tympany is not apt to occur.

Intestinal hemorrhage, if it be slight, does not call for other measure of treatment than absolute rest, restriction or complete withholding of food for a time, and moderate doses of opium, either by the mouth or by suppository. Drink must be given in small quantities, repeated at short intervals, or lumps of ice may be held in the mouth and swallowed. The action of the bowels is to be as far as possible controlled. If the blood-loss be profuse, danger to life becomes imminent, and more active measures are to be employed. An ice-bag or bladder filled with broken ice mixed with bran is to be applied to the abdomen. Opium is to be cautiously administered until drowsiness is produced. Fluid extract of ergot may be administered by the mouth; but, if this is not practicable, ergotin may be injected hypodermically at intervals of a half-hour or an hour; if properly administered, there is little danger of the production of abscesses. Gallic acid, turpentine, alum, and lead are recommended. Small enemata of iced water may be administered, and repeated at short intervals. It is not to be hoped that any direct effect upon the intestinal lesions will follow the use of the astringent preparations of iron either by the mouth or by the rectum. The pillows should be removed, and the foot of the bed elevated by blocks.

Upon the occurrence of large intestinal hemorrhage, an area of local dulness may sometimes be detected upon careful percussion in a region of the abdomen previously tympanitic.

---

[1] "Therapeutics of Infancy and Childhood," Archives of Pediatrics, December, 1888.

Peritonitis, whether due to perforation of the intestine or to other causes, calls for the free administration of opium. For a time, at least, no nourishment except concentrated meat juices, a spoonful at a time, or brandy-and-water in not larger amounts, is to be administered. The abdomen may be smeared with a mixture of equal parts of sweet oil, laudanum, and turpentine, and covered with a large, finely-spread flaxseed poultice, or ice-poultices may be applied. If opium be not well borne by the stomach, morphine is to be administered hypodermically. Should the patient's life be prolonged, it is of the utmost importance that the bowels be confined as long as it is possible to keep them so. As a general rule, an action will occur at the end of several days, even under the continued use of opium; if not, at the end of a week small lukewarm enemata or a glycerin suppository may be employed. Palpation of the abdomen is to be practised with great caution, on account of the danger of exciting peritonitis, of causing perforation, or of rupturing the spleen. Peritonitis due to perforation is almost invariably followed by death. Should this accident occur, as it not infrequently does, at the close of defervescence or during convalescence at a period when the appetite is returning, the nutrition improving, and the strength of the patient returning, the propriety of laparotomy should be seriously considered. Perforations are usually single: thus, of Murchison's cases there was only one perforation in twenty-eight instances, two in five, and three in four.

The suprapubic region must be examined by percussion as a matter of routine, and whenever necessary the catheter is to be employed.

Frequent exploration of the chest by the methods of physical diagnosis is necessary. Complications capable of determining a fatal result may often be arrested by the prompt detection and treatment of pulmonary lesions attended by insignificant subjective symptoms. Hypostatic congestion is to be prevented by guarding against the heart-failure to which it is chiefly due. Among the evidences of debility of the heart are extreme pallor, cyanosis of the lips, ears, and finger-tips, mottling of the surface, coldness of the extremities, frequent or irregular pulse, and enfeeblement of the first sound. The patient's position is to be changed from time to time from the dorsal to the lateral decubitus. Strophanthus, convallaria, sparteine sulphate, and caffeine may be administered under such circumstances, according to the special indications of the case. Alcohol is pre-eminently useful: for prompt action, it should be administered in the form of champagne. Digitalis must be used with caution and closely watched. The occasional application of turpentine stupes to the chest is of great advantage.

Bronchitis, if of moderate degree, requires no special treatment; if severe, it must be managed in accordance with general principles.

Bed-sores do not occur in childhood, except as the result of inefficient nursing. They are to be prevented by frequent change of position, removal of pressure by means of cold-water bags or air-cushions, scrupulous cleanliness, and attention to the bed. If erosions by any chance appear, they are to be treated in accordance with general surgical principles.

The fever when of moderate degree, the evening temperature not exceeding 103.5°–104° F., requires no special treatment. This is especially the case if the morning remissions are considerable. A higher temperature than this demands antipyretic treatment.

Antipyretics may be divided into two groups, internal and external.

The internal antipyretics available for this purpose are antipyrin, acetanilide, phenacetin, sodium salicylate, and quinine. Of this group, phenacetin and quinine are difficult of administration, and the use of sodium salicylate is attended with the danger of deranging the stomach. Antipyrin may be administered by the mouth, by the rectum, or hypodermically, in doses varying from grs. iiss–v. These doses may be repeated once or twice at intervals of one or two hours, the effect being carefully watched. The dose of acetanilide is from gr. ¼–iiss: it cannot be administered hypodermically nor in enemata, owing to its imperfect solubility. The administration of small doses of quinine or tepid baths in connection with these antipyretics is often followed by marked reduction of temperature without other unfavorable effects. After some hours the temperature tends to rise again, and the administration of the drugs may be repeated after it reaches its maximum; but the antipyretic course should not be resumed at intervals shorter than twenty-four or forty-eight hours.

External antipyretic treatment consists in the systematic application of cold water or ice, and includes (*a*) cold sponging, (*b*) cold compresses, (*c*) the application of ice, (*d*) the cold pack, (*e*) the cold or gradually-cooled bath, (*f*) cold affusion, and (*g*) iced-water enemata. The therapeutic action of external antipyretics, whether in the form of baths or of other cold-water applications, is greater in children than in adults in proportion as the extent of surface to which the application may be made is relatively greater as compared with the volume to be cooled. The effect of cold applications is more rapid and greater, circulation is more easily disturbed, and the reaction is more tardy. The routine employment of external antipyretics is neither desirable nor safe in enteric fever in childhood, and the extreme method of Brand is inadmissible.

(*a*) Cold Sponging.—The water may be of the temperature of the room, or slightly cooled with ice. A little alcohol or vinegar may be added, or Labarraque's solution. A sponge or wash-cloth may be used, and more or less moderate friction according to the sensation of the patient. In all use of water, great care must be taken to protect the bed. Every part of the body is in turn bared, washed, dried, and again covered. Spongings may be repeated at intervals of two or three hours. In the hands of a skilful nurse they not only add greatly to the comfort of the patient, but also exert a favorable influence upon the nervous system and upon the circulation of the blood, by causing it to flow more freely in the vessels directly under the skin. They lower the temperature only slightly, unless the water be very cold and the spongings frequently repeated.

(*b*) Cold Compresses.—For this purpose three or four thicknesses of old table-linen or towelling which is porous enough to hold a good deal of water are most useful. The compress is wrung out of water of the required temperature and reapplied as it becomes warm. Or two compresses may be used alternately, each being cooled in turn by placing it on a block of ice in a basin or pan at the bedside. Cold compresses are often used for the head, and are commonly very acceptable to patients. They are without appreciable effect upon the general temperature. Very large cold compresses, extending over the entire thorax and abdomen, and frequently renewed, exert a decided effect upon the internal fever. The compresses are sometimes allowed to remain continuously in position, a small quantity of cold water being from time to time added to replace that lost by evaporation.

Leiter's coils, which may be fitted to the head, or applied over the heart or to other regions of the body, in such a manner as to reduce local temperature by means of cold water flowing through them from a reservoir over the bed, exert an influence analogous to but not exactly the same as that of the cold compress.

(*c*) The Application of Ice.—Ice is commonly applied by means of a bladder or gum ice-bag. It must be cracked into pieces the size of a walnut and introduced into the bag with a little water, the bag being about half or two-thirds full. The air is then squeezed out and the stopper adjusted. If the bag be filled, or air enough be left in it to distend it, it will not conform itself to the part to which it is applied. A much more effectual method of applying ice to the abdomen or over the heart is by spreading out a thick layer of finely-cracked ice between the folds of a coarse towel, which is then placed directly over the skin. This method requires constant watching, and is almost sure to wet the bedding. It is not available for prolonged use.

(*d*) The Cold Pack.—A blanket is spread evenly over a couch or bed; over this blanket is laid a coarse sheet wrung lightly out of water of the prescribed temperature and folded once. The patient is lifted upon the bed thus prepared and quickly wrapped in the wet sheet by the attendant in such a manner that it lies as smoothly as possible over every part of the body except the head. If the extremities feel cold before the packing, they must be warmed by friction or else not included in the packing.

So soon as the damp linen is everywhere in contact with the body, the attendant folds the blanket over the patient in the same way, first drawing over and tucking one side smoothly under, and then the other, seeing that the chin is free, and that the blanket is folded evenly, but without tension at the neck. Finally, the long end is drawn down and folded smoothly under the feet.

Three or even four thicknesses of wet sheets spread upon the blanket are necessary to reduce the temperature effectively.

The reduction of temperature from a single pack is usually transient, and repeated packings, even to the number of five or six, are often administered, the rise of temperature being slower after each. When the temperature does not rise above normal, or when shivering takes place, the packing must not again be renewed. When repeated packings are necessary, two couches are used side by side, and the patient is lifted directly from one pack on to the other. The same effect is produced, but less completely, by unfolding the blanket and sprinkling the sheet afresh with cold water.

The patient is allowed to remain in the last pack from half an hour to an hour: at the expiration of this time the skin generally becomes pleasantly warm, and in many cases outbreaks of perspiration take place.

During the packing the pulse is felt at the carotid or temporal artery and the temperature taken in the mouth.

(*e*) The Cold or Gradually-Cooled Bath.—The gradually-cooled bath is generally employed. The quantity of water used should be sufficient wholly to immerse the body of the patient. The tub must stand at the bedside. During the bath the skin should be gently rubbed. The temperature of the water should be about 100° F., or even higher than this, at the first bath. As the patient becomes accustomed to the bath, it is gradually cooled by the addition of cold water to 80° F., or lower. Under no circumstances should it be cooled below 75° F. The average duration of the bath is five minutes. But if shivering or great uneasiness occur, the patient is at once lifted into bed, placed upon a sheet previously made ready, and wiped dry, with brisk rubbing of the extremities and back. The moist sheet is then removed. The patient is covered up, and some hot soup or wine or brandy-and-water administered. The temperature is not always immediately reduced, but, as measured in the rectum, usually falls within an hour from one and a half to four or five degrees. In the course of some hours it rises again, and the bath is then repeated. If cold baths are not well borne, good results in lowering the temperature often follow prolonged lukewarm baths. Sometimes it becomes necessary to repeat the bath once or twice in the course of twenty-four hours. A patient who is quietly sleeping, even if his temperature be high, should not be roused and immediately placed in the bath.

When young children are treated by this method, the temperature of the bath at the beginning should be warm, and a blanket spread over the tub, in which the little patient is gradually lowered into the water.

Not only is the temperature lowered by this means, but also a very favorable influence is exerted upon the state of the nervous system. The intellect clears up, the dulness diminishes.

(*f*) Cold Affusion.—While the patient is in the tub, cold water—60° F.—is thrown by means of a sponge over his head, face, neck, shoulders, and chest. This is repeated once or twice just before he is removed from the bath. It is done rather for the sake of its good effects upon the nervous system in cases of great stupor and other evidences of serious nervous derangement than merely as a means of reducing high temperature. Cold affusions may be practised in bed, the patient being suitably protected by water-proof sheets.

(*g*) Iced-Water Enemata.—Rectal injections of iced water are sometimes followed by a fall of temperature. They are, when carefully administered, rather grateful than otherwise to patients. The quantity of water employed should not in enteric fever exceed three or four fluidounces.

The patient's head and face must always be well bathed with cold water just before and during applications of cold to the general surface of the body. The occurrence of chill or rigor may be delayed by more or less vigorous rubbing or chafing of the body.

The reduction of temperature by one or two degrees and marked tranquillizing influence often follow the administration of a tepid bath—85°–95° F.—of a duration of from five to ten minutes.

The contra-indications to the use of external antipyretics are marked general debility, feebleness of the heart's action, coolness of the surface and extremities, and intestinal hemorrhage.

Chest-complications, even when severe, do not of themselves necessarily contra-indicate the cautious employment of antipyretic treatment when the temperature becomes dangerously high.

Other complications and sequels are to be treated in accordance with general therapeutic indications.

(*c*) *The Specific or Etiological Plan.*—The assumption that enteric fever can be artificially aborted has been made upon insufficient evidence. Undoubted cases of spontaneous abortion of the disease are occasionally observed. The alleged termination of enteric fever in the course of a few days as a result of some special form of treatment demands the incontrovertible evidence of a large series of cases to establish its correctness.

The systematic use of calomel in the early days of the attack, and the administration of antiseptic remedies subsequently, must be regarded as coming under this general plan. The use of carbolic acid, either alone, or in combination with iodine, as suggested by Bartholow, during the whole course of the attack, has in my experience appeared to be followed by excellent results in children as in adults. Clinical researches in the direction of a plan of treatment having for its object some degree of influence upon the fluids of the body such as shall diminish their availability as culture-media for infectious germs are not only justifiable, but in the present state of knowledge are imperatively demanded by the known facts in regard to the causation not only of enteric fever but also of the other infectious diseases. It cannot be said that investigations of this kind have thus far yielded definite results.

4. *The Management of the Patient during Convalescence.*—During the early days of convalescence the temperature remains labile, and abrupt recrudescences of the fever are apt to arise from slight causes. It is therefore important that the patient be cared for assiduously for some time after

convalescence is complete. For at least a week, morning and evening temperature observations should be taken, and during this time the diet is to be restricted to milk, eggs, custards, animal broths or jellies, and the lighter farinaceous foods. At the end of a week, wholesome, easily-digested solid food, including meat, may be resumed; but the effect of such changes of diet upon the temperature and general condition of the patient is to be carefully watched.

If diarrhœa persist, it is to be treated by bismuth and small doses of opium, either alone or in connection with the mineral acids. If there be a tendency to constipation, simple enemata or ninety-per-cent. glycerin suppositories may be employed for its relief. Laxative medicines, with the exception of castor oil in small doses, are inadmissible.

Milk punch, egg-nog, and wine, once or twice a day, may be of service during convalescence; but it is important wholly to dispense with alcohol as early as possible. Quinine, iron, and cod-liver oil may be administered if the convalescence be tardy and anæmia persist.

TYPHUS FEVER.

By ALEXANDER COLLIE, M.D.

Definition.—An acute infectious disease, characterized by an eruption on the skin of ill-defined brownish-red spots (Collie "On Fevers," etc.).

Synonymes.—Typhus (French); Fleckfieber, Flecktyphus, Typhus-exanthematismus (German); Typhus (Eng.); Petechial Fever (general term).

Definition of Children.—Ten and under.

History.—On this point there is very little to be said. The disease is not mentioned by any physician of ancient times; but it has been asserted that the great plague of Athens was typhus, an opinion which could not have been held by any one familiar with typhus and with Thucydides. It was first clearly described by Fracastorius in the fifteenth century; but from this time forward it was confounded with other diseases until, about 1846, Jenner showed it to be quite distinct from enteric fever and consequently from everything else. The last great epidemic of typhus was that which affected the French troops during the Crimean War.

Etiology.—Typhus does not arise primarily among children, but when it appears among adults, children of all ages take it. Its cause is unknown. It has been confidently asserted that it arises from destitution, overcrowding, and insanitary conditions; but of this there is no more evidence than that small-pox or any other infectious disease so arises. Destitution, however, leads to overcrowding, and this increases the probability of the existence of a case from which it may spread.

Small-pox appeared among the soldiers in the Franco-German War. No one, however, supposes that it originated from the aggregation of individual soldiers, but rather that it was carried by some individual soldier from his home and that from him it spread. So with typhus: overcrowding and general unsanitary conditions do not originate it, but they supply the condition for its spread; and the proof of this is obvious; for overcrowding and general unsanitary conditions are permanent, whereas typhus is occasional. It is essentially a disease of temperate climates. It has not been met with in the tropics nor in the tropical parts of America. It has from time to time appeared in most parts of Europe, in the United States, in Canada, in Australia, and in New Zealand; but it is most common in Ireland and among the Irish inhabitants of Great Britain.

It may occur at any season of the year, but it is more common in winter than in summer. One attack protects from another; and, although there are said to be exceptions to this, the writer has never met with one. It is rarely contracted from a single exposure, as is often the case with measles, scarlet fever, and small-pox. On the contrary, a more or less lengthened exposure is necessary, nurses in typhus wards requiring usually an exposure of from six weeks to three months; but to this there are exceptions, especially when the exposure is close, the cubic space small, and the ventilation deficient. Bodily exhaustion, the depressing emotions, etc., are said to predispose to it; but of this there is no evidence. Complete exposure to the air is fatal to the life of the typhus contagium, an excellent proof of which is found in the Irish epidemic of 1848; for those who lay in the open by the roadside and in tumble-down shanties recovered in larger numbers than those who were received into the hospitals. The contagium does not extend far beyond the patient, and therefore, *with due regard to cubic space and ventilation*, other patients may be treated with safety in the same ward, *so long as they remain in bed;* but when they are "up and about" they run the risk of contracting the disease by direct communication with the typhus sick; and this is the practical reason against the treatment of typhus in the general wards of a hospital.

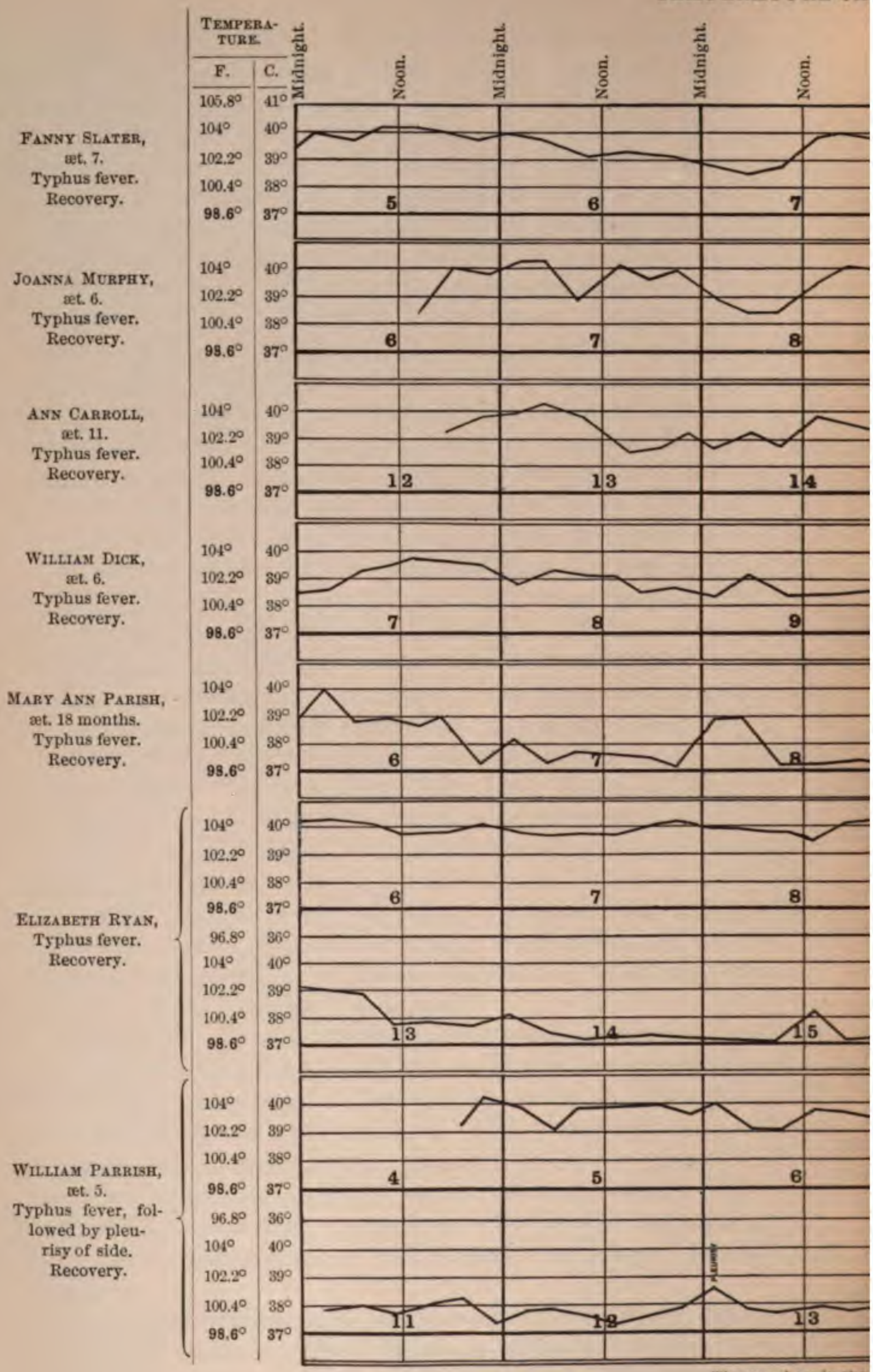
Pathology and Morbid Anatomy.—In the present state of knowledge a pathology of typhus cannot be written. The characteristic feature of the disease is the eruption, which is described under symptomatology.

In adults after death "the blood is more liquid than natural, and coagulates imperfectly. The heart is soft, flabby, and friable, and there is more or less fatty, waxy, or granular degeneration of the muscles. The liver, the spleen, the kidneys, and the mucous membranes are more or less congested; but there are no specific changes in these organs. In the nervous system there are few changes beyond congestion. In former times meningitis was said to be a common occurrence in typhus; but that was at a time (not yet wholly passed by) when most changes characterized by delirium were mistaken for typhus." (Collie "On Fevers.")

Symptoms.—These in children are not usually well marked, although cases are met with presenting all the severity so common among adults. The first symptoms are usually headache and shivering, followed by a feeling of soreness in the limbs and back. The temperature rises, the pulse and respirations are quickened, the tongue is furred, the appetite is diminished, there is some thirst, and a general feeling of weakness like to that produced by a severe cold. Towards the fifth day the characteristic eruption appears, and if the case be well marked it will present the following characters:

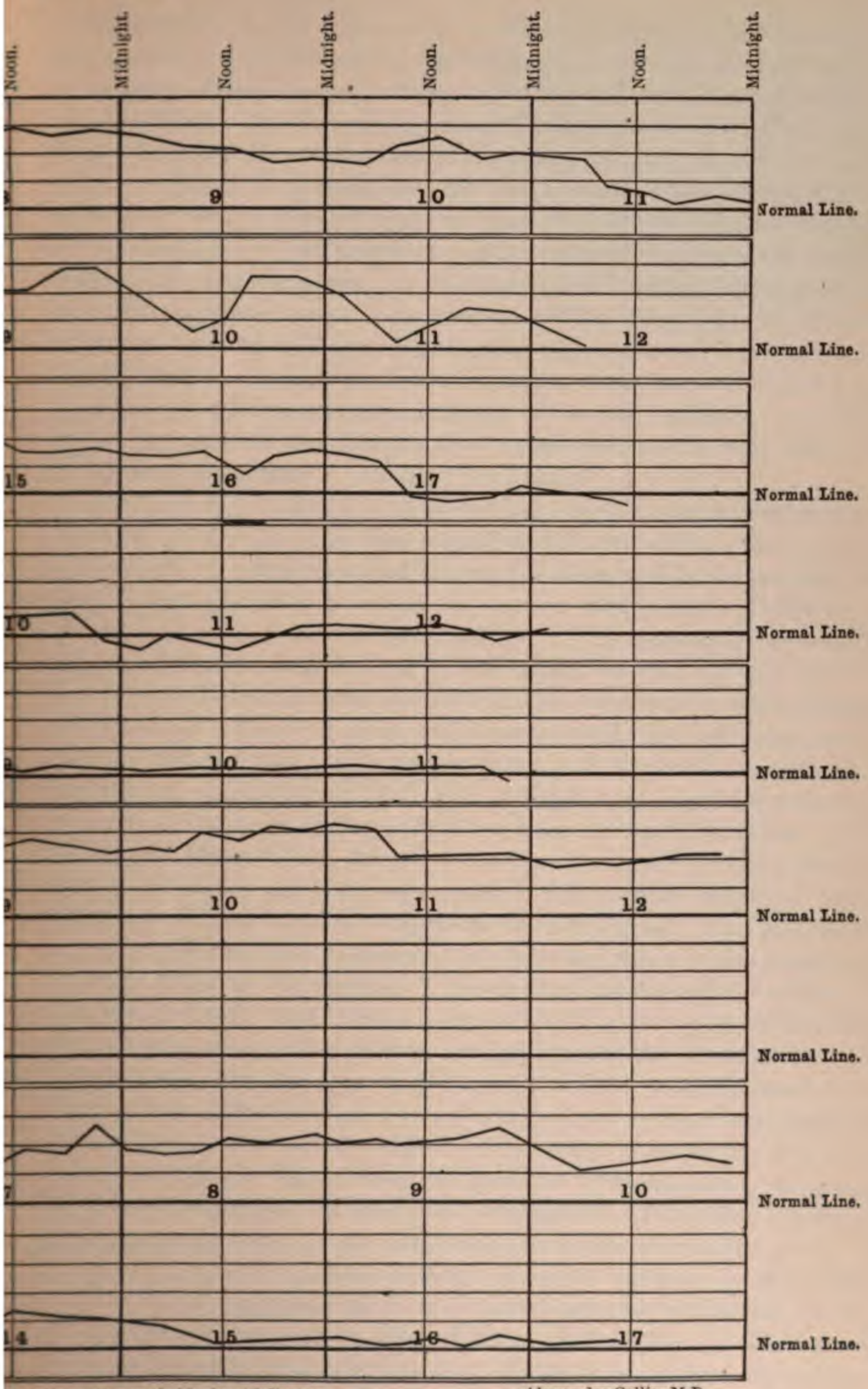
"It consists of three parts: (*a*) rose-colored spots which disappear on pressure, (*b*) dark-red maculæ which are modified by pressure, and (c petechiæ upon which pressure produces no effect, and which persist aft death. It is seen usually first in the subclavicular regions, along the lo

TEMPERATURE-CH



The numbers in the

YPHUS FEVER.



rt indicate the probable day of disease. Alexander Collie, M.D.

border of the pectoralis major, on the wrists, the back of the hands, and the epigastrium, from which parts it gradually covers the body in from one to three days. It is not usually found on the face and neck, but to this there are exceptions. At the very beginning the eruption consists of red spots which disappear on pressure, and if the attack be mild the eruption may consist chiefly of such throughout the whole course of the disease; but in the severer cases, in addition to the red spots there are dark-red maculæ, not unlike 'freckles' in form, connected together by faintly-visible streaks which partially disappear on pressure, to which are added, later on, distinct petechiæ upon which pressure makes no impression. When the eruption is well out, it remains out until it disappears altogether, which it usually does towards, or soon after, the crisis of the fever. It differs from enteric in that it does not come out in successive crops, although at the very beginning spots may be seen to come out and disappear on various parts of the body until the general eruption appears." (Collie "On Fevers.")

The patient's condition in well-marked cases is now pathognomonic. Generally he is drowsy, his face somewhat dull and dusky; his eyes are injected, and his senses somewhat blunted; but his mind in the majority of cases is clear, so that when roused he answers intelligently. Delirium is unusual, and when present is rarely marked. The temperature is usually low; but in cases of some severity it fluctuates between 39° and 40° C. (See Chart.) The pulse varies from 100 to 120; the respirations are slightly accelerated; the tongue is moist and furred,—rarely dry and brown, as is often the case in adults; the abdomen is flat, and the bowels are variable. Throughout, the child sleeps fairly well, although he is sometimes disturbed by dreams. The fever continues about ten days, when a crisis occurs. The temperature falls suddenly, the pulse becomes slower, the eye clear, the expression intelligent, and the tongue clean. The appetite returns at once, and in two or three days the temperature becomes normal. As a rule, there are no complications and no sequelæ, but the writer has met with noma and pleurisy. There are no relapses. The complications met with in adults are pneumonia, pleurisy with effusion, parotid swelling, laryngitis, diphtheria, and nephritis; the sequelæ, deafness and gangrene of the extremities.

Diagnosis.—In the case of children it should be remembered that although they contract typhus it is not usually found among them except in association with cases among adults: so that when a case is met with in a child a well-marked case or cases will probably be found among its adult relations or neighbors. It should be remembered, further, that typhus is usually epidemic either in the district, the town, the village, the street, or the house: so that if one case be found there will probably be others not far off. The diseases with which it is most frequently confounded, in the case of adults, are acute pneumonia and chronic kidney-disease. The former may be excluded by examining the chest, the latter in children can hardly be said to exist. The most important and characteristic feature of typhus is the eruption; but it is unfortunate for the purpose of diagnosis that it is rarely well marked in children. In the case of these, therefore, help must be looked for in the general surroundings of the patient. The history of the case should be carefully considered; the presence of other cases among the adult relations of the child should be inquired after, and the existence of other cases of "fever" in the neighborhood and among the child's friends and relations, because solitary cases are not met with in children, and consequently when they are a case or cases will be met with among their adult relations or neighbors. Therefore an ill-defined "fever" in a child whose adult relations or immediate neighbors were or had been affected by typhus would almost certainly be typhus, assuming of course that there were no symptoms or physical signs pointing to local disease.

Next in importance to these considerations is the general aspect of the patient. The expression is dull and heavy, the eyes are injected, and generally he is somewhat drowsy. With these symptoms there are no local changes to account for them, for example, no chest or abdominal symptoms, and although the patient may complain of headache he does not cry out from acute pains, as in meningitis. If with these symptoms there be a faint mottling of the skin, particularly of the wrists and subclavicular regions (care having been taken to eliminate "flea-bites"), there can be little doubt that the disease is typhus. Enteric fever should not, as a rule, present any difficulty, because the symptoms in this "fever" are a complete contrast to those of typhus. In the former the face is pale, the expression intelligent, and the eye bright.

Measles might in some cases give rise to difficulty, for in these the eye is suffused and injected and the expression drowsy; but the eruption is most abundant and most marked on the face, whereas in typhus the eruption is rarely on the face, and when it is it is but slightly marked and never characteristic. From ordinary small-pox the diagnosis is so obvious that nothing need be said; but in adults the hemorrhagic form of that disease has been mistaken for typhus, and the same mistake is possible in the case of unvaccinated children. This depends mainly on two conditions: first, that in pure hemorrhagic small-pox there are no papules, and, second, that instead of these there is an abundant purpuric eruption. The diagnosis may be made from attention to the following considerations. In pure hemorrhagic small-pox the spots are very large, often like the spots which may be made by throwing ink from a pen on white blotting-paper, and they exist in large numbers closely packed over the lower abdomen; in typhus they are minute, about one-sixteenth to one-twelfth of an inch in diameter, and they are distributed all over the body. In hemorrhagic small-pox the petechial and purpuric spots are almost invariably accompanied by blood-clots in the conjunctivæ, and sometimes by large patches exactly like the bruises produced by blows; in typhus there is nothing of the kind. In hemorrhagic small-pox the eruption is accompanied by hemorrhage from one or all, usually several, of the mucous surfaces; in typhus there is nothing like this. In hemorrhagic small-pox these eruptions are well out on the third or at the latest on the fourth day; in typhus the eruption does not begin to come out until about the fifth day, is not well out till about the seventh day, and does not become petechial till about the tenth day, and in children it is rarely petechial, because of the mildness of the disease and of the occurrence of the crisis at or before that day.

Prognosis.—This is usually favorable; and in the writer's experience it has invariably been so. It may be gathered from the following tables:

LONDON FEVER HOSPITAL.

Age of Patients.	Number of Cases.											Number of Deaths.											Mortality Per Cent.
	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Total.	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Total.	
Under 5 years	9	.	.	.	.	.	.	.	.	.	9	.	.	.	.	.	.	.	.	.	.	.	. .
From 5 to 9 years	30	.	.	.	.	.	.	.	.	.	30	.	.	.	.	.	.	.	.	.	.	.	. .
From 10 to 14 years	56	.	.	.	.	.	.	.	.	.	56	1	.	.	.	.	.	.	.	.	.	1	1.78

(LONDON) EASTERN HOSPITALS (FEVER).

Age of Patients.	Number of Cases.											Number of Deaths.											Mortality Per Cent.
	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Total.	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Total.	
Under 5 years	.	3	2	12	2	3	1	1	.	.	24	.	.	.	.	.	.	.	.	.	.	.	. .
From 5 to 9 years		5	7	24	2	10	5	.	1	.	54	.	.	.	.	.	.	.	.	.	.	.	. .
From 10 to 14 years	.	14	32	38	3	16	6	1	2	1	113	.	.	.	2	.	.	.	.	.	.	2	1.76

These statistics are confirmed by the experience of Dr. McCombie, of the South-Eastern Hospital (fever), who during the years 1880–87 had twenty-six cases of typhus under ten years of age and no deaths. But apparently this experience is not universal; because, during the years 1865–70, of one hundred and forty-eight cases under five treated in the London Fever Hospital there were ten deaths, a mortality of 6.7 per cent., and of six hundred and sixty-four admissions from five to ten there were fifteen deaths, a mortality of 2.2 per cent. The following table gives the experience of the South-Western Hospital (London):

SOUTH-WESTERN HOSPITAL (FEVER).

Age of Patients.	Number of Cases.											Number of Deaths.											Mortality Per Cent.
	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Total.	1871.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Total.	
Under 5 years	.	3	10	12	.	1	5	.	6	.	37	.	.	.	1	.	.	1	.	.	.	2	5.40
From 5 to 9 years	.	13	25	51	.	3	19	.	3	2	116	.	.	.	1	.	.	.	.	.	.	1	0.86
From 10 to 14 years	.	12	45	61	3	2	25	.	7	.	155	.	.	3	2	2	.	2	.	.	.	9	5.80

Unfavorable signs are extensive congestion and inflammation of the lungs, bronchitis, pleurisy, nephritis, laryngitis, and diphtheria; but in children under ten these complications are very rare.

Treatment.—Typhus is for the most part so mild in children that active treatment is rarely required. The child should be placed in a large, well-ventilated room, the temperature of which should be about 50° F. during the acute stage. He should be placed in charge of a trustworthy nurse, who should not, if possible, exceed twenty-five, so that should she contract the disease she may have the best possible chance of recovering from it. He should not be nursed by elderly persons, because of the infectious nature of the disease and of its high mortality among those who have passed thirty. He should be fed on milk, beef tea, chicken broth, bread steeped in the beef tea, and raw eggs, of which he should have as much as he chooses to take. His mouth should be kept scrupulously clean by washing with a soft tooth-brush as often as may be necessary. He should be supplied with as much cold water as he wishes, and this may sometimes be iced. He should have a tepid bath and his linen should be changed at least once daily. No drug beyond a dose of castor oil will be required, and the following is an excellent method of administering it:

"Giving medicine so as to cause the patient as little disgust as possible is no slight art, and worth any trouble to accomplish. In the first place, the glass or vessel in which it is given must be thoroughly clean, not having been used to give a previous dose and remaining unwashed. The medicine should never be poured out within sight or smell of the patient, if it is in any way disagreeable to him; and if he takes anything after it the same should be ready with the dose, that he may take it instantly after the medicine. These remarks apply particularly to the administration of castor oil or cod-liver oil, both of which are generally very obnoxious to adults; at the same time it is easy to give them both in such a manner as to render them very little disgusting. I have often been told by patients that they could not take castor oil, but I never found any one who could not do so perfectly well when prepared as follows. Put a teaspoonful of brandy into a glass and wet the sides well with it, then pour very slowly the oil on to it, taking care to keep it so directly in the middle of the glass that none shall touch the sides; on the top of the oil put another teaspoonful of brandy. Before the patient takes it, give him a little brandy with which thoroughly to rinse his mouth, that the oil if it touches should not stick to it. If he then opens his mouth wide and swallows the oil boldly, taking another sip of brandy immediately after it, he will not taste it." (Veitch.) It may be given in orange-juice in the same manner.

Antipyretics, such as the cold bath, quinine, antipyrin, kairin, and antifebrin, will rarely be required in the case of children, because of the mildness of the disease.

Generally in "fevers" irritating applications, such as hot and mustard poultices, should be avoided, because of the tendency of the skin to slough.

Bedsores should be extremely rare in children, and may almost invariably be prevented. For this purpose the sacral and scapular regions should in severe cases be examined daily, and, if the skin be observed reddening, some spirit lotion should be employed and a water-bed supplied. Alcohol will rarely if ever be required for the typhus; but it may for an accidental complication, such as noma. When there is much restlessness, sponging with cold water will be found very grateful. To favor sleep, the patient should be kept quiet; and if it be necessary to procure it by artificial means, a warm bath followed by a dose of port wine may be given, and, generally, opium should be avoided. In adults retention of urine is an occasional occurrence; and, as it might arise in children, the lower abdomen should be examined daily to see if the bladder be full; and the statement of the nurse that the patient is passing water freely should not be accepted, because when the bladder is full sometimes it runs over, and this dribbling, making a great show, leads the nurse to believe that it is being emptied. When the temperature has fallen, the child may return at once to his ordinary diet.

RELAPSING FEVER.

BY ROLAND G. CURTIN, M.D.

Synonymes.—Febris recurrens, Recurrent typhus, Typhus recurrens, Hunger typhus, Hunger pest, Spirillum fever, etc.

Definition.—Relapsing fever is a contagious, infectious fever of specific origin (a spirillum found in the blood being the supposed cause). Its greatest ravages are among the ill-fed, unclean, and crowded poor. Its advent is remarkable for abruptness, coming on with a pronounced chill (often a rigor) without prodromata; following this, a sudden rise of temperature occurs, running as high as 103° to 105° F. within twenty-four hours. The attack consists of two or more paroxysms, divided by an apyretic interval. The initial paroxysm is generally the longest. This is followed by an abrupt subsidence of the temperature to a subnormal point about the fifth, sixth, or seventh day, with a profuse and exhausting perspiration. The second paroxysm begins about the fourteenth day from the initial chill. It is usually shorter and milder. Headache, sleeplessness, vomiting, jaundice, and conjunctivitis are frequent concomitants. It has no peculiar eruption, and no pathognomonic lesion of the solids. The spleen and liver are enlarged, and tenderness is found over these organs, also at the epigastrium. Death is exceedingly rare, occurring usually in the cases of those who are constitutionally weak or unsound, or from complications.

History.—It is quite evident that relapsing fever is a very ancient disease. It was accurately described by John Rutty in 1739 and 1741. Before this time, writers had noted the principal diagnostic points in the disease. During an epidemic in Dublin, Drs. Barker and Cheyne, in an article entitled "An Account of the Rise, Progress, and Decline of the Fever lately epidemical in Ireland, etc.," 1821, remark, "Certain it is that the fever of 1800 and 1801 very generally terminated on the fifth or seventh day by perspiration; that the disease was then very liable to recur; that the poor were chief sufferers by it."

In 1827 the disease was first definitely isolated from typhus and typhoid fevers.

The name relapsing fever was first applied to this disease by a Scotch physician named Paterson, in 1847.

The first recognized introduction of relapsing fever into the United

States was in 1844, it having been then traced directly to an immigrant-ship. It was afterwards similarly introduced into this country in 1848, and again in 1850, once more in 1863, and lastly in 1869. The disease was in every case imported from Europe. It was during this last epidemic that the writer's opportunity of observing the disease occurred.

Etiology.—Relapsing fever is essentially a contagious disease, as it occurs in epidemics, these epidemics arising from a focus and spreading by contact and fomites.

Murchison and Begbie state as their opinion that relapsing fever is more contagious in childhood than typhus. In Murchison's table (in his work on Continued Fevers), in two thousand one hundred and eleven cases he gives the following data:

Under 5 years	39 cases;
From 5 to 9 years	126 cases;
From 10 to 14 "	234 cases;
From 15 to 19 "	405 cases;
Making under 20 years	804 cases.

Dr. Pepper, in his "System of Medicine," giving the statistics of the Philadelphia epidemic, says that in the eleven hundred and sixty-four cases collected by him, two hundred and twenty-five were under twenty years of age, the youngest being children between two and three years old. It would seem from the foregoing statistics that the disease is very rare in early infancy. In the first year or two, as is shown by these tables, as in many other contagious diseases, perhaps the disease, if present at all, is light and often unrecognized. It is not now believed that scanty food and filth originate the disease, but we do know that they favor its propagation. Employment does not seem to influence the attack. The essential cause seems to be a spirillum in the blood, discovered by Obermeier in 1873. His observations have been confirmed by Koch, Albrecht, Mülhauser, and others, which seems to prove that the disease is due to these filaments. The cause being present, the disease spreads under conditions contributing thereto, especially among the lower classes.

Relapsing fever has not found a home in the United States. It occurs only along the lines of commercial intercourse in slight epidemics, and then only to reappear when again introduced from Europe. The probable cause of this is that our people are better fed and more free from squalor than the poor of some foreign countries, where famine and distress attend the failure of their few and sometimes scanty crops. The last epidemic in Philadelphia, in 1869, followed an epidemic of typhus (1867–1868), which also succeeded another epidemic, one of cerebro-spinal meningitis (in 1866 and 1867). No famine, at home or abroad, preceded this outbreak of relapsing fever.

Pathology.—Cadaveric rigidity in relapsing fever is of short duration. The blood has sometimes been found in an altered condition, the white corpuscles being in excess, the red ones crenated and distorted and not forming in rouleaux. The blood is found either liquid or soft and grumous.

The lungs are congested, and sometimes evidences of the early stage of pneumonia are found. Lobar pneumonia was found in thirty-three per cent. of the autopsies of the Philadelphia epidemic of 1869 and 1870, as reported by Dr. Pepper; the statistics of other writers show an average of about twenty-four per cent.

The muscular tissue of the heart is found in a state of acute fatty degeneration, especially where death occurs in the later stages of the disease.

The brain is usually soft and flabby, but otherwise normal.

Little or no change is found in the spinal cord.

The mucous membrane of the stomach is sometimes congested, and may be even ecchymotic, but it is often normal.

The intestines are usually normal, being in some rare cases dotted with spots of ecchymosis.

The liver is found enlarged, congested, and fatty. The larger ducts are often found in a state of catarrhal inflammation; and Münch has observed the same condition in the minute bile-ducts.

The spleen is enlarged, sometimes enormously (one having been found to weigh sixty-eight ounces), and filled with free granular blood. Occasionally abscesses are found, which may be quite large or perhaps minute. On section of the capsule, the pulp sometimes puffs out, as if under pressure from within. Hemorrhagic infarcts are not rare.

General peritonitis is sometimes found, but it is much more frequently localized over the spleen,—in some instances produced by rupture of the capsule of this organ, from extensive and rapid distention.

Where jaundice has been present during life, the internal tissues are found stained.

Emaciation is not marked, especially where death occurs early in the disease.

Symptomatology.—Relapsing fever is a disease which affects both sexes, and all ages, with perhaps the exception of infants under one year of age. In children, as a rule, the symptoms are less marked, and not unfrequently the disease is of the abortive type; that is, there exists a tendency to run a shorter and milder course than in adults.

The invasion of this fever is usually as follows. The adult patient is at work, or the child is at play, during the day, apparently in full health; an abrupt chill or rigor comes on, and in an hour the temperature may run up to 105° F., with a quick rapid pulse, sharp pain in the head, darting pain in the limbs and loins, no desire for food, and great thirst. The digestive tract especially suffers, as shown by vomiting and anorexia.

The enlargement of the spleen is rapid and often great; the liver is similarly affected, but usually not to so great an extent as the spleen.

Relapses.—The fever usually rises the second time. The paroxysm in each succeeding relapse is usually shorter and less severe; the temperature is less; during the interval there is less perspiration; and exhaustion is not so marked as in the first intermission.

Tongue.—At first a white fur uniformly covers the whole organ, but in two days the appearance is marked; the tips and borders become more deeply red in color than is natural; the color of the centre will be found of a silvery whiteness. Occasionally, later on, it appears as if robbed of its mucous membrane; but this is seldom the case except in the typhoid form. During the intermission it usually becomes clean, but may again return to the condition described in the first paroxysm, and corresponding changes may take place with every exacerbation, continuing to the end.

Appetite.—The appetite is rapidly lost, at the commencement of the disease; and even disgust for food often ensues, with inability to retain it; but during the intermissions it is sometimes continuously craving. This appetite should be gratified with care and judgment.

Vomiting.—In about one-third of the cases there is vomiting; the stomach is irritable during the first paroxysm, and less so in those which follow. The matter first vomited consists of the contents of the stomach, and, after much retching, it is sometimes bilious, though very rarely bloody.

Diarrhœa.—This is quite frequently present late in this disease. It is generally dyspeptic in character. The stools are dark and offensive,—the latter condition being caused by the undigested food. The abdomen is normal in every respect, except in the tenderness found over the region of the epigastrium, spleen, and liver.

Liver.—This organ is enlarged, sometimes greatly so, diminishing again during the interval between the paroxysms. In the negro race the liver is usually more extensively affected.

Jaundice to a greater or less degree was associated with the epidemic of 1869 and 1870 in Philadelphia,—in about from twenty to twenty-five per cent. of the cases, as reported by Dr. Pepper. It is much more frequent in the African race than in the white. This is probably due to obstruction of the bile-ducts from catarrhal inflammation, rather than to suppression, as before stated; for the large and small ducts are often found in a state of inflammation after death.

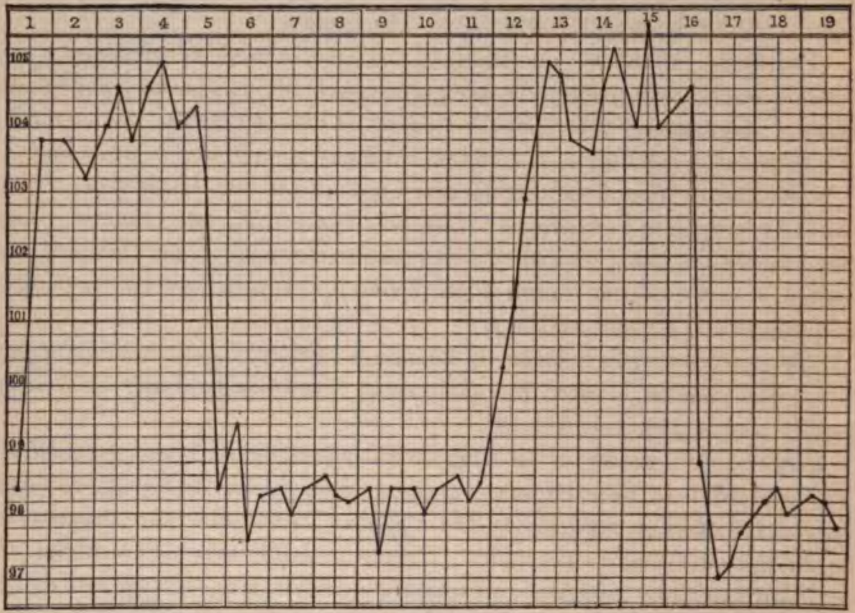
Spleen.—This organ is invariably enlarged, and becomes so rapidly, causing tenderness over the splenic region, with heavy, dull, dragging, aching pain. As in the case of the liver, the enlargement generally subsides perceptibly during the interval between the paroxysms, increasing again during the exacerbation. Sometimes the capsule cannot withstand the strain of the pressure from within, rupture takes place, and abscesses occasionally form, which may open into the peritoneal cavity. Peritonitis, either general or local, may be caused by this accident. The spleen sometimes continues enlarged after the patient recovers.

Epistaxis is quite frequent, but usually much less so than in typhoid fever,—some statistics giving five per cent., others as high as twenty-five per cent., varying with the type of the epidemic.

Heart.—This the central organ of the circulation is greatly crippled, especially in the latter stages of the disease, from impairment of the muscular tissue. The systolic sound is consequently weakened or inaudible, and the diastolic sound is sharpened. During convalescence an anæmic murmur is heard with the systole, together with a hum in the right jugular vein. Dr. Parry noted a blowing murmur at the base of the heart and over the great vessels during both paroxysms. This murmur disappeared during the interval.

Pulse.—The pulse is frequent during paroxysms, subsiding to normal during the intermissions. It is usually strong and full, earlier in the disease; but later it becomes weak and frequently dicrotic.

A CHART FROM MURCHISON "ON RELAPSING FEVER."



The temperature is a little irregular. The patient had passed through a similar attack three months previously.

Pain and Soreness.—Besides the pain in the head hereafter mentioned, the muscles of the limbs and back are found to be sore on pressure, and are sometimes excruciatingly painful. The joints are often painfully affected. In some cases these symptoms continue during the intervals between the paroxysms.

Nervous System.—Neuralgias of spinal origin are often present. Paralyses of muscles or limbs occur, but of short duration, rapid recovery always taking place.

Delirium is remarkably rare, considering the great elevation of temperature.

Headache is quite common, generally frontal, rarely extending to the occipital region.

Temperature.—The temperature is so peculiar that the disease may be usually diagnosticated by a chart. Some exceedingly rare cases of malarial fever may resemble it, but by close observation of two or more charts a positive diagnosis may be made. The rise is abrupt, and as high as from 103° to 105° F. early in the first paroxysm, reaching 106° or 107° on the day of the remission, then falling to a subnormal point to ascend again to 105° or 106°, or even higher, during the second paroxysm. (See temperature-chart.)

Complications.—In the lungs we may have pneumonia, or hypostatic congestion, from which the negro race suffers more than the white. Acute desquamative nephritis is occasionally noted, but it is quite rare. The practitioner should be on the watch for renal symptoms, as this complication is a fruitful source of danger.

Parotitis may occur, but is much more rare in this disease than in typhus or typhoid fever.

Glandular swellings and abscesses are sometimes present, especially in ill-conditioned children.

Some authors state that diarrhœa occurs in one per cent. of the cases. There have been epidemics where diarrhœa has been present in fifty per cent. of the cases.

Hemorrhages from the stomach or bowels, or from both, are not very rare complications in some epidemics.

Types.—A mild form is seen occasionally among children. Among healthy children with favorable surroundings the type is less severe than in the case of adults. It is true of this, as of almost all other contagious diseases, that the later cases in an epidemic are likely to be less severe than the earlier.

The bilious form of relapsing fever (so considered by most authors) is separated by Lebert (Ziemssen, vol. i., Am. ed.), who calls it typhus biliosus, or bilious typhoid fever. It is seen in some virulent epidemics, prevailing especially in the negro race. In Ireland it was called the congestive form. It is the malignant type, and is associated with typhoid symptoms. Jaundice usually occurs at the onset of the disease, associated with hemorrhages from the nose, stomach, and bowels, petechial eruptions, diarrhœa, and a lower temperature than is found in sthenic cases. Death takes place from exhaustion and collapse during the intermission, occasionally associated with coma, and sometimes with uræmic symptoms.

The mortality in relapsing fever varies with the epidemic, class, age, etc.

Death is usually due to complications, sequelæ, or some weakness of the patient, such as organic heart-disease, renal diseases, etc.

Diagnosis.—The principal points in the diagnosis of the disease are:

1. The spirillum, which is to be found in the blood by a microscopical examination,—that is, if the conclusions of Obermeier and others are correct.

2. The temperature. In relapsing fever the rise of temperature is

abrupt and rapid. Lebert says (Ziemssen, vol. i. p. 283, Am. ed.), "The course of relapsing fever is so characteristic, that even in the beginning of an epidemic a mistake can scarcely be made, providing the temperature be noted." Within twenty-four hours the temperature reaches 103° to 105° F. On the fifth or seventh day it falls precipitately to a subnormal point, where it remains until about the fourteenth day, when it rises abruptly again, which is the commencement of the paroxysm. After a few days it drops again as before. (See temperature-chart.)

The splenic, hepatic, and gastric symptoms are marked. The spleen becomes suddenly greatly enlarged, and this enlargement is associated with great tenderness over that region. The liver is also usually enlarged and tender, and the stomach is apt to be very irritable, and sore on pressure. Muscular pains and pain and soreness in the joints are frequently present. Diarrhœa is a common symptom, and ophthalmia is more frequently found than in any other disease likely to be confounded with relapsing fever.

There is no specific eruption in relapsing fever, but sometimes patients are seen with herpes labialis, sudamina, and less commonly slate-colored spots. Occasionally a rubeoloid eruption has been noted, with catarrhal symptoms, much less marked than those which are usually present in measles. The brain-disturbances are mild when compared with those occurring in other fevers with high temperature.

Differential Diagnosis.—*From the Eruptive Fevers.*—There is no specific eruption in relapsing fever, and there are no abdominal symptoms such as are found in typhoid fever. In relapsing fever there is sometimes seen a rubeoloid eruption appearing about the fifth day, at the crisis or subsequently, with mild catarrhal symptoms as compared with measles.

TABLE EXHIBITING THE DIFFERENTIAL DIAGNOSIS OF RELAPSING, REMITTENT, AND YELLOW FEVERS.

	RELAPSING FEVER.	REMITTENT FEVER.	YELLOW FEVER.
Source	From Europe.	From fresh-water lowlands, and stagnant water.	From the Gulf of Mexico.
Contagion	Very great.	Not.	Infection.
Pulse	Very rapid, 140 to 170.	Not rapid.	Rarely over 100.
Temperature	105° to 107° F., first week.	Remittent.	Rarely over 104° F.
Jaundice	The exception, and late.	The exception.	The rule.
Hemorrhage	Rare.	None.	Very frequent.
Frost	No effect.	Arrests.	Arrests.
Quinine	No influence.	Cures.	No effect.
Number of paroxysms	Usually two.	Usually one.	One.
Enlargement of liver and spleen	Both much enlarged.	Both enlarged.	Very little, if any.
Color of liver	Engorged and dark.	Bronzed.	"Butter-yellow."
Fatality	Rare.	Rare.	Very great.

From typhoid or typhus fever it is distinguished by the muscular pains, the remarkable splenic symptoms, and the peculiar temperature.

Prognosis.—In youth, relapsing fever, like typhus, is generally of a mild type; hence the prognosis is more favorable than in the case of adults. The robust, and (when the disease is uncomplicated) almost all patients, recover. In the case of the aged, the intemperate, or those with weak hearts, either from fatty degeneration or from valvular lesion, the prognosis is less favorable.

The mortality ranges from two to twenty-eight per cent., varying with the type of the epidemic. The greatest number die in the epidemics when the malignant form is found. The mortality may be increased by the surroundings of the patient, by his habits, or by the circumstance of race (the negro especially suffering). Early in the epidemic the disease is generally more severe.

When uræmic symptoms are found, with albuminuria and suppressed renal secretion, the case becomes exceedingly grave.

Great care should be taken to avoid violent or too long continued exercise. This precaution may save life which would otherwise be lost.

Treatment.—*Hygiene.*—Prevent contagion by isolation, ventilation, fumigation, and cleanliness of the room and of the patient.

None but the nurse should visit the room, and no one (not even the nurse) should occupy the bed used by the patient. If the patient is an inmate of a home or an asylum, he should be removed to an isolated and quiet location.

A plentiful supply of air should be in the room, but no draughts should be allowed, otherwise there may be danger of pneumonia, and other complications, the result of exposure.

Fumigation with burning sulphur and other disinfectant substances is recommended.

Cleanliness of the room and of the patient should be carefully attended to. The walls should be cleansed by whitewashing or scrubbing, or, if papered, the paper should be removed. The bedclothes, and the clothing of the patient, should be frequently changed, and as soon as they are removed they should be immersed in a carbolic-acid solution (ʒi to Oi), or in a dilute solution of chloride of lime, or in a corrosive-sublimate solution (1 to 1000). The furniture may be washed with a solution of corrosive sublimate of the strength of 1 to 2000.

The body of the patient should be washed with some disinfecting solution. This is for a twofold purpose: first, to cleanse the skin; secondly, to render inert the poisonous exhalations from the body.

This may also be accomplished by carbolic acid, a drachm to a pint of water; or by a solution of the sodium silico-fluoride, two grains to the ounce. There are other washes that will destroy the germ and at the same time not injure the skin.

Nourishment.—Give good, nourishing, and easily-digested food, in moderate quantities, and at suitable intervals. Do not overload or crowd the stomach.

Medication.—All authors of experience unite in the opinion that there is no specific treatment for relapsing fever; that is, there is no remedy that will prevent a recurrence of the paroxysms. Early in the disease, and in mild cases, which is the type usually seen among children, also among the robust at any age, very little medicine is required: a laxative, or an alterative and a laxative combined, as pil. hydrarg. or hydrarg. chloridum mite, followed by a saline. Where vomiting is present (as is frequently the case, and especially where it is quite persistent), small doses of calomel and opium combined will generally allay it.

In severe cases the general principles laid down for low fevers are to be observed, with a few specific suggestions, which are here given for the guidance of the practitioner.

For headache during the paroxysms, apply ice to the head in an ice-bag or bladder, or cold water in an india-rubber bag, or cold-water cloths. At the same time place the feet in hot mustard-water. This treatment should be repeated if necessary. Leeching, and dry or wet cupping, are sometimes of great service, especially in plethoric cases; blistering is better than taking blood for the weak and anæmic. Antipyrin may be of service in such cases.

Sleeplessness is often the cause of great solicitude and discomfort. Potassium bromide is sometimes of value; opium and morphine can be given, but the latter two are inadvisable in cases with kidney-complications. Chloral should be given with caution, especially in the later stages, at which time the muscular weakness is marked, in which the heart may participate. Chloral is not so dangerous when combined with one or more heart tonics, such as digitalis, strophanthus, fluid extract of cactus grandiflora, citrate of caffeine, or caffeine.

Heart-failure occurs either during the stages of depression, attended by exhausting sweats, or late in the disease. Order absolute rest in a recumbent position, and at the same time administer heart tonics (as mentioned in the last paragraph), along with alcohol, carbonate of ammonium, and other stimulants, besides turpentine stupes, stimulating frictions, and dry cupping over heart and lungs.

In high temperature, sponging the surface with cold or tepid water, or with vinegar and water combined, is of great benefit; or large doses of quinine or antifebrin or antipyrin may be given. Phenacetine, from its less depressing influence on the heart, seems better fitted to the later stages of the disease. Avoid aconite, veratrum viride, and all depressing medicines, particularly in the later stage. A nice fever mixture which may be given to children with irritable stomach is the following:

℞ Sodii bicarb., gr. iv;
Creasoti, gtt. ½;
Liq. potass. citrat., ʒivss;
Syr. aurantii cort., q. s. ad f℥i.
M. Sig.—A teaspoonful every two hours for a child four years old.

Jaundice should be treated by the milder remedies usually employed in catarrhal jaundice.

Muscular pains may be palliated by rubbing the seat of the pain with chloroform liniment, alcohol, soap liniment, etc.

For splenic pains, apply a cold-water bottle or bag, or a poultice, over the splenic region, being governed by the sensation of the patient.

Typhoid symptoms are to be treated in the same manner as any disease with the typhoid condition. Support the heart and nervous system with stimulants, either general or diffusible, such as alcohol, digitalis, ammonium carbonate, and tonics.

Vomiting, which has been before alluded to with suggestions for treatment, may be often allayed by plain soda-water in small quantity, or by temporary abstinence from food.

During convalescence, caution the patient and nurse as to the importance of absolute rest, as there is often great danger of heart-failure if this injunction is unheeded.

The treatment for the convalescent stage consists in rest, together with tonics, such as cinchona, iron, and extract of malt, alcohol also if required, and good, nourishing, and easily-digested food. Later on, a change of air may be directed, always cautioning the patient to avoid fatigue. Frictions over the entire surface with alcohol, bay rum, whiskey, or New England rum are grateful as well as strengthening to the patient.

Pneumonia should be treated in the same manner as typhoid pneumonia complicating any fever.

Abscesses should be treated on general principles.

CEREBRO-SPINAL FEVER.

By J. LEWIS SMITH, M.D.

Definition.—Probably a microbic disease. It is manifested chiefly by the occurrence of cerebro-spinal meningitis. Its prominent symptoms are such as meningitis gives rise to,—to wit, fever, headache, tonic contraction of the muscles of the nucha, hyperæsthesia, and neuralgic pains in the trunk and extremities. It is non-contagious, or contagious in a very low degree, and, as with most of the microbic diseases, its victims are chiefly the young. It is ordinarily a primary disease, but it sometimes occurs as a complication of other acute as well as chronic maladies. It begins abruptly or without a premonitory stage, and it is often speedily fatal from the intense hyperæmia of the nervous centres or the severity of the cerebro-spinal meningitis. In other cases, after weeks or months of suffering and progressive loss of flesh and strength, death occurs in a state of extreme prostration. In those who recover, convalescence is protracted and slow.

This disease has been designated by different terms in different countries, as spotted fever, cerebro-spinal fever, malignant purpuric fever, typhus petechialis, typhus syncopalis, and febris nigra, expressive of its constitutional nature. Those who employ such terms regard it as a general or systemic disease with the meningitis as its local manifestation, just as pharyngitis is a local manifestation of scarlet fever or bronchitis of measles or pertussis. This opinion of its nature receives strong support from the clinical fact that, in severe forms of the disease, extravasations of blood occur early under the skin, indicating a profoundly altered state of the blood and systemic infection. The disease has also been designated by terms expressive of its local nature, as epidemic meningitis, epidemic cerebro-spinal meningitis, typhoid meningitis, malignant meningitis. We will treat hereafter of the nature of this malady, and endeavor to justify the opinion which has led to the use of terms that indicate its constitutional character.

My views upon this disease have been carefully elaborated in my various publications upon the subject, both in medical journal literature and in my text-book upon the Diseases of Children. On that account I shall freely quote from these sources in the preparation of this article.

History.—Whether cerebro-spinal fever occurred previous to the present century is uncertain. If it did, it was confounded with other diseases.

Vieussens in 1805 was apparently the first who wrote a clear and unmistakable description of it, designating it "a malignant non-contagious fever." He described an epidemic of it which appeared in Geneva, Switzerland, in a family of three children, of whom two died in twenty-four hours. Two weeks later, four children in another family died of it, after an illness of less than a day, and a young man in another house died with similar symptoms after an equally brief illness, his surface having a deeply congested or violet appearance. In these and subsequent cases the attack began in the latter part of the day, or at night, and was attended by vomiting, violent headache, convulsions, dysphagia, petechiæ, and tonic contraction of the posterior muscles of the neck and trunk, producing retraction of the head and opisthotonus. Thirty-three lost their lives during this epidemic, after a sickness varying from twelve hours to five days. Within the next two years epidemics of cerebro-spinal fever occurred in Bavaria, Holland, Germany, and at about the same time or soon after in parts of England.

The first American cases of the disease, so far as is now known, were at Medfield, Massachusetts, in 1806. From 1806 to 1816 occasional outbreaks of it occurred in England, France, and America, in several localities. It appeared in both Canada and the United States. From 1816 to 1828, so far as is now known, only two epidemics of it occurred, and they were limited to small areas and were of brief duration. The one was at Middletown, Connecticut, and the other at Vesoul, France. In 1828 it occurred in Trumbull County, Ohio, in 1830 at Sunderland, England, and in 1833 at Naples. After the Naples epidemic a respite from the disease appears to have occurred, in both the Eastern and the Western hemisphere, until 1837. In that year it appeared in the south of France, in and around Bayonne, and gradually extended to isolated localities over almost the whole of France. It occurred at this time among troops in their barracks as well as civilians, and in some localities of the troops affected from fifty to seventy-five per cent. died. Even Versailles and Paris did not escape. During the twelve years from 1837 to 1849 France suffered far more than any other country from this disease. It was especially common and fatal among the soldiers in many localities, and at some of the military stations in France several successive epidemics occurred. In the decade from 1839 to 1849, cerebro-spinal fever extended to Naples, the Romagna, Sicily, Gibraltar, Algeria, and various places in Denmark, England, and Ireland.

In 1842 the United States were again visited by cerebro-spinal fever, in localities at a distance from the seaboard, and therefore, apparently, not by communication from Europe. In 1842–43 it occurred in Kentucky, Tennessee, Alabama, Illinois, Mississippi, and Arkansas. From 1848 to 1850 it visited Montgomery in Alabama, Beaver County in Pennsylvania, Cayuga County in New York, and New Orleans in Louisiana. Between 1850 and 1854 there is no record of its occurrence in either hemisphere, but from 1854 to 1860 it ravaged the Scandinavian peninsula and caused more than four thousand deaths.

Since 1860 certain localities in nearly every civilized country have been severely visited by this disease. In all these countries it is justly regarded as one of the most fatal and important of the epidemic maladies.

An interesting fact in regard to these many epidemics on both continents, which have been reported by competent observers, is that they have occurred in isolated localities far apart, and without the least evidence of transportation. Cerebro-spinal fever has not, so far as I am aware, in any instance extended from one locality to an adjacent one in the manner of contagious diseases. The cause of the malady has evidently arisen or been created in the places where the cases have occurred, and is not susceptible of transportation so as to produce the disease elsewhere. Cerebro-spinal fever resembles in this respect the diseases due to marsh miasm.

But since 1860 this disease has appeared in this country in another phase. It has become or is being established, or, to use the phrase commonly employed in medical literature, naturalized, in the cities of the United States. For some years not a week has passed without the report of deaths from this cause in New York, Philadelphia, Jersey City, and Chicago. It is probably already permanently established in Cincinnati, St. Louis, Minneapolis, Newark, and San Francisco, since deaths from it have been reported in these cities during many consecutive weeks.

In New York City prior to 1866 only four deaths occurred from what was perhaps cerebro-spinal fever, since in 1838 two deaths were reported from so-called spotted fever, one in 1850, and one in 1861. What was the nature of this spotted fever is now a matter of conjecture. In 1866, eighteen patients died of cerebro-spinal fever within the city limits, and not a year has passed since, and in the last few years not a week, without deaths from it. From 1866 to 1872 the annual deaths from this disease in New York varied from eighteen to forty-eight. Commencing in December, 1871, and continuing during the first half of 1872, a severe epidemic occurred, producing a large mortality. Many who recovered permanently lost their hearing and some their sight from the attack. In this epidemic the physicians of New York were fully aroused to the importance of the disease, which was causing so much suffering, and which attacked the lower animals, especially the jaded horses of the city car- and stage-lines, not a few of them dropping down in harness, so suddenly did the attacks occur. In 1872, seven hundred and eighty-two deaths, chiefly of children, resulted from cerebro-spinal fever within the city limits. This epidemic appeared to produce a greater dissemination of the disease and more firmly establish it in the city; for since then the annual deaths from it have varied between ninety-seven in 1878 and four hundred and sixty-one in 1881. In Philadelphia cerebro-spinal fever began in 1863, causing forty-nine deaths in that year, and it has never been absent from that city since. Prof. Stillé states that between 1863 and 1882 it has caused two thousand and forty-nine deaths within the city limits. In Philadelphia, as in New York, it has for some years produced a nearly uniform weekly mortality. The prevalence of cerebro-spinal fever in the United States and its probable importance in the future may be inferred from the fact that it has recently occurred also in Cincinnati, Minneapolis, Denver, Norfolk, Boston, Worcester, New Haven, Albany, Syracuse, Auburn, Milwaukee, Wilmington, Detroit, Baltimore, Charleston, Toledo, Mobile, Salt Lake, Grand Rapids, Providence, Chattanooga, Hartford, New Orleans, Fall River, Richmond, Knoxville, and Nashville.

Etiology.—That this disease is produced by a micro-organism is generally believed. Dr. A. Fränkel and other European microscopists have carefully examined the bacteria found in the blood and tissues of those affected by it. At a meeting of the Berlin Medical Society, held February 12, 1883, Herr Leyden showed under the microscope specimens of micrococci found in a case of cerebro-spinal fever. They had an oval shape, were mostly in pairs, and were faintly tremulous. They resembled those found in pneumonia and erysipelas; but Leyden did not think them identical. At the same meeting Herr Baginsky related cases which seemed to show that in some instances the cause of cerebro-spinal fever and that of pneumonia might be identical.[1]

Dr. V. O. Pushkareff, connected with one of the barrack-infirmaries of St. Petersburg, states that in five cases of croupous pneumonia in which cerebro-spinal meningitis occurred as a complication he discovered in the pus taken from the cerebral meninges swarms of micrococci, whose appearance under the microscope seemed identical with that of Friedländer's pneumococcus. They were either isolated or in groups of two, seldom in four, having distinct capsules, and they were absent from the fluid taken from the meninges in simple pneumonia. Pushkareff was able to cultivate the micrococcus taken from the meningeal pus, and the cultivated microbes, like their parents, presented an appearance identical with that of the pneumococcus.[2] Moreover, Eberth, in a case of meningitis following pneumonia, believes that he found the same micrococcus in the lungs and in the liquid exuded from the inflamed pia mater. Fränkel also states that he obtained from the purulent exudation in the pia mater, in a case of meningitis occurring with pneumonia, a microbe resembling that in the pneumonic exudation.[3]

From the investigations of so many competent microscopists, therefore, it appears that the microbe found in the exudate of the meninges in cerebro-spinal fever, and which is supposed to sustain a causal relation to this disease, bears a close resemblance in form to the pneumococcus, if it be not identical with it. But we would infer from the fact that croupous pneumonia is so universal a disease occurring in localities where there is no cerebro-spinal fever, that the cause of the two must be different; or, if there be a form of croupous pneumonia which is produced by the same microbe as that of cerebro-spinal fever, the pneumonia which is universal must have a different origin. The microbic causation of cerebro-spinal fever needs further investigation, which it will doubtless receive, before positive statements can be made.

---

[1] Deutsch. Med. Wochenschr., April 4, 1883.

[2] Ejen. Klin. Gazeta, April 21, 1885.

[3] Deutsch. Med. Wochenschr., Nov. 13, 1886.

Among the conditions which are favorable for the occurrence of cerebro-spinal fever, and may therefore be regarded as predisposing to it, we may mention the winter season. Statistics collected in Europe and the United States show that while one hundred and sixty-six epidemics occurred in the six months commencing with December, only fifty were in the remaining six months of the year. According to the statistics of Prof. Hirsch, which were collected mainly from Central Europe, fifty-seven epidemics were in winter or in winter and spring, eleven in spring, five between spring and autumn, four commenced in autumn and extended into winter, or into winter and the ensuing spring, and six lasted the entire year. I suspect that the opinion expressed by Prof. Hirsch is correct, that the excess of epidemics in the winter months is due mainly to the greater crowding and less ventilation in the domiciles during the cold than during the warm months, especially among European peasantry. In New York City, where the state of the domiciles is about the same the year round, the season appears to exert little influence on the prevalence of the disease.

The fact has repeatedly been observed that anti-hygienic conditions increase the liability to cerebro-spinal fever. Soldiers in barracks and the poor in tenement-houses suffer most severely when the epidemic is prevailing. In New York City the fact is often remarked that multiple cases occur for the most part where obvious insanitary conditions exist, as in apartments which are unusually crowded and filthy, or in tenement-houses around which refuse matter has collected or which have defective drainage. The interesting chart prepared under the direction of Dr. Moreau Morris for the Health Board shows that comparatively few cases occurred in the epidemic of 1872 in those portions of the city where the sanitary conditions were good. Anti-hygienic conditions probably predispose to cerebro-spinal fever, in the same way that they do to other grave epidemic disease, as, for example, to Asiatic cholera, whose ravages are chiefly where hygienic requirements are most neglected. We will presently relate striking examples which show how foul air increases the number and malignancy of cases. Insanitary conditions not only enervate the system and render it more liable to contract any prevailing disease, but probably promote the development and activity of the specific principle.

Is Cerebro-Spinal Fever Contagious?—It is the almost unanimous opinion of those who are most competent to judge from their observations, that it is either not contagious or is contagious in a very slight degree. It is certain that the vast majority of cases occur without the possibility of personal communication. Thus, in the commencement of an epidemic, the first patients are affected here and there, at a distance from each other, often miles apart, and throughout an epidemic usually only one is seized in a family. Children may be around the bedside of the patient, passing in and out of the room without restriction, and yet we can confidently predict that none of them will contract the malady, if there be proper ventilation and cleanliness and none of the conditions of insalubrity exist within or around the domicile. Moreover, when multiple cases occur in a family, the disease begins at such irregular intervals in the different patients that there can be little doubt in most instances that it is not communicated from one to the other, but, like the fevers from marsh miasm, is produced by exposure to the same morbific cause, existing outside the individuals, but within or around the premises. Thus, in the Brown family treated by the late Dr. John G. Sewall,[1] of New York, the first child sickened January 30, and subsequently the remaining five children at intervals respectively of five, seven, eleven, twenty-five, and forty-five days. That so many were affected in one family was attributed by the doctor to the filthy state of the house and the bad plumbing which allowed the free escape of sewer-gas. In my own practice, in the family which suffered the most severely of all, four patients were seized in succession, and yet I could see no evidence of contagiousness. The family occupied a small plot of ground, not more than thirty feet by one hundred, and their occupation was to prepare for the meat-market what is known as head-cheese. They lived on the second floor of the two-story wooden house in which the work was carried on. At the time of the sickness the shop contained four hundred heads of animals from which the meat for the cheese was obtained, and it was evident that decaying animal matter was present. The occupation and surroundings of this family afforded sufficient explanation of the fact that so many were attacked. Two workmen contracted the disease within about one week of each other, and were removed from the house. On January 26, four weeks after the commencement of the malady in the workman who was first attacked, one child sickened with it, and died on February 1. Fifteen days subsequently (February 16) a second child was attacked, and, after a tedious sickness, finally recovered. The long and irregular intervals between these cases indicate that the disease was not contracted by one from the other. The important factor in causing so severe an outbreak of cerebro-spinal fever in this family was probably the miasm produced by such an occupation in the house where the family resided, with neglect of ventilation and cleanliness.

But the strongest evidence that cerebro-spinal fever is either non-contagious or very feebly contagious is afforded by the fact that a large majority of the cases occur singly in families, although there is no isolation of the patients. The following are the statistics relating to this point, in the cases which I have observed since cerebro-spinal fever commenced in New York, in 1871: single cases occurred in seventy families; dual cases occurred in nine families; three cases occurred in one family, and four cases in one family. Intercourse with the sick-room was unrestricted in all these families, so that children frequently went out and in, and sometimes assisted in the nursing.

---

[1] Medical Record, July, 1872.

The most striking example of apparent contagiousness which has come to my knowledge was related by Hirsch, and is quoted by Von Ziemssen. A young man sickened with cerebro-spinal fever on February 8. The woman who nursed him returned to her home in a neighboring village, and there died of the same disease on February 26. To her funeral mourners came from a neighboring township, and after their return home three of them died with the same disease, one within twenty-four hours, another on March 4, and a third on the 7th.

In one instance only in my practice did the facts point to contagiousness. A boy of twelve years died of cerebro-spinal fever and was buried on Saturday or Sunday. On Monday the mother washed the linen and bedclothes of the boy, which had accumulated and were in a very filthy state. Two days subsequently she was attacked, and her infant soon afterwards, both perishing. The state of the bedding and apartments in this house, as seen by myself, was such as would be likely to concentrate and intensify the poison, rendering it peculiarly active, for they were very dirty, and the mother, exhausted by her long and incessant watching and lack of sleep, and depressed by grief, rendered her system more liable to the disease by her self-imposed duties on the day after the funeral. One in her state of mind and body, standing for a considerable part of a day over the bedclothes and bedding of her child, soiled by the excreta, would certainly be in a condition to contract the disease if it were contagious in any, even in the lowest, degree. In the present state of our knowledge, therefore, upon this important subject, the evidence leads us to believe that with proper ventilation and cleanliness, and the suppression of anti-hygienic conditions in an infected domicile, those who are in a good state of body and mind will not contract the disease, but in the opposite conditions it is not improbable that the poison may be so intensified, and the system rendered so liable to receive the prevailing malady, through impairment of the general health and diminished resisting power, that cerebro-spinal fever may, though rarely, be communicated either by the breath of the patient, or by exhalations from his surface or from soiled clothing.

The occurrence of cerebro-spinal fever in certain of the lower animals is a very interesting fact, especially as the question is sometimes asked whether it may not be communicated from them to man. In the epidemic of 1811 in Vermont, according to Dr. Gallop, even the foxes seemed to be affected, so that they were killed in numbers near the dwellings of the inhabitants. Cerebro-spinal fever, previously unknown in New York City, began, as stated above, in 1871, among the horses in the large stables of the city car- and stage-lines, disabling many and proving very fatal, while among the people the epidemic did not properly commence till January, 1872, although a few isolated cases occurred in December of 1871. No evidence exists, so far as I am aware, that the disease was in any instance communicated by these animals to man. Those who had charge of the infected horses, as the veterinary surgeons and stable-men, did not contract the malady, certainly not more frequently than others who were not so exposed. Although we may admit slight contagiousness, there has probably been no well-established example of the transmission of cerebro-spinal fever from animals to man. If transmission ever does occur, it is so rare that practically no account need be made of it.

In some instances we are able to discover an exciting cause. An individual whose system is affected by the epidemic influence may perhaps escape by a quiet and regular mode of life, but if there be any unusual excitement, or if the normal functional activity of the system be seriously disturbed, an outbreak of the malady may occur. Among the exciting causes we may mention overwork and lack of sleep, fatigue, mental excitement, depressing emotions, prolonged abstinence from food followed by overeating, and the use of indigestible and improper food. Thus, in one instance among my cases, a delicate young woman, at the head of one of the departments in a well-known Broadway store, was anxious and excited, and her energies overtaxed, at the annual reopening. Within a day or two subsequently the disease began. Another patient, a boy, was seized after a day of unusual excitement and exposure, having in the mean time bathed in the Hudson when the weather was quite cool. Those children have seemed to me especially liable to be attacked who were subjected to the severe discipline of the public schools, returning home fatigued and hungry and eating heartily at a late hour. In one instance which I observed, a school-girl, ten years of age, returned from school excited and crying because she had failed in her examination and had not been promoted. In the evening, after she had closely studied her lessons, the fever began with violent headache.

Dr. Frothingham[1] writes as follows of the brigade in which cerebro-spinal fever occurred in the Army of the Potomac: "Under General Butterfield, a stern disciplinarian, . . . the men were drilled to the full extent of their powers, often to exhaustion. I did not at the time recognize this as the cause of the disease in question, but I learnt that in the present epidemic in Pennsylvania the attack generally follows unusual exertion and exposure to cold."

Many observers have noticed that bodily fatigue and mental depression and excitement are important factors in causing an attack of cerebro-spinal fever, when this disease is epidemic. Dr. Gallop, in his history of cerebro-spinal fever as it occurred in Vermont in 1811, directs attention to the severity of the cases among the troops under General Dearborn, who were fatigued by marches and greatly dispirited on account of a repulse which they had sustained from the British. In one case, which occurred in my practice, a boy, six years and eleven months of age, was punished at school and came home with cheeks flushed from excitement, the excitement continuing during the ensuing night. On the following day cerebro-spinal fever began with vomiting and chilliness, the attack ending fatally on the seventeenth day. In another case, which was related to me by the mother and the physician, the patient, a bright girl, twelve years of age, of nervous temperament, and forward in her studies, had been much excited in competing for a prize in athletic exercises. In the evening of the same day a violent thunder-storm occurred, and after a severe clap she started from bed, pallid and excited, and expressed the belief that she had been struck by lightning. The disease began immediately after this, and terminated fatally on the fifth day.

---

[1] American Medical Times, April 30, 1864.

Secondary Cerebro-Spinal Fever.—Fagge[1] says, "Several observers have found that during or just after an epidemic of cerebro-spinal fever meningitis has presented itself with unusual frequency as a complication of other acute diseases." He mentions croupous pneumonia, pleurisy, acute tonsillitis, and scarlatinal nephritis as the diseases upon which it is very liable thus to supervene. In this respect cerebro-spinal fever resembles diphtheria and erysipelas, which we know are very liable to occur in those who are suffering from other diseases.

A striking example of cerebro-spinal fever occurring as a complication was recently seen by me in consultation. A child of about ten years with typical typhoid fever had reached about the twelfth day of a mild form of the disease. The initial headache had ceased, there was no delirium, the temperature was but moderately elevated, and no doubt had arisen in the mind of the experienced physician in attendance that the disease, which presented the characteristic signs, would terminate favorably after the usual time. Suddenly violent headache occurred, the temperature rose to 103° or 104° F., and in a few days fatal coma terminated the case. Another disease in which I have seen cerebro-spinal fever occur as a complication is gastro-intestinal catarrh.

Sex.—It is stated by certain writers that more males are affected than females. The statistics of hospitals and camps show this, for men subject to lives of hardship are especially liable to be attacked; but in family practice, in which a large proportion of the patients are children, the number of males and females is about equal. Thus, in one hundred and five cases, occurring chiefly in my practice, but a few of them in the practice of two other physicians of this city, I find that fifty-nine were males and forty-six females. Ninety-one of these were children. In New York City, during the epidemic of 1872, nine hundred and five cases of cerebro-spinal fever were reported to the Health Board between January 1 and November 1, and of these four hundred and eighty-four were males and four hundred and twenty-one females. Dr. Sanderson's statistics of the epidemic in the provinces around the Vistula, the cases being chiefly children, give also but a slight excess of males. Probably, therefore, in the same conditions and occupations of life the sexes are equally liable to contract this malady, and the excess of males is due to the fact that they lead a more irregular life and are more subject to privations and exposures. That soldiers on duty or in barracks have been attacked while families in the vicinity escape, thus increasing the proportion of male cases, must be due to irregularities, hardships, and perhaps the lack of sanitary regulations in their mode of life.

---

[1] Practice of Medicine, vol. i. p. 614.

Age.—My observations lead me to think that the younger the patient the more frequently is cerebro-spinal fever overlooked and some other disease diagnosticated. Nevertheless, all published statistics, so far as I am able to ascertain, show that a large proportion of cases occur under the age of five years, and that a larger proportion of fatal cases are in the first year of life than in any other year. Thus, in New York City the ages of those who died from this disease in 1883 were as follows:

Under 1 year	57	From 20 to 25 years	7
From 1 to 2 years	31	From 25 to 30 "	3
From 2 to 3 "	22	From 30 to 35 "	4
From 3 to 4 "	12	From 35 to 40 "	3
From 4 to 5 "	9	From 40 to 45 "	1
From 5 to 10 "	37	From 45 to 50 "	2
From 10 to 15 "	18	From 50 to 60 "	1
From 15 to 20 "	15	Over 60 years	1

The following are the statistics of the New York Health Board relating to the ages of the cases during the epidemic of 1872:

Under 1 year	125	From 15 to 20 years	54
From 1 to 5 years	336	From 20 to 30 "	79
From 5 to 10 "	204	Over 30 years	71
From 10 to 15 "	106	Total	975

In the cases which occurred in my own practice, and in a few cases in the practice of other physicians added to mine, I find that the ages were as follows:

Under 1 year	16	From 10 to 15 years	10
From 1 to 3 years	27	Over 15 years	15
From 3 to 5 "	25		
From 5 to 10 "	20	Total	118

In my practice, therefore, three-fourths of the cases have been under the age of ten years; and the statistics of epidemics in other localities correspond with mine in giving a large excess of cases in childhood. Thus, Dr. Sanderson, in examining the records of deaths in one epidemic, ascertained that two hundred and eighteen had perished under the age of fourteen years, and only seventeen above that age; and although this does not show the exact ratio of children to adults in the entire number of cases, it is evident that the children were greatly in excess.

The more advanced the age after the tenth year, the less the liability to this malady, so that very few who have passed the thirty-fifth year are attacked, and old age possesses nearly an immunity. In New York City, in which, as we have seen, cerebro-spinal fever has been occurring since 1871, only two cases have come to my knowledge which had passed the fortieth year. The age of one was forty-seven, and of the other sixty-three years. But nearly every year the statistics of the Health Board show that one or two old people have died of this disease.

Not a few cases occur in this city in infants of the age of three or four months. An infant of four months died of cerebro-spinal fever in the New York Infant Asylum, the nature of the disease not being known until it was revealed by the autopsy.

Symptoms.—During the prevalence of cerebro-spinal fever cases now and then occur in which the symptoms are mild and transient and the health is soon fully restored. It seems proper to regard some, at least, of these as genuine but aborted forms of the disease. The following cases which occurred in my practice may be cited as examples:

A boy, eight years of age, previously well, was taken with headache and vomiting, attended by moderate fever, on April 2, 1872. The evacuations were regular, and no local cause of the attack could be discovered. On the following day the symptoms continued, except the vomiting, but he seemed somewhat better. On April 4 the febrile movement was more pronounced, and in the afternoon he was drowsy and had a slight convulsion. The forward movement of the head was apparently somewhat restrained. On the 6th the symptoms had begun to abate, and in about one week from the commencement of the attack his health was fully restored.

A boy, aged six, was well till the second week in May, 1872, when he became feverish and complained of headache. At my first visit, on May 14, he still had headache, with a pulse of 112. The pupils were sensitive to light, but the right pupil was larger than the left. The bromide and iodide of potassium were prescribed, with moderate counter-irritation behind the ears. The headache and febrile movement in a few days abated, the equality of the pupils was restored, and within a little more than one week from the commencement of the disease he fully recovered.

These cases occurred when the epidemic of 1872 was at its height; but if the symptoms are so mild, and the duration of the disease short, as in these two cases, the diagnosis must sometimes be doubtful. Observers in different epidemics report similar cases, and as the symptoms, so far as they appeared in my patients, seemed characteristic, I have not hesitated to regard them as genuine but aborted cases. On such patients the epidemic influence acts so feebly, or their ability to resist it is so great, that they escape with a short and trivial ailment.

Occasionally, also, during the progress of an epidemic, we meet patients who present more or fewer of the characteristic symptoms, but in so mild a form that they are never seriously sick, and never entirely lose their appetite, but the disease, instead of aborting, continues about the usual time.

Thus, on January 4, 1873, I was called to a girl aged thirteen, who had been seized with headache followed by vomiting in the last week in December. During a period of six to eight weeks, or till nearly March 1, she had the following symptoms: daily paroxysmal headache, often most severe in the forenoon; neuralgic pain in the left hypochondrium, and sometimes in the epigastric region; pulse and temperature sometimes nearly normal, and at other times accelerated and elevated, both with daily variations; inequality of the pupils, the right being larger than the left during a portion of the sickness. The patient was never so ill as to keep the bed, usually sitting quietly during the day in a chair or reclining on a lounge, and she never fully lost her appetite. Quinine had no appreciable effect on the fever or paroxysms of pain.

There can, in my opinion, be little doubt that this girl was affected by the epidemic, but so mildly that there was, for a considerable time, much uncertainty in the diagnosis.

Cases like these, in which the disease is so feebly developed that the patient is never seriously sick, though unimportant pathologically, must be recognized in a treatise on cerebro-spinal fever.

Mode of Commencement.—Cerebro-spinal fever rarely begins in the forenoon after a night of quiet and sound sleep. In the cases which I observed in the severe and fatal epidemic of 1872, and in the thirty-six cases of which I have records observed since 1872, the commencement was almost without exception between mid-day and midnight. The fact that this disease does not commence after the repose of night, till several hours of the day have passed, shows the propriety and need of enjoining a quiet and regular mode of life, free from excitement, and with sufficient hours of sleep, during the time in which the epidemic is prevailing.

The commencement is usually without premonitory stage, and sudden, —unlike, therefore, the beginning of other forms of meningitis, which come on gradually and are preceded by symptoms which, if rightly interpreted, direct attention to the cerebro-spinal system. Exceptionally certain premonitions occur for a few hours or days before the advent of the disease, such as languor, chilliness, etc. Mild cases more frequently begin gradually, and with certain premonitions, than severe cases. The ordinary mode of commencement is as follows: the patient is seized with vomiting, headache, and perhaps a chill or chilliness, so that there is a sudden change from perfect health to a state of serious sickness. Rigor or chilliness is a common initial symptom, especially in adult patients. One patient, an adult female, had three or four chills of considerable severity in the commencement of the attack. Children often have clonic convulsions in place of the chill, or immediately after it, partial or general, slight or severe. Stupor more or less profound, or less frequently delirium, succeeds. In the gravest cases semi-coma occurs within the first few hours, in which patients are with difficulty aroused, or profound coma, which, in spite of prompt and appropriate treatment, is speedily fatal. Those thus stricken down by the violent onset of the disease, if aroused to consciousness, complain of severe headache, with or without, or alternating with, equally severe neuralgic pains in some part of the trunk or in one of the extremities. The pain frequently shifts from one part to another. Among the early symptoms of cerebro-spinal fever are those which pertain to the eye. The pupils are dilated, or less frequently contracted, and they respond feebly, or not at all, to light if the attack be severe and dangerous; often they oscillate, and occasionally one is larger than the other. Vomiting with little apparent nausea, and often projectile, is common in the commencement of cerebro-spinal fever. It occurred as an early symptom in fifty-one of fifty-six cases observed by Dr. Sanderson. In ninety-seven cases occurring in New York, most of them observed by myself, but a few of them related to me by the late Dr. John G. Sewall, vomiting occurred as an early symptom in sixty-eight cases. Its absence on the first day was recorded in only three cases, while in the remaining twenty-seven patients the records of the first day make no mention of its presence or absence. It was probably present in most of these twenty-seven cases as one of the first symptoms.

Since the epidemic of 1872, in examining patients, now numbering thirty-six, as has been already stated, I have made careful inquiry in regard to the mode of commencement, and with only two or three exceptions either the previous health had been good, or, if symptoms of ill health antedated the cerebro-spinal fever, they were due to some ailment entirely distinct from this disease. In a boy four and a half years of age, living in Broadway, it was stated to me that the cerebro-spinal fever came on gradually, with pains in the head and elsewhere: this case was mild throughout, and the patient was never in imminent danger. In nearly all the cases, if the patients were at home and under observation, the exact moment of the beginning of the disease could be stated. Thus, a man aged twenty-eight returned from his work at mid-day, April 23, 1883, in good health and cheerful, ate a hearty meal at twelve M., and at one P.M. had a chill, with intense headache and severe vomiting. Minute red points appeared on his face after vomiting, from capillary extravasations. In this case the interesting fact was observed of a cessation of the symptoms, so that on the 24th and 25th, being free from pain, he went to Brooklyn. On the 26th, however, the symptoms returned. He had pains in the head, back, and extremities, and was seriously sick. Occasional remissions, so that very grave symptoms become mild for a time and then return in full severity, as well as distinct intermissions as in this case, have been frequently noticed by observers in different epidemics. A little girl, previously entirely well, was slightly punished on June 11, 1882; immediately she vomited, and seemed quite sick; by kind nursing on the part of the mother she became better, so that on the 12th she had some appetite and went out. On the 13th, cerebro-spinal fever began, with a temperature of 103° F., and its course was tedious. A robust girl, aged thirteen, vivacious and cheerful, went as usual in the morning to one of the public schools, entirely well. Before the school was dismissed she returned home crying, on account of dizziness and violent pain in the top of her head, in her knees, and in the calves of the legs. The case was attended by Prof. Alonzo Clark, Prof. Knapp, and myself, and was fatal after four and a half weeks. A boy, aged ten, returned from another public school in a similar manner, having gone to it in the morning in apparently perfect health.

We may, therefore, summarize as follows the symptoms which commonly attend the commencement of cerebro-spinal fever: violent pain in some part of the head, and sometimes also in the trunk or limbs, vomiting, a chill or chilliness, clonic convulsions, dizziness, dilated, sluggish, or altered pupils, fever of greater or less intensity according to the severity of the attack, heat of head, and in most patients heat of the surface generally. If the disease be of a severe and dangerous type, these symptoms are frequently followed within a few hours by delirium, semi-coma, or coma.

Nervous System.—Since in cerebro-spinal fever extensive and intense inflammation of the cerebral and spinal meninges occurs, with more or less congestion of the brain and spinal cord, lesions which we will consider hereafter, we should expect that this disease would be attended by severe and dangerous symptoms, inasmuch as the cerebro-spinal axis exerts such a controlling influence upon the functions of the body. Also we should expect that the symptoms would vary according to the portion of the meninges which happens to be most severely inflamed. There is, indeed, variation in symptoms according to the extent and intensity of the meningitis and the degree in which the cerebro-spinal axis is congested or implicated, but certain symptoms occur in all or nearly all cases, and, as they are characteristic, they render diagnosis easy.

Pain, already described as an initial symptom, continues during the acute period of the malady. It is ordinarily severe, eliciting moans from the sufferer, but its intensity varies in different patients. Its most frequent seat is the head, and the location of the cephalalgia varies in different patients and in the same patient at different times. One refers it to the top of the head, another to the occiput, and another to the frontal region, and the same patient at different times may complain of all these parts. The pain is described as sharp, lancinating, or boring. It is also common in the neck, especially in the nucha, the epigastrium, the umbilical and lumbar regions, along the spine (rachialgia), and in the extremities, where it shifts from one part to another. It is more common and persistent in the head and along the spine than elsewhere. The patient, if old enough to speak, and not delirious or too stupid, often exclaims, "Oh, my head!" from the intensity of his suffering, but after some moments complains equally of pain in some other part, while perhaps the headache has ceased or is milder. In a few instances the headache is absent, or is slight and transient, while the pain is severe elsewhere. After some days the pain begins to abate, and by the close of the second week is much less pronounced than previously. Vertigo occurs with the headache, so that the patient reels in attempting to stand or walk. I have stated above that vertigo may be a prominent initial symptom, as in the girl of thirteen years who suddenly became sick in the public school where she was attending, and reached her home with difficulty on account of the headache and dizziness. Contributing to the unsteadiness of the muscular movements is a notable loss of flesh and strength, which occurs early and increases.

The state of the patient's mind is interesting. It is well expressed in ordinary cases by the term apathy or indifference, and between this mental state and coma on the one hand, and acute delirium on the other, there is every grade of mental disturbance. Some patients seem totally unconscious of the words or presence of those around them, when it subsequently appears that they understood what was said or done. Delirium is not infrequent, especially in the older children and in adults. Its form is various, most frequently quiet or passive, but occasionally maniacal, so that forcible restraint is required. It sometimes resembles intoxication or hysteria, or it may appear as a simple delusion in regard to certain subjects. Thus, one of my patients, a boy of five years, appeared for the most part rational, protruding his tongue when requested, and ordinarily answering questions correctly, but he constantly mistook his mother—who was always at his bedside—for another person. Severe active delirium is commonly preceded by intense headache. In favorable cases the delirium is usually short, but in the unfavorable it often continues with little abatement till coma supervenes.

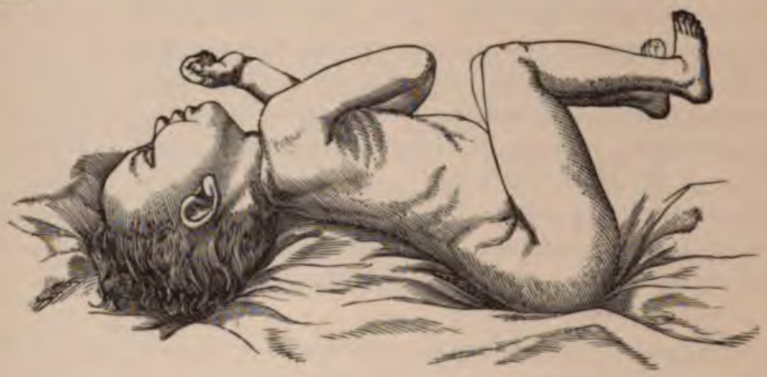
On account of the pain and the disordered state of the mind, patients seldom remain quiet in bed unless they are comatose or the disease be mild or so far advanced that muscular movements are difficult from weakness. In severe cases they are ordinarily quiet for a few moments, as if slumbering, and then, aroused by the pain, they roll or toss from one part of the bed to another. One of my patients, a boy of five years, repeatedly made the entire circuit of the bed during the spells of restlessness. In mild cases, or cases attended by less headache or mental disturbance, patients are quiet, usually with their eyes closed, unless when disturbed.

Hyperæsthesia of the surface is another common symptom. Few patients, not comatose, are free from it during the first weeks, and it materially increases the suffering. Friction upon the surface, and even slight pressure with the fingers upon certain parts, extort cries. Gently separating the eyelids for the purpose of inspecting the eyes, and moving the limbs, or changing the position of the head, evidently increase the suffering, and are resisted. I have sometimes heard such expressions of suffering from slowly introducing the thermometer into the rectum that I was led to believe that the anal and perhaps rectal surfaces were hypersensitive. The hyperæsthesia has diagnostic value, for there is no disease with which cerebro-spinal fever is likely to be confounded in which it is so great. It is due to the spinal meningitis, and is appreciable even in a state of semi-coma. The headache and hyperæsthesia fluctuate greatly in the course of the disease, and the former sometimes recurs at times, especially from mental excitement, or from an afflux of blood to the brain from physical exertion, for months after the health is otherwise fully restored.

Some contraction of certain muscles or groups of muscles is present in all typical cases. In a small proportion of patients it is absent or is not a prominent symptom,—to wit, in those in whom the encephalon is mainly involved, the spinal cord and meninges being but slightly affected or not at all. This contraction is most marked in the muscles of the nucha, causing retraction of the head, but it is also common in the posterior muscles of the trunk, causing opisthotonus, and in less degree in those of the abdomen and lower extremities, and hence the flexed position of the thighs and legs, in which patients obtain most relief. The muscular contraction is not an initial symptom. I have ordinarily first observed it about the close of the second day, but sometimes as early as the close of the first day, and in other instances not till the close of the third day. Attempts to overcome the rigidity, as by bringing forward the head, are very painful, and cause the patient to resist. In young children having a mild form of the fever, with little retraction of the head, the rigidity is sometimes not easily detected. I have been able in such cases to satisfy myself and the friends of its presence by placing the child in an upright position, as on the lap of the mother, and observing the difficulty with which the head is brought forward on presenting to the patient a tumblerful of cold water, which is craved on account of the thirst. The usual position of the patient in bed, in a typical or marked case, is with the head thrown back, the thighs and legs flexed, with or without forward arching of the spine. The muscular contraction and rigidity continue from three to five weeks, more or less, and abate gradually; occasionally they continue much longer. Through the kindness of Dr. Henry Griswold I was allowed to see an infant of seven months in the tenth week of the disease. It was still very fretful, and exhibited decided prominence of the anterior fontanel, probably from intracranial serous effusion and marked rigidity of the muscles of the nucha, with retraction of the head.

Paralysis is another occasional symptom, but complete paralysis of any muscle or group of muscles is less frequent than one would suppose from the nature of the malady. It may occur early, but is sometimes a late symptom. It may be limited to one or two of the limbs, as the legs, or an arm and a leg, or it may be more general. In a case occurring in Roosevelt Hospital, and published in the *New York Medical Record* for October 10, 1878, the patient, a boy of ten years, was unable to move his legs one hour after the commencement of the disease. This sudden development of paraplegia in the commencement of cerebro-spinal fever resembled that of infantile paralysis, and was probably due to the same cause, to wit, active inflammatory congestion of the anterior cornua of the spinal column. The sudden and complete loss of speech which occurs in certain cases, when consciousness is retained and the vocal organs are in their normal state, seems to be due to the fact that the portion of the brain which controls the function of speech is acutely congested, or is the seat of effusion. Thus, in June, 1882, a girl of three years whom I attended lost her speech on the second day of cerebro-spinal fever, and she was unable to articulate even the simplest word for two and a half months. Finally she began to utter slowly and with difficulty the easiest monosyllables; and after the lapse of more than a year her speech was slow and lisping, her hands were tremulous and unsteady, she was easily fatigued, and cried often from oversensitiveness. During the long period of speechlessness she daily made efforts to talk, but without uttering a sound. Strabismus, to which we will allude hereafter in treating of the eye, is a common symptom, either transient or protracted, due to paralysis of certain of the motor muscles of the eye.

Paralysis of more or fewer muscles has been noticed and recorded by many observers in this country and in Europe. Dr. Law observed a patient in the epidemic of 1865, in Dublin, who could move neither arms nor legs, and Wunderlich saw one who had paralysis of both lower extremities and of a considerable part of the trunk. As this symptom is due to the inflammatory process in the cerebro-spinal axis, it usually disappears in a few weeks as the inflammation abates and absorption of the inflammatory products occurs; but it may be more protracted. In Wunderlich's case there was only partial recovery from the paralysis after the lapse of five months.



Clonic convulsions have already been alluded to among the early symptoms of the attack. They indicate a grave form of the disease, and are not infrequent in young children, in whom they appear to occur in place of the chill which is common in those of a more advanced age. The eclamptic attack may be short and not repeated, or it may be protracted, or return again and again when the medicines which control it are suspended. Under such circumstances it is likely to end in profound coma, and is, of course, a symptom of great gravity. Thus, an infant of seven months had unilateral eclamptic attacks daily during the first week of the attack. The mother informed me that the convulsions seldom lasted longer than three minutes, and that the intervals between them were short. The child recovered with loss of sight from the cerebro-spinal fever, but still after the lapse of a year, when I examined him, had symptoms which were apparently due to hydrocephalus. Another infant of eleven months had clonic convulsions nearly constantly during the first twenty-four hours, but with occasional brief intermissions. On the following day he was in profound coma, and apparently dying, with a temperature of 105° F. To my astonishment, he gradually emerged from the state of unconsciousness, and after a week was able to sit in his cradle long enough to take drinks.

Occasionally eclampsia does not occur in the first days, but in the second or third week, when it is usually accompanied by an increase of other symptoms, due to a recrudescence of the disease. A female infant, aged eleven months, treated by me in 1882, had been sick one week, when, during an increase in the febrile movement, she had one eclamptic seizure. Her recovery, though slow, was complete. A boy, aged eleven and one-half years, whose attack began with a chill, violent headache, and a febrile movement, and whom I visited frequently, died on the fourth day. Clonic convulsions did not occur in his case until within twenty-four hours of his death, when he had six seizures, which ended in coma.

Though adult patients are much less liable to eclampsia than children, they are not entirely exempt. A male patient, aged twenty-eight years, whom I saw in consultation, had a single clonic convulsion lasting ten to fifteen minutes on the third day of his illness. In five weeks he had fully recovered, except that his headache returned upon any excitement. Even drinking a cup of beer caused it. Clonic convulsions are, however, much less common than tonic muscular contraction and rigidity already alluded to. This occurs to a greater or less extent in nearly all cases, and is a symptom of diagnostic value, the rigidity often extending to the muscles of the extremities. Thus, in a child, aged three years, who had no eclampsia, the tonic contraction of the muscles of the extremities did not relax till after the twelfth day.

Choreic or choreiform movements are occasionally observed. I do not refer to the tremulousness which sometimes occurs from weakness, or as a premonition of eclampsia, but to a movement which has the character of true chorea. An infant, aged ten months, began to have choreic movements during the acute stage of the disease, most marked in the upper extremities, and ceasing in sleep. They continued during the remainder of the life of the child, death occurring ten months subsequently from diphtheria. Rarely a choreiform movement of the eyes is also observed, a lateral movement from right to left, and from left to right. I have seen from recollection two such cases.

Drowsiness, already spoken of, is a common symptom, and it exists in all grades, from slight stupor to profound coma. In some patients it is present from the first hour, while in others it occurs after a period of restlessness or delirium, or it alternates with it. Stupor more or less profound is common after the attack of eclampsia or the chill. That it is a frequent symptom in severe cases receives ready explanation from the state of the brain and its meninges, for the exudation which occurs upon the surface of the brain and the serous effusion within the ventricles are sufficient to cause it, by compressing the cerebral substance. It is surprising in some cases how profound the stupor may be, a state indeed of coma, and yet the patient gradually emerges from it and recovers. In the epidemic of 1872, in New

York City, when the malady was new with us, many physicians predicted certain death, and employed remedies without expectation of any benefit, on account of the apparently hopeless state of patients, who seemed to be in profound coma, and yet not a few of them gradually and fully recovered.

Digestive System.—Vomiting, which is the most prominent symptom referable to the digestive system, has already been mentioned. Occurring early in the disease, it may cease in a few hours, or not till after several days, and often it returns during the periods of recrudescence which are common in the progress of the fever. It occurs with little effort, and without previous nausea, or with little nausea, as is usual when it has a cerebral origin. It does not differ as a symptom from the vomiting which is so common in other forms of meningitis. The substance vomited consists of the ingesta and the secretions, as mucus and bile. Having a similar origin is a sensation of faintness or depression referred to the epigastrium.

The appetite is usually impaired or lost during the active period of the attack, and it is not fully restored till convalescence is well advanced. Occasionally considerable nutriment is taken, and with apparent relish, as by one of my patients, twenty-eight years of age, who always had some appetite. Ordinarily, on account of repeated vomitings, constant febrile movement, impaired appetite and digestion, patients progressively lose flesh and strength, so that in protracted cases emaciation is always a prominent symptom, and is often extreme. Great emaciation and loss of strength, which attend many cases after the lapse of several weeks, greatly diminish the chances of a favorable termination. Thirst, already referred to, and constipation are common in this as in other forms of meningitis, but retraction of the abdomen is not a notable symptom, except in protracted and greatly-wasted cases. The diarrhœa which is occasionally present in cerebro-spinal fever in the summer months must be regarded as a distinct disease and a complication. The tongue and the buccal and faucial surfaces present nothing unusual in their appearance. It is seldom, even in the most protracted and emaciated cases, that the sordes and dry and brownish fur occur which are so common in typhus and typhoid fevers. The tongue is usually moist and but slightly furred.

I have seen in consultation two patients that perished early with inability to swallow as the prominent symptom, attended in both by an abundant secretion upon the faucial surface, without any redness, swelling, or other evidence of inflammation. The early death of these young children, whose ages were ten months and two years, rendered the diagnosis less certain than in most other patients, but the attending physician as well as myself diagnosticated cerebro-spinal fever with suddenly developed paralysis of the muscles of deglutition, so that no nutriment could be taken. If our understanding of these interesting cases is correct, the paralysis was caused by lesion of that portion of the medulla oblongata which controls the function of deglutition, or else by injury of the intracranial portions of the nerves which supply the muscles concerned in this act. The following were the cases in question:

O——, male, two years of age, became feverish and dull, but without vomiting, on October 22, 1882; axillary temperature, 102° F. On the following day inability to swallow occurred, and the muscles of deglutition appeared totally inactive. Death occurred on the third day, suddenly, and apparently easily, as if from arrested function of important nerves, especially the pneumogastric. The abundant secretion of thin mucus or transudation of serum covering the faucial surface, and reaccumulating as soon as removed, without any notable change in the appearance of the fauces, was remarkable. The physician in attendance, who for more than thirty years had had a large city practice, had seen no similar case, nor had I at the time.

Soon afterwards the second case occurred. An infant of ten months, without cough or embarrassment of respiration, or faucial redness or swelling, lost the power of deglutition soon after the commencement of the supposed cerebro-spinal fever, so that in the attempts to swallow the drinks entered the larynx, and the secretion or exudation was abundant as in the other case. Death occurred in forty-eight hours. The rectal temperature was only 101° F.

In another case, which was ultimately fatal, and in which the diagnosis of cerebro-spinal fever was certain, a robust girl, aged twelve, suddenly lost the power of deglutition at one time during her sickness, although she was entirely conscious and repeatedly endeavored to swallow. The ability to swallow returned in a few days.

Pulse.—This is usually accelerated, and the more severe and dangerous the attack, the more rapid is the heart's action, except occasionally in the comatose state, when probably, in consequence of compression of the brain from an abundant exudation, the pulse may be subnormal. Thus, in one of my patients, an adult, the pulse fell to 40 per minute, and in two others to between 60 and 70 per minute. With the exception of these three, the pulse in all cases which I have observed, so far as I recollect, has varied from the normal number of beats per minute to such frequency that it was difficult to count it. As death draws near, the pulse ordinarily becomes more frequent and feeble. Intermissions in the pulse do not seem to be as common as in other forms of meningitis, but marked variations in its frequency during different hours of the day, and on consecutive days, constitute a conspicuous symptom. Thus, in a case which was fatal in the fifth week, consecutive enumerations of the pulse, in the acute stage, were as follows: 128, 120, 88, 130, 84, 112.

Temperature.—Some of the older writers, before the days of clinical thermometry, stated that the temperature is not increased. North remarked as follows: "Cases occur, it is true, in which the temperature is increased above the natural standard, but these are rare;" and Foot and Gallop make similar statements. Some recent writers have held the same opinion. Thus, Lidell wrote as follows in a treatise bearing the date of 1873: "Febrile symptoms do not necessarily belong to epidemic cerebro-spinal meningitis as a substantive disease, for it may, and not unfrequently does, occur without exhibiting any such symptoms." We should naturally expect that meningitis, accompanied as it is by active congestion of the brain and spinal cord, would produce more or less fever, and in eighty-six cases which I examined by the thermometer I found elevation of temperature in every case during the acute stage, except in the beginning of the attack in two instances. In a young man, aged twenty-eight years, who had severe headache and seemed seriously sick, the thermometer under the tongue showed no rise of temperature on the first and second days, but on the third day it was at 100° F., and it remained elevated till his death, on the thirteenth day. The second case was that of a young woman whom I saw in consultation, and who at the time of my visit had fever, but had had none previously, according to the statement of the attending physician.

In the eighty-six cases which I examined, the heat of the surface occasionally did not seem above normal to the touch, and now and then the thermometer, applied in the axilla or groin, did not indicate fever, but the rectal temperature was always elevated above that of health after the disease was fully established. The temperature fluctuated from day to day, and in different hours of the same day, but there was no exception after the second day to the rule that it was above the normal during the active stage of the malady. Sometimes the elevation of temperature was slight, as in a female patient, forty-seven years of age, in whom the thermometer showed no elevation of temperature when it was placed in the mouth and axilla, but on introducing it into the rectum it rose to 99½° F.

The highest temperature which I have thus far observed was 107⅖° F., in a child aged two years. This was in the commencement of the attack. Subsequently it fell a little, but rose again on the third day to 107°, when she died. In two other cases the temperature was 106° F. on the first day, and it did not afterwards reach so high an elevation. One of these died on the ninth day, and the other in the ninth week. The next highest temperature was 105⅘° F., also on the first day, in an infant aged eight months, who died on the ninth day. The first and last of these cases occurred in an old wooden tenement-house in the suburbs of the city and upon an elevated outcropping of rock. The highest temperature in any case in New York City which has come to my notice was observed in a male patient, aged twenty-eight years, who had active delirium and died on the fifth day in Roosevelt Hospital. The temperature on the last day, taken four times, was as follows: 102½°, 106¾°, and, when the pulse had become imperceptible, 109° and 107¾° F. Wunderlich has recorded a temperature of 110° F. in one or two cases, but so great an elevation must be very rare, and is, of course, prognostic of an unfavorable ending.

The external temperature undergoes still greater fluctuations than the internal, rising above and falling below the normal standard several times in the course of the same day. Similar fluctuations occur in other forms of meningitis, but they are, according to my experience, less pronounced than in cerebro-spinal fever, especially as I observed them in the epidemic of 1872. Perhaps since that epidemic they have been less marked in the cases occurring in this city. The more grave the attack in those not comatose, the greater these variations. The following is a common example of these sudden thermometric changes, occurring in a child of two years. The internal temperature varied from 101° to $104\frac{4}{5}$° F. as the extremes, while that of the fingers and hands at the first examination was $90\frac{1}{2}$°, at the second 90°, at the third 103°, and at the fourth 83°. Hence at the third examination the temperature of the extremities had risen 13°, so as nearly to equal that of the blood, and at the fourth examination it had fallen 20°. The patient recovered. These great and sudden variations in the pulse, and the internal and external temperature, have considerable diagnostic value in obscure and doubtful cases.

Respiratory System.—This system is not notably involved in ordinary cases. Intermittent, sighing, or irregular respiration appears to be less frequent than in tubercular meningitis, but it does occur. In most patients the respiration is quiet, but somewhat accelerated, and without any marked disturbance in its rhythm. In thirty-one observations in children who had no complication, I found the average respirations 42 per minute, while the average pulse was 137. Therefore the respiration, as compared with the pulse, was proportionately more frequent than in health, due perhaps to the fact that certain muscles concerned in respiration, as the abdominal, are embarrassed in their movements by tonic contraction.

Various observers, in different epidemics, have recorded an unusual prevalence of croupous pneumonia occurring simultaneously with cerebro-spinal fever. Bascome, in his history of epidemics, stated that "epidemic encephalitis and malignant pneumonias prevailed in Germany in the sixteenth century" (Webber). Webber, in his prize essay, describes a variety of cerebro-spinal fever, which he designates pneumonic, in which the cerebro-spinal axis is involved but slightly or not at all, and the brunt of the disease falls upon the respiratory organs. According to him, in certain epidemics the pneumonic form has been common and in others infrequent. This fact is interesting taken in connection with the examination of the microbes of croupous pneumonia and cerebro-spinal fever, as detailed in our remarks under the head of etiology.

Cutaneous Surface.—The features may be pallid, of normal appearance, or flushed, in the first days of the disease, but in advanced cases they are pallid, as is the skin generally. A circumscribed patch of deep congestion often appears, as in sporadic meningitis, upon some part of them, as the forehead, the cheek, or an ear, and after a short time disappears. The hyperæmic streak, the *tache cérébrale* of Trousseau, produced by drawing the finger firmly across the surface, also appears as in other forms of meningitis, if the temperature of the surface be not too much reduced.

The following are the abnormal appearances of the skin most frequently observed: 1. Papilliform elevations, the so-called goose-skin, due to contractions of the muscular fibres of the corium. This is not uncommon in the first weeks. 2. A dusky mottling, also common in the first and second weeks in grave cases, and most marked when the temperature is reduced.

3. Numerous minute red points over a large part of the surface, bluish spots a few lines in diameter, due to extravasation of blood under the cuticle, resembling bruises in appearance, and large patches of the same color, an inch or more in diameter, less common than the others, of irregular shape as well as size, and usually not more than two or three upon a patient. These last resemble bruises, and they may sometimes be such, received during the times of restlessness; but ordinarily extravasations of this kind result entirely from the altered state of the blood. In New York in the epidemic of 1872 they were common, but since this epidemic, in the thirty-six cases which I have observed, I have rarely seen either the reddish points or the extravasations of blood. They were probably common in the epidemics in the first part of this century in this country, since the disease was designated by the name spotted fever by the American physicians who wrote upon it at that time. That they are unusual in the European epidemics at the present time we infer from the fact that Von Ziemssen expresses surprise that the disease should ever have been designated in America by such a title. 4. Herpes. This is common. It sometimes occurs as early as the second or third day, but in other instances not till towards the close of the first week or in the second. The number of herpetic eruptions varies from six or eight to clusters as large as or larger than the hand. This cutaneous disease evidently has a nervous origin, the vesicles occurring in most instances on those parts of the surface which are supplied by branches of the fifth pair of nerves. Its most common seat is upon the lips, but occasionally it appears upon the cheek, upon and around the ears, and upon the scalp. Erythema and roseola, both transient skin-eruptions, occasionally appear, and in one instance in my practice erysipelas occurred. During the first days the skin is frequently dry; afterwards perspirations are not unusual, and free perspirations sometimes occur, especially about the head, face, and neck.

Urinary Organs.—In other forms of meningitis it is well known that the quantity of urine excreted is usually diminished, but in this disease it is normal, and it may be more than normal. Polyuria has been noticed in different cases by various observers. Mosler observed a boy aged seven years who had an excessive secretion of urine, which dated back to an attack of cerebro-spinal fever in his third year. The polyuria is probably due to injury of the nervous centre, since it is established by physiological experiment that irritation of the central end of the vagus, of certain parts of the cerebellum, and of the walls of the fourth ventricle, sometimes produces this effect. The urine occasionally contains a moderate amount of albumen, and in exceptional instances cylindrical casts and blood-corpuscles.

Arthritic inflammation, apparently of a rheumatic character, has been occasionally observed. It is commonly slight, producing merely an œdematous appearance around one or more joints. Thus, in one case which came under my notice, and which was subsequently fatal, the parents, who were poor, and were therefore without medical advice till the case was somewhat advanced, had already diagnosticated rheumatism on account of the puffiness which they had noticed around one of the wrists.

The Special Senses.—Taste and smell are rarely affected, so far as is known, but it is possible that they are sometimes perverted, or even temporarily lost, during the time of greatest stupor. In one case which I saw, the sense of smell was entirely lost in one nostril, and I do not know whether it was ever fully restored.

The affections of the eye and ear are important and of frequent occurrence. Strabismus is common. It may occur at any period of the fever, continuing a few hours or several days, and it may appear and disappear several times before convalescence is established: occasionally it continues several weeks, after which the parallelism of the eyes is gradually and fully restored. In other instances it is permanent.

Changes in the pupils are among the first and most noticeable of the initial symptoms, as I have already stated in describing the mode of commencement. These are dilatation, less frequently contraction, oscillation, inequality of size, feeble response to light, etc. Most patients present one or more of these abnormalities of the pupils, and they continue during the first and second weeks, and gradually abate, if the course of the disease be favorable. Inflammatory hyperæmia of the conjunctiva often occurs. It begins early, and now and then the conjunctivitis is so intense that considerable tumefaction of the lids results, with a free muco-purulent secretion. The false diagnosis has indeed been made of purulent ophthalmia, in cases in which this affection of the lids was early and severe. But such intense inflammation is quite exceptional. More frequently there is a uniform diffused redness of the conjunctiva, not so dusky as in typhus, and the injected vessels cannot be so readily distinguished as in that disease.

In certain cases almost the whole eye (all, indeed, of the important constituents) becomes inflamed; the media grow cloudy, the iris discolored, and the pupils uneven and filled up with fibrinous exudation. The deep structures of the eye cannot, therefore, be readily explored by the ophthalmoscope, but they are observed to be adherent to each other and covered by inflammatory exudation. They present a dusky red or even a dark color, when the inflammation is recent. Exceptionally the cornea ulcerates and the eye bursts, with the loss of more or less of the liquids, and shrinking of the eye. "But ordinarily no ulceration occurs, and, as the patient convalesces, the œdema of the lids, the hyperæmia of the conjunctiva, the cloudiness of the cornea and of the humors, gradually abate, and the exudation in the pupils is absorbed. The iris bulges forward, and the deep tissues of the eye, viewed through the vitreous humor, which before had a dusky red color from hyperæmia, now present a dull white color." The lens itself, at first transparent, after a while becomes cataractous. Sight is lost totally and forever.

If the patient live, the volume of the eye diminishes, as the inflammation abates, to less than the normal size, even when there has been no rupture, and escape of the fluids, and divergent strabismus is likely to occur. Prof. Knapp, whose description of the eye I have for the most part followed, says, "The nature of the eye-affection is a purulent choroiditis, probably metastatic." Fortunately, so general and destructive an inflammation of the eye as has been described above is comparatively rare. On the other hand, conjunctivitis of greater or less severity, and hyperæmia of the optic disk, consequent upon the brain-disease, are not unusual, but they subside leaving the function of the organ unimpaired. "In some cases incurable blindness is noticed under the ophthalmoscope picture of optic nerve atrophy, probably the sequence of choked disk." (Knapp.)

Inflammation of the middle ear, of a mild grade, and subsiding without impairment of hearing, is common.' The membrana tympani, during its continuance, presents a dull-yellowish, and in places a reddish, hue. Occasionally a more severe otitis media occurs, ending in suppuration, perforation of the membrana tympani, and otorrhœa, which ceases after a variable time. But otitis media is not the most severe of the affections of the organs of hearing. Certain patients lose their hearing entirely and never regain it, and that, too, with little otalgia, otorrhœa, or other local symptoms by which so grave a result can be prognosticated. This loss of hearing does not occur at the same period of the disease in all cases. Some of those who become deaf are able to hear as they emerge from the stupor of the disease, but lose this function during convalescence, while the majority are observed to be deaf as soon as the stupor abates and full consciousness returns.

Two important facts have been observed in reference to the loss of hearing in these patients,—to wit, it is bilateral and complete. When first observed it is in some, as stated above, complete, but in others partial, and when partial it gradually increases till after some days or weeks, when it becomes complete. I have the records of ten cases of this loss of hearing, most of them occurring in my own practice in the epidemic of 1872, but a few of them detailed to me by the physicians who observed them in the same epidemic. According to these statistics, about one in every ten patients became deaf, but in the milder form of cerebro-spinal meningitis which has prevailed since 1872 the proportionate number thus affected has been less among my patients, and the same may be said in reference to the loss of sight. One of the ten cases was a young lady, but the rest were children under the age of ten years. Prof. Knapp has examined thirty-one cases. "In all," says he, "the deafness was bilateral, and, with two exceptions of faint perceptions of sound, complete. Among the twenty-nine cases of total deafness, there is only one who seemed to give some evidence of hearing afterwards." The same author has recently informed me that further experience has confirmed his previous statement, that while the blindness produced by cerebro-spinal fever is in the majority of cases monolateral, but one case had come to his notice in which the deafness was on one side only.

One theory attributes the loss of hearing to inflammatory lesions either at the centre of audition, within the brain, or in the course of the auditory nerves before they enter the auditory foramina. The other theory, which is the better established of the two, and must be accepted, attributes the loss of hearing to inflammatory disease of the ear, and especially of the labyrinth.

Symptoms of Endemic or Naturalized Cerebro-Spinal Fever.—The numerous monographs on this disease which have appeared during the last few years relate to its epidemic form, and no published observations, so far as I am aware, describe the character or symptoms which it presents, or the changes which it undergoes, when it occurs as an endemic or naturalized disease. The endemic disease must, of course, be observed in the cities or populous towns, for there is no rural locality, so far as I am aware, in which this disease is permanently established. In New York the naturalized disease appears to be accompanied by a less profound blood-change than occurs in epidemic cases. Although every year seeing a considerable number of cases, I have not in the last ten years seen one with the livid spots upon the surface, due to subcutaneous extravasation of blood, which were so common in the epidemic of 1872, and which have been so common in epidemics both in this country and in Europe as to give rise to the term spotted fever. Occasionally petechiæ occur in severe cases of the naturalized disease.

Nature.—The theory that cerebro-spinal fever is a local disease, occurring epidemically, was commonly held in the first part of this century, but is now discarded. Job Wilson, in 1815, considered it a form of influenza, and could see no utility in drawing a distinction between spotted fever and influenza. We at the present time can see no resemblance between the two, except that both occur as epidemics. The theory that cerebro-spinal fever is a peculiar local disease occurring in epidemics is more plausible than that which holds that it is a form of influenza. Even Niemeyer says that it presents no symptoms except such as are referable to the local affection. But the evidence is strong that cerebro-spinal fever is a constitutional malady with the meningitis as a local manifestation, just like measles with its bronchitis, or scarlet fever with its pharyngitis. The abrupt and severe commencement, unlike that of those forms of meningitis which are known to be strictly local, and the early blood-change, as shown in certain cases by the appearance of the skin and extravasations under it, indicate a general disease. Constitutional diseases having prominent local symptoms and lesions are usually regarded at first as local. It is only as time goes on, and they are more thoroughly studied and understood, and clinical observations multiply, that their constitutional nature is recognized.

The theory that cerebro-spinal fever is a form of typhus once had advocates, but it is now so generally discarded, as untenable and absurd, that it would be a waste of time to consider the facts which differentiate the two maladies. Cerebro-spinal fever should, therefore, be considered as distinct from all other diseases, a malady *sui generis*, and in nosological writings it should be classified with those constitutional maladies which have specific causes.

Although this disease ordinarily occurs in an epidemic form in localities widely separated from one another, and, after continuing a few weeks or months, totally disappears, perhaps never to return, or not till after the lapse of years, nevertheless in localities it becomes established, so that it is proper to describe it as an endemic, a fact to which we have already referred as regards certain American cities. I do not know that it is endemic in any village or rural locality in this country. The large cities, with their promiscuous population, foreign and native, their crowded tenement-houses, and their many sources of insalubrity, furnish in an eminent degree the conditions which are favorable for the development and perpetuation of the specific principle. Those diseases which in the present state of our knowledge we have reason to believe are caused by micro-organisms, we should expect to prevail most where domiciles are crowded and filthy, and systems are enervated by impure air, hardships, and privation. Hence in New York City, in the crowded quarters of the poor, cerebro-spinal fever, like diphtheria, is seldom or never absent.

	Number of Deaths.		Number of Deaths.
1872	782	1880	170
1873	290	1881	461
1874	158	1882	238
1875	146	1883	223
1876	127	1884	210
1877	116	1885	202
1878	97	1886	223
1879	108	1887	203

It is seen that the greatest mortality was in the first year after the introduction of the disease into the city, after which the number of deaths gradually diminished, year by year, till 1878, when the lowest mortality was reached. After 1878 the mortality gradually increased till 1881, in which year the number of deaths was double that of any other year except 1872.

The mortuary reports of Philadelphia likewise show that cerebro-spinal fever has remained in that city since its introduction in 1863, a period of twenty-five years, the annual deaths produced by it varying between 36, the minimum, in 1869 and 1870, and 384, the maximum, in 1864. In Providence, also, as appears from Dr. Snow's reports, cerebro-spinal fever has caused annually more or fewer deaths since 1871. Therefore, we repeat, this fact may be added to the sum of our knowledge of this disease, that once gaining a lodgement, where the conditions are favorable for it, as in a large city, it may become established and remain an indefinite time.

Anatomical Characters.—I have notes of the post-mortem appearances in seventy-six cases, published chiefly in British and American journals: twenty-nine died within the first three days, twenty-eight between the third and twenty-first days, and the duration of the remaining nineteen was unknown. These records furnish the data for the following remarks.

The blood undergoes changes which are due in part to the inflammatory and in part to the constitutional and asthenic nature of the disease. The proportion of fibrin is increased in cases that are not speedily fatal, as it ordinarily is in idiopathic inflammations. Analyses of the blood by Ames, Tourdes, and Maillot show a variable proportion of fibrin from three and four-tenths to more than six parts in one thousand. In sthenic cases accompanied by a pretty general meningitis, cerebral and spinal, there is, after the fever has continued some days, the maximum amount of fibrin, while in the asthenic and suddenly fatal cases, with inflammation slight or in its commencement, the fibrin is but little increased. The most common abnormal appearance of the blood observed at autopsies is a dark color with unusual fluidity and the presence of dark soft clots. Exceptionally bubbles of gas have been observed in the large vessels, and the cavities of the heart. An unusually dark color of the blood, small and soft dark clots, and the presence of gas-bubbles, when only a few hours have elapsed after death, indicate a malignant form of the disease, in which the blood is early and profoundly altered. In certain cases this fluid is not so changed as to attract attention from its appearance. The points or patches of extravasated blood which are observed in and under the skin during life in some patients usually remain in the cadaver. When an incision is made through them the blood is seen to have been extravasated not only in the layers of the skin, but also in the subcutaneous connective tissue. Extravasations of small extent are likewise sometimes observed upon and in thoracic and abdominal organs.

In those who die after a sickness of a few hours or days, namely, in the stage of acute inflammatory congestion, the cranial sinuses are found engorged with blood and containing soft dark clots. The meninges enveloping the brain are also intensely hyperæmic in their entire extent, in most cadavers; but in some cases the hyperæmia is limited to a portion of the meninges, while other portions appear nearly normal. In those cases which end fatally within a few hours, this hyperæmia is ordinarily the only lesion of the meninges; but if the case be more protracted, serum and fibrin are soon exuded from the vessels into the meshes of the pia mater, and underneath this membrane, over the surface of the brain. Pus-cells also occur mixed with the fibrin, sometimes so few that they are discovered only with the microscope, but in other cases in such quantity as to be much in excess of the fibrin and to be readily detected by the naked eye. Pus, which in these cases probably consists of white blood-corpuscles which have escaped with the fibrin from the meningeal vessels, often appears early in the attack. The arachnoid soon loses its transparency and polish, and presents a cloudy appearance over a greater or less extent of its surface. This cloudiness is usually greatest along the course of the vessels in the sulci and depressions, and where the fibrinous exudation is greatest, but it occurs also in places where no such exudation is apparent to the naked eye.

The exudation—serous, fibrinous, and purulent—occurs, as in other forms of meningitis, within the meshes of the pia mater, and underneath this membrane over the surface of the brain. The fibrin is raised from the surface of the brain with the meninges in making the autopsy. It is most abundant in the intergyral spaces, around the course of the vessels, over and around the optic commissure, pons Varolii, cerebellum, and medulla oblongata, and along the Sylvian fissures. It is most abundant in the depressions, where it sometimes has the thickness of one-tenth to one-fourth of an inch, but it often extends over the convolutions so as to conceal them from view.

Most other forms of meningitis have a local cause, and are therefore limited to a small extent of the meninges, as, for example, meningitis from tubercles or caries of the petrous portion of the temporal bone, in both of which it is commonly limited to the base of the brain; or from accidents, when the meningitis commonly occurs upon the side or summit of the brain. The meningitis of cerebro-spinal fever, on the other hand, having a general or constitutional cause, occurs with nearly equal frequency upon all parts of the meningeal surface, except that it is perhaps most severe in the depressions, where the vascular supply is greatest. In cases of great severity the inflammatory exudation, fibrinous or purulent, or both, covers nearly or quite the entire surface of the brain.

In those who die at an early stage of the attack, the vessels of the brain, like those of the meninges, are hyperæmic, so that numerous "puncta vasculosa" appear upon its incised surface. At a later period this hyperæmia, like that of the meninges, may disappear. If there be much effusion of serum within the ventricles, and over the surface of the brain, the convolutions are liable to be flattened, and the pressure may be so great that the amount of blood circulating in the brain is reduced below the normal quantity. Thus, in the case of a child of three years, who lived sixteen days, and was examined after death by Burdon-Sanderson, the ventricles contained a large amount of turbid serum, and the brain-substance was everywhere pale and anæmic from compression.

Cerebral *ramollissement* occurs in certain cases. At one of the examinations in Charity Hospital, the patient having been only three days sick, the brain was found much softened. The dissection was made seven hours after death, so that the softening could not have been the result of decomposition. At one of the post-mortem examinations in Bellevue Hospital, softening of the fornix, corpus callosum, and septum lucidum was observed, and in another, softening in the neighborhood of the subarachnoid space. In a case related by Dr. Moorman,[1] it is stated that portions of the brain, medulla oblongata, and pons Varolii were softened. In a case observed by Dr. Upham, softening of the superior portion of the left cerebral hemisphere had occurred. Occasionally the whole brain is somewhat softened. Burdon-Sanderson, Russell, and Githens each relate such a case. Moreover, the walls of the lateral ventricles are ordinarily more or less softened in fatal cases of cerebro-spinal fever, as they are in other forms of meningitis. In rare instances the brain is œdematous, as in a case published by Dr. Hutchinson.[1] In this case the patient was only four days sick, and the whole brain was œdematous, serum escaping from its incised surface.

---

[1] American Journal of the Medical Sciences, October, 1866.

The ventricles contain liquid, in some patients transparent serum, in others serum turbid and containing flocculi of fibrin, or fibrin with pus. The liquids in the different ventricles, since they intercommunicate, are the same. The choroid plexus is either injected or it is infiltrated with fibrin and pus. With the abatement of the inflammation absorption commences. The serum, from its nature, is readily absorbed, and the pus and fibrin more slowly by fatty degeneration and liquefaction. Occasionally the serum remains, and chronic hydrocephalus results. An infant who contracted the disease at the age of five months, and appeared to be convalescent, had, two months subsequently, great prominence of the anterior fontanel, and other symptoms indicating the presence of a considerable amount of effusion within the cranium. In another case, one year afterwards, examination showed the enlargement of the head and prominence of the fontanel which characterize chronic hydrocephalus. A boy of ten years, treated in Roosevelt Hospital in 1878, died three months after the commencement of cerebro-spinal fever. The records of the autopsy state, "Body a skeleton; brain, dura mater, and pia mater appear normal, except a little thickening of latter at base of brain; ventricles much enlarged and full of clear serum; surface of walls of ventricles appears normal, but is soft; spinal cord and membranes apparently normal; heart, lungs, stomach, and intestines normal; liver congested; kidneys pale." In this case, therefore, all the other lesions of the cerebro-spinal axis, except the serous effusion, had nearly disappeared. No post-mortem examinations, so far as I am aware, have yet revealed the state of the brain and its meninges in those who have had this malady at some former time and have fully recovered, whether there may not be some traces of it which are permanent, as opacity or adhesions.

The remarks made in reference to the cerebral apply, for the most part, also to the spinal meninges. There is at first intense hyperæmia of the membranes, usually over the entire surface of the cord, soon followed by fibrinous, purulent, and serous exudation in the meshes of the pia mater and underneath this membrane. This exudation is sometimes confined to a portion of the meninges, more frequently that covering the posterior than the anterior aspect of the cord, and when it is general it is ordinarily thicker posteriorly than anteriorly. In severe cases nearly or quite the entire spinal pia mater may be infiltrated by inflammatory products. Thus, in the case of an infant that died of cerebro-spinal fever at the age of ten weeks, in the service of Dr. H. D. Chapin, in the out-door department at Bellevue, the entire spinal cord was covered by a fibrino-purulent exudation, except a space about six lines in extent upon the anterior surface.

---

[1] American Journal of the Medical Sciences, July, 1866.

No constant or uniform lesions occur in the organs of the trunk, and those observed are not distinctive of this disease. Hypostatic congestion of the lungs, bronchitis, atelectasis, and broncho-pneumonia are common. Pleuritic, endocardial, and pericardial inflammations have occasionally been observed, but are rare. Effusion of serum, sometimes blood-stained, occasionally occurs in the pleural and other serous cavities. The auricles and ventricles of the heart, as already stated, contain more or less blood, with soft dark clots in the more malignant and rapidly fatal cases, but larger and firmer in those which have been more protracted. The spleen is enlarged in less than half the patients. The absence of uniformity as regards the state of the spleen, the fact that in many it undergoes no appreciable change, is important, since this organ is so generally enlarged and softened in the infectious diseases. The stomach, intestines, and liver are sometimes more or less congested, but in other cases their appearance is normal. The agminate and solitary glands of the intestines have ordinarily been overlooked, but in certain cases they have been found prominent. The kidneys are normal, or they exhibit the lesions of nephritis. In one of eight autopsies made by Prof. Welch acute diffuse nephritis had been present, as shown by the state of the kidneys. In the case of a child of nine years, treated by Dr. F. A. Burrall, in the Presbyterian Hospital, the urine was very albuminous and the kidneys presented a fatty appearance. Anatomical changes in these organs, however, are not common, unless in slight degree, so that in most patients their function is fully and properly performed.

Prognosis.—Cerebro-spinal fever is justly regarded as one of the most dangerous maladies of childhood. It is dreaded not only on account of the great mortality which attends it, but also on account of its protracted course, the suffering which it causes, the possible permanent injury of the important organ which is chiefly involved, and the not infrequent irreparable damage which the eye and ear sustain.

I have the records of the result in fifty-two cases which I attended or saw in consultation in the epidemic of 1872. Of these just one-half recovered. Sixteen of the twenty-six who died were hopelessly comatose within the first seven days, most of them dying within that time, and some even on the first and second days, while others of the sixteen lingered into the second week and died without any sign of returning consciousness. The remaining ten, who subsequently died, but did not become comatose in the first week, were nevertheless seriously sick from the first day, but their symptoms, though severe, were not such as necessarily indicated a fatal result, so that there was some expectation of a favorable ending till near death, which occurred for the most part from asthenia. One succumbed to purpura hæmorrhagica, the hemorrhages occurring from the mucous surfaces. The patient died after a sickness of more than two months, in a state of extreme emaciation and prostration. The twenty-six who recovered convalesced slowly, and usually after many fluctuations. Their highest temperature and most severe and dangerous symptoms occurred in the first week. Most of them were several weeks under observation and treatment before they sufficiently recovered to be out of danger. The statistics of this epidemic therefore show, and the same is true of other epidemics, that the first week is the time of greatest danger, and if no fatal symptoms are developed during this week recovery is probable with proper therapeutic measures and kind, intelligent, and efficient nursing, which is very important.

Since 1872 I have seen a larger number, but have preserved records of thirty-eight cases which I was able to follow to the close. Some were seen in consultation. Of these thirty-eight, twenty recovered and eighteen died. Of the eighteen fatal cases, nine died in the first week, five in the second week, one on the twenty-fifth day, one on the thirty-first day, and one in the sixteenth week. This last patient, a boy of ten years, would, in my opinion, have recovered with better nursing. His death occurred from large bed-sores which extended to the bones, produced by lying a long time in one position on a hard bed, when he was too weak to move, and often with soiled bedclothes underneath him. The remaining case of the eighteen died after a prolonged sickness.

There is probably no disease which falsifies the predictions of the physician more frequently than cerebro-spinal fever. This is due partly to the severity of the cerebral symptoms in the commencement, which, did they occur in other forms of meningitis with which he is more familiar, would justify an unfavorable prognosis, and partly to the remissions and exacerbations, the occurrence alternately of symptoms of apparent convalescence and recrudescence or relapse, which characterize the course of this malady. Grave initial symptoms, which may appear to have a fatal augury, are often followed by such a remission that all danger seems past, and in a few hours later, perhaps, the symptoms are nearly or quite as grave as at first.

Under the age of five years and over that of thirty the prognosis is less favorable than between these ages. An abrupt and violent commencement, profound stupor, convulsions, active delirium, and great elevation of temperature are symptoms which should excite solicitude and render the prognosis guarded. If the temperature remain above 105° F., death is probable, even with moderate stupor. Numerous and large petechial eruptions show a profoundly altered state of the blood, and are therefore a bad prognostic, and so is continued albuminuria, since it shows great blood-change, or nephritis, while other organs than the kidneys are probably also involved. In one case, a boy, whom I examined nearly a year after the cerebro-spinal fever, the kidneys were still affected. He had anasarca of the face and extremities, with albuminuria. Chronic Bright's disease had occurred from the acute nephritis which complicated cerebro-spinal fever. Profound stupor, though a dangerous symptom, is not necessarily fatal so long as the patient can be aroused to partial consciousness and the pupils are responsive to light; so long as it does not pass into actual coma it is less dangerous than active or maniacal delirium, which is likely to eventuate in this coma.

A mild commencement, with general mildness of symptoms, as the ability to comprehend and answer questions, moderate pain and muscular rigidity, some appetite, moderate emaciation, little vomiting, etc., justify a favorable prognosis, but even in such cases it should be guarded till convalescence is fully established.

We may repeat and emphasize the important fact shown by the above statistics, that patients who live till the close of the second week without serious complications will probably recover. The danger after this period is, in most instances, from exhaustion and feeble action of the heart, resulting from the impaired nutrition and the protracted course of the disease.

Complications, which most frequently pertain to the lungs, increase greatly the gravity of many cases and contribute to the fatal ending. The fact that Webber, in his prize essay, describes a variety of cerebro-spinal fever which he designates pneumonic, and that those who make post-mortem examinations find that "œdema, hypostatic congestion of the lungs, bronchitis, atelectasis, and broncho-pneumonia are extremely common lesions in cerebro-spinal meningitis" (Welch), indicates a source of danger in addition to that located in the cerebro-spinal system. One close observer of an epidemic writes, "In all the fatal cases which came under my notice, the most prominent symptoms which preceded death were those which indicate impairment and perversion of the respiratory functions. As the breathing became more hurried and difficult, the general depression became more intense, the pulse became weaker and quicker, and the temperature of the skin more elevated."

Parenchymatous degeneration of the liver and kidneys is another serious complication. The kidneys are probably more frequently, and to a greater extent, diseased than the liver. We have already stated that nephritis was present in one of the eight cases examined by Prof. Welch. In the *Revue Médicale* for June 3, 1882, M. Ernest Gandier published the case of a female who died comatose on the sixth day of cerebro-spinal fever. Examination of the urine had revealed the presence of "retractile albumen of Prof. Bouchard, attributable to renal lesions, and non-retractile albumen, considered as an indication of some general infection of the system." Microscopic examination of the kidneys "showed considerable swelling and granular degeneration of the renal epithelial cells, with effusion of granular matter within the lumina of the tubules." We have seen from the case referred to above that the renal complication may persist and become chronic. Those who fully recover often exhibit symptoms usually of a nervous character, as irritability of disposition, headache, etc., for months or years after convalescence is established.

Diagnosis.—Cerebro-spinal fever, on account of the nature and severity of its symptoms and the suddenness of its onset, may be mistaken for scarlet fever, and *vice versâ*. In one instance, to my knowledge, this mistake was made. High febrile movement, vomiting, convulsions, and stupor are common in the commencement of scarlet fever, and the same symptoms commonly usher in the severer forms of cerebro-spinal fever. It will aid in diagnosis to ascertain whether there be redness of the fauces, for this is present in the commencement of scarlet fever, and a few hours later the characteristic efflorescence appears on the skin.

The diagnosis of cerebro-spinal fever from the common forms of meningitis is ordinarily not difficult, for while in the former the maximum intensity of symptoms occurs in the first days, in the latter there is a gradual and progressive increase of symptoms, from a comparatively mild commencement. Moreover, cases of ordinary or sporadic meningitis occurring at the age when cerebro-spinal fever is most frequent are commonly secondary, being due to tubercles, caries of the petrous portion of the temporal bone, or other lesion, and are therefore preceded and accompanied by symptoms which are directly referable to the primary disease. We have seen how different it is in cerebro-spinal fever, which in most patients begins abruptly in a state of previous good health. Again, in cerebro-spinal fever, after the second or third day, hyperæsthesia, retraction of the head, and other characteristic symptoms occur, which are either not present or are much less pronounced in ordinary meningitis. Some of the milder cases of cerebro-spinal fever might be mistaken for hysteria, but the pain in the head and elsewhere, the muscular rigidity, and especially the occurrence of more or less febrile movement, enable us to make the diagnosis. Continued fever, typhus or typhoid, resembles cerebro-spinal fever in certain particulars, but it lacks the muscular contraction and rigidity which characterize the latter. It does not usually begin so abruptly, with such severe symptoms, especially such severe headache, has less marked fluctuations, and a more definite duration. These facts, in connection with the character of the prevailing epidemics, will enable us to make the diagnosis. In one instance commencing retro-pharyngeal abscess, probably associated with vertebral caries, was at first mistaken by me for cerebro-spinal fever. The patient was an infant, had a temperature of 104° F., stiffness of the neck with some retraction of the head, and cried from pain when the head was brought forward. The speedy occurrence of two large abscesses in other parts of the system, difficult deglutition, and noisy respiration, led to a digital exploration of the fauces, when the abscess was found and opened.

Treatment.—Since in epidemics of cerebro-spinal fever cases are more frequent and severe where anti-hygienic conditions exist, it is evident that measures looking to the removal of such conditions, measures designed to procure pure air in the domicile, wholesome diet, and a quiet and regular mode of life,—in fine, measures designed to produce the highest degree of health,—are of the first importance for the prevention of the disease. Cleanliness of the streets and areas, as well as of the apartments, good sewerage and drainage, the prompt removal of all refuse matter, avoidance of overcrowding,—in a word, the strict observance of sanitary requirements in every particular,—will, there can be little doubt from what we know of the causation and nature of cerebro-spinal fever, diminish the number and severity of the cases. The avoidance of fatigue and overwork and of mental excitement, the use of plain and wholesome diet, sufficient sleep, the utmost regularity in the mode of life, with the least possible exposure to depressing agencies, are the important preventive measures which should be recommended during an epidemic of cerebro-spinal fever.

The enjoining of a quiet and regular mode of life as a preventive measure, during the occurrence of an epidemic of cerebro-spinal fever, is not inconsistent with the theory that the cause is a micro-organism. It is not unreasonable to suppose that the system may be more or less under the influence of the specific principle, and that this principle may obtain lodgement in the blood or tissues without result until some exciting cause occurs which depresses the system and disturbs the functions, when the resisting power fails and cerebro-spinal fever appears; just as those exposed to Asiatic cholera may remain well until some imprudence in the diet or the mode of life causes an outbreak of the malady.

Curative Treatment.—In the commencement of cerebro-spinal fever, intense inflammatory congestion occurs of the cerebral and spinal meninges, and also to a certain extent of the brain and spinal cord. As regards treatment, the obvious indication is to reduce the hyperæmia of the vessels as quickly as possible and subdue or diminish the inflammation. For this purpose bags or bladders of ice should be immediately applied over the head and to the nucha, and constantly retained there as long as there is no complaint of chilliness, no marked diminution of temperature, and the patient experiences some relief from the intense headache and other symptoms. Bran mixed with pounded ice produces a more uniform coldness and is sometimes more agreeable to the patient than the ice alone. The bag or bags should be about one-third full, so as to fit upon the head like a cap, and the nurse should be instructed to renew the ice as soon as it melts. In severe cases, with marked elevation of temperature, it is proper to apply cold over the dorsal and lumbar vertebræ, as well as upon the head and nucha. A hot mustard foot-bath or a general warm bath in those cases in which convulsions are present or threatening, or in which there is delirium or great agitation or severe peripheral pains, is also useful, since it has a calmative effect and acts as a derivative from the hyperæmic nerve-centres. One writer states that he obtained marked benefit in a case by immersing the body to the neck in hot water.

The abstraction of blood, usually by leeches applied to the temples, behind the ears, or along the spine, has been employed, but even in the commencement of the present century, when it was customary to bleed generally and locally in the treatment of inflammatory and febrile diseases, a majority of the American physicians whose writings are extant discountenanced the use of such measures in the treatment of this disease. Drs. Strong, Foot, and Miner, though under the influence of the Broussaian doctrine, were good observers, and they soon abandoned the use of the lancet and leeches in the treatment of these patients for more sustaining measures. Strong[1] states that certain physicians employed venesection as a means of relieving the internal congestions, but, finding that the pulse became more frequent after a moderate loss of blood, they soon laid aside the lancet. Some experienced physicians of that period, however, continued to recommend and practise depletion, general as well as local, as, for example, Dr. Gallop, who treated many cases in Vermont in the epidemic of 1811.

Venesection in the treatment of cerebro-spinal fever is universally discarded at the present time in this country and Europe, but some intelligent physicians, as Sanderson and Niemeyer, approve of local bleeding in certain cases. It is, in my opinion, after examining the histories of many cases, uncertain whether the abstraction of blood should ever be recommended, but if it be prescribed it should be on the first day, when the hyperæmia is greatest, by the application of only a few leeches behind the ears, and never except when coma or convulsions are present or threatening and the patient is robust. The fact should not be forgotten that cerebro-spinal fever is in its nature asthenic and protracted, and that the intense inflammatory congestion of the nervous centres can ordinarily be relieved, if relieved at all, by the other measures recommended, which do not reduce the strength. The alarming symptoms which usher in an attack, the intense headache, restlessness, delirium, sometimes eclampsia or coma, seem to demand the most energetic treatment, and yet it is surprising to one who has his first experiences with this malady how patients under proper treatment, without the abstraction of blood, emerge from an apparently almost hopeless state and ultimately recover. There may be total unconsciousness, the pupils dilated like rings and insensible to light, the head intensely hot, tonic convulsions present or alternating with frequent clonic convulsions, and yet these symptoms, which in any other disease would be regarded as sufficient to justify the prognosis of certain death, may gradually pass off towards the close of the first or in the second week, and the case afterwards progress favorably. In the New York epidemic of 1872, previous to which physicians of this city had no personal experience with cerebro-spinal fever, many cases were pronounced hopeless which ultimately did well without abstraction of blood. In a case occurring in the practice of Dr. Griswold the patient was comatose for three days, with pupils not responding or but very feebly responding to light, but he recovered without the abstraction of blood, and with the remedies ordinarily employed. In a case which we will presently relate in speaking of another local treatment, the patient was still insensible in the third week, with pupils greatly dilated and insensible to light, and yet recovered without losing blood. Such cases show that the most urgent symptoms, such as seem to indicate the prompt employment of leeches in order to reduce the meningeal hyperæmia and the consecutive congestion of the nerve-centres, may be relieved and the patient recover without such depletion, and with the preservation of the blood, which is so much needed in the subsequent asthenic course of the malady.

---

[1] Medical and Physiological Register, 1811.

In only one case have I recommended the abstraction of blood, and this was so instructive that I will briefly relate it. A girl, four years of age, was seized on March 7, 1873, with vomiting, chilliness, and trembling, followed by severe general clonic convulsions lasting about fifteen minutes; was semi-comatose; pulse 132, and a few hours later, 156; temperature 101¼° F.; respiration 44; eyes closed, pupils moderately dilated and feebly responsive to light, dusky mottling of skin, constant tremulousness with twitching of limbs. Bromide of potassium was administered in hourly doses of four grains, ice applied to the head and nucha, and a hot mustard foot-bath followed by sinapisms to the nucha. On the following day, March 8, she was partly conscious, when aroused, but immediately relapsed into sleep, head retracted, bowels constipated; pulse 136; temperature 102°; vomited occasionally. It was thought proper, on account of the extreme stupor, to apply one leech to each temple, and the bites trickled slowly nearly five hours. The other treatment was continued. On the 9th the pulse was 180, so feeble that it was counted with difficulty; temperature 101½°. The patient was evidently sinking. It was necessary to order whiskey in teaspoonful doses every two hours, with beef tea and other most nutritious drinks. Evening, pulse 172, still feeble. March 10, pulse 180, barely perceptible; great hyperæsthesia; axillary temperature 100°; axes of eyes directed downward. After this the patient gradually rallied for a time, the pulse becoming stronger and less frequent, but death finally occurred after nine weeks in a state of extreme emaciation and exhaustion. Slight convulsions occurred in the last hours.

It is seen that in the above case, which may be regarded as typical, the patient passed into a state of extreme prostration after the application of the leeches, so that for three days I did not believe that she would live from hour to hour, and death occurred after an illness of nine weeks, apparently from sheer exhaustion. Experience like this, which corresponds with that of most other observers, shows the necessity of preserving the blood and thereby the strength, however urgent the initial symptoms, inasmuch as cerebro-spinal fever in its subsequent course is attended by such marked asthenia. On May 3, 1878, a boy of ten years was admitted into one of our best hospitals, in the service of a prominent New York physician. It was stated that he had been four days sick with cerebro-spinal fever, and among other characteristic symptoms he had had delirium every night and on May 2 delirium in the daytime, which had abated considerably after free epistaxis. In the hospital the application of ten leeches along the spine was ordered, but it does not appear to have diminished the delirium or any other symptom, and on the following day the pulse was so frequent and feeble that active stimulation by brandy was resorted to. He had three strong convulsions on May 13, which were relieved by ice to the head and nape of neck, and by six minims of Magendie's solution. Severe pains occurred at times in the back and limbs, and on the 29th, one month after the commencement of the disease, the same pain frequently recurring, twelve leeches were ordered to be applied to the spine. On June 2 the limbs were flexed and quite stiff, and the effort to move them was attended by great pain. The pain in the back was also more constant, and in consequence sixteen leeches were applied to the spine. The next day there was no pain, but the patient was very stupid. On June 6 the records state that he was obviously losing strength day by day, that his emaciation was extreme and his anæmia very marked. But he had great vitality, and, although he had strabismus, bed-sores, incontinence of urine and fæces, and extreme prostrasion, he lingered till August 1. At the autopsy, "body a skeleton; brain, dura mater, and pia mater appear normal, except a little thickening of latter at base of brain; ventricles much enlarged and full of clear serum; surface of walls of ventricles looks normal, but is soft; spinal cord and membranes appear normal to the naked eye." No disease was discovered in other organs, except that the liver appeared congested and the kidneys pale. It can scarcely be doubted that, although some temporary relief from the pain may have resulted to this patient by the repeated application of leeches, which diminished the meningeal hyperæmia, yet his chances for ultimate recovery would have been far better without such depletion. Therefore the histories of cases show that the result of abstraction of blood has been unsatisfactory, on account of the asthenic nature and protracted course of cerebro-spinal fever, and it should never be recommended as a remedial agent.

Some benefit is apparently derived from the application of stimulating and moderately irritating lotions along the spine. A liniment consisting of equal parts of camphorated oil and turpentine briskly applied by friction with flannel up and down the spine till redness is produced appears to cause some alleviation of the suffering, and it does not conflict with the use of the ice-bag. Dr. William H. Sutton, of Dallas, Texas, has published the following interesting case, showing the benefit from stimulating and irritant applications over the spine made in an unusual manner. A child, aged three and one-half years, had been three weeks under treatment, through error of diagnosis, for supposed continued fever. When Dr. Sutton assumed charge of the case, November 20, 1877, the pupils were greatly dilated and insensible to light; features pallid and pinched; pulse 130; temperature 103° F.; patient totally unconscious. November 21, morning temperature 105°, pulse 140; evening temperature 101¼°, pulse 120. November 22, morning temperature 106½°, pulse 160; restless; evening temperature 105½°, pulse 120; had not slept, except for moments, for nearly two weeks. A strip of flannel saturated with turpentine was placed over the spine from the neck to the sacrum, and a hot smoothing-iron was run up and down it, and eight drops of the fluid extract of ergot were given every three hours. Dr. Sutton adds, "The father stated to me that as soon as the application was finished the child fell asleep, and slept several hours,—the first for two weeks,—and the fever rapidly declined. From this time he began to improve, and gradually and fully recovered." The use of irritating applications over the spine in the treatment of cerebro-spinal fever has been long and favorably known, but the mode of applying it practised in the above case is novel.

Internal Treatment.—It will aid in the selection of the proper remedies to recall to mind the pathological state which we know to be present from the many autopsies which have been recorded. We have seen that the largest mortality, and consequently the most dangerous period, is in the first days, when there is intense suddenly-developed inflammatory congestion of the meninges, with more or less secondary hyperæmia of the underlying brain and spinal cord, producing great headache, delirium, or somnolence, with exaggerated reflex irritability of the spinal cord, so that eclampsia is a common and fatal complication.

Fortunately, a remedy has been discovered in modern times, the bromide of potassium, which acts promptly and efficiently. It can be safely administered in large and frequent doses to the youngest child. It is quickly eliminated from the system through the kidneys and other emunctories in children, so as to prevent the occurrence of bromism, at least to the extent of causing any unpleasant consequences. It causes contraction of the minute vessels of the nervous centres so as to diminish the hyperæmia, as shown by the experiments and observations of Dr. Putnam-Jacobi and others, and at the same time it diminishes, in a marked degree, the reflex irritability of the spinal cord, two most beneficial and important effects of its use in this disease. Many children by its timely employment are saved from the dangers of eclampsia, and by its sedative effect on the nervous system and contractile action on the capillaries it probably diminishes the intensity of the inflammation and the amount of exudation. I usually prescribe it, as recommended by Dr. Squibb, dissolved in simple cold water. In ordinary cases not attended by eclampsia or marked symptoms which show that eclampsia is threatening, I generally prescribe at my first visit about four grains every two hours to a child of two years who has the usual restlessness and apparent headache, and six grains to a child of five years. If eclampsia occur, the bromide should be given more frequently, as every five or ten minutes, till it ceases. It is important to be able to determine when the quantity of the bromide administered should be diminished, and when its use should be discontinued. I have very rarely observed bromism in children, and never to the extent of doing any serious harm, though for many years I have administered it in large and frequent doses whenever the occasion seemed to require it; but the symptoms of bromism cannot readily be discriminated from those which may result from cerebro-spinal fever, such as muscular weakness, dilated pupils, with perhaps impaired vision, unsteady gait, nausea or vomiting, and abdominal pains. If the case progress favorably, frequent and large doses should, in my opinion, be given only in the first week, after which this agent should be given at longer intervals, or in smaller doses. But during exacerbations, which are liable to occur from time to time till the patient is well on the way to recovery, the use of the bromide in full doses is again indicated till the urgent symptoms begin to abate.

Antipyrin promises also to be another useful remedy in this disease, from its well-known action in relieving headache, reducing fever, and procuring sleep. It may be administered with the bromide. It appears to be a very useful adjuvant to the bromide during the first week, when the temperature is most elevated and the headache severe. At a later stage, when asthenic symptoms are more pronounced, its use appears to be contra-indicated, unless in exceptional instances.

Ergot is another very important remedy. It is scarcely less useful than the bromide, from its action in contracting the arterioles and diminishing the flow of arterial blood. The fluid extract, tincture, or wine of secale cornutum can be employed, or its active principle ergotin. In New York City, Squibb's fluid extract has been more used than any other preparation. I have commonly prescribed it except for patients old enough to take ergotin in the pill. The doses employed by different physicians vary greatly. Dr. William A. Thomson, Professor of Materia Medica in the New York University, has prescribed, so far as I am aware, the largest doses in the treatment of this disease,—to wit, one teaspoonful of the fluid extract of secale cornutum every three hours, to a boy of ten years in Roosevelt Hospital in 1878, with apparent benefit as regards the meningeal hyperæmia, although the case was fatal after the lapse of several months from asthenia. The alkaloid ergotin, to which the beneficial effects of the secale cornutum are due, may be given in the pill or in solution. In case of much irritability of the stomach it can be employed hypodermically, dissolved in water with glycerin. The efficacy of this agent is most marked during the first and second weeks, when the congestion of the nervous centres is greatest. At a more advanced stage, when there is less congestion and the danger arises from the inflammatory products and structural changes, the time for the use of ergot is past, or if it is still of some service it is less needed than at first and should be given less frequently.

The severe headache and restlessness which attend many cases require the occasional use of an opiate, or the hydrate of chloral. Chloral in proper dose never fails to give quiet sleep, and it is supposed by some who have studied its therapeutic action that it diminishes the cerebral circulation. It is therefore a useful adjuvant to the bromide. Five grains usually suffice for a child of six to eight years. Chloral is especially useful in cases attended by eclampsia, or by symptoms which threaten eclampsia, since it acts promptly and decidedly in diminishing reflex irritability. Formerly it was considered injudicious and unsafe to prescribe opiates in meningeal inflammation, since it was supposed that they increased the liability to coma, but experience shows that they are sometimes very useful in this disease when administered in small or moderate doses, and without the risk which was once supposed to be incurred by their use. The thirty-second part of a grain of morphia administered at intervals of some hours was sufficient to relieve the suffering of one of my patients at the age of six years.

Quinia apparently does not exert any marked controlling effect on the course of cerebro-spinal fever or its symptoms, although the paroxysmal character of the severe pains in many patients suggests the use of this agent as an antiperiodic. It was frequently prescribed by New York physicians in the epidemic of 1872, but I believe that the opinion was unanimous that it was not the proper remedy. I have prescribed it in large and small doses, in one instance giving fifteen grains to a child of thirteen years, but do not know that I have derived any benefit from its use in this malady.

When the acute stage has abated, measures designed to remove the serum which sometimes remains, constituting a hydrocephalus, are indicated. For this purpose the iodide of potassium is probably more useful than any other agent. It is administered by some physicians early, along with the bromide, as they have been in the habit of treating other forms of meningitis. I have prescribed it with the bromide, and alone when the bromide was discontinued, but whether it produces any marked sorbefacient effect in this disease seems to me doubtful.

The result depends to a great extent on the nursing. The skill of the physician may be thwarted and the life of the patient lost by inefficient nursing. No other disease more urgently requires kind, intelligent, and constant attendance night and day on the part of the nurses. Not only should the medicines and nutriment be given punctually and regularly, but the great restlessness of the patient in the first days requires constant readjusting of the ice-bags, and during the long period of convalescence the utmost care is required to remove at once the excretions in order to prevent bed-sores, and to give the proper amount and kind of nutriment to prevent the emaciation and weakness from which many perish.

The diet, from the beginning to the end of the malady, should be the most nutritious, and such as is easily digested. It is necessary to give it in the liquid form, unless in mild cases in which the appetite may not be entirely lost. It is proper to aid the digestion by pepsin preparations. Nutritive enemata, consisting of beef tea, or one of the extracts of beef, milk, and brandy, aid in averting the fatal prostration in protracted cases. After the acute stage has passed and the meningeal hyperæmia has abated, the alcoholic compounds in moderate doses, which in the beginning would be very injurious, may now be useful, administered regularly by the mouth. The room should be dark, well ventilated, and quiet. All sympathizing friends who are not required in the nursing should be excluded. I know no other disease in which this is so necessary, for mental excitement may produce dangerous aggravation of symptoms.

SCARLET FEVER.

By SAMUEL C. BUSEY, M.D., LL.D.

Definition.—Scarlet fever is an infectious disease, due to a specific contagion, and characterized by a peculiar exanthem, more or less diffused over the entire surface, an angina of variable intensity, and a fever, which may be appreciable only with the thermometer or so intense as speedily to destroy life. It is irregular in form, intensity, and prevalence. These diversities are exhibited in individual cases, in the constitution of epidemics, and in the morbid process in the organs which may be involved. Its epidemicity and contagiousness are established, yet the intensity of the contagion is so variable and individual susceptibility and immunity are so inconstant that those who may escape a prevalent and virulent epidemic may be seized, subsequently, during an epidemic of lesser prevalence and malignity, and in the same epidemic individual cases will, without apparent cause, vary from the lowest to the highest grade of intensity. The difference in the susceptibility of persons and the variability of the poison in virulence and diffusion are more markedly exhibited in this than in any other of the exanthematous affections.

Synonymes.—Scarlatina ; Scarlet rash.

History.—Until Sydenham established the unity and specific nature of scarlet fever it had been considered a variety of measles, differing only in the form of the exanthem. During the prevalence of the epidemic in London from 1661 to 1665 the scarlatinal eruption was carefully studied and its distinctive characters and differentiation were definitely established. To what extent it had prevailed previous to that period cannot be ascertained, but it is generally believed to have appeared in mild epidemics, limited to circumscribed regions, for a very long time anterior,—perhaps quite as early as measles. How, when, or where it originated are questions which cannot be answered. During the past two hundred years its course, progress, prevalence, and epidemic character have been carefully recorded and studied. This later history justifies the conclusion that it has continuously increased in prevalence, extending over larger areas and invading widely-separated countries, and that with the more frequently recurring and more widely spread epidemics it has assumed more dangerous forms. At the present time it is the most prevalent and fatal of the exanthematous maladies.

Its greater prevalence is undoubtedly due to the rapid increase in population; and its wider dissemination and invasion of remote regions and countries are attributable to the increased facilities of intercommunication. The first epidemic in this country occurred in 1735.

Etiology.—Scarlet fever is due to a specific poison capable of reproducing itself. That such a contagion exists, possessing the power of infecting unprotected persons with a disease similar in all its essential characteristics, and that every such case is the result of such infection, cannot be doubted. It is true that cases occasionally occur independent of any epidemic, and, apparently, of any contagious element; but such cases, like those occurring under circumstances readily explained, are due to infection with the scarlatinous poison. The failure to establish the origin of such cases and to connect them with the specific contagion is partly due to defective methods of investigation, but mainly to the special qualities of the contagion, which characterize it as a volatile, diffusible, portable, minutely divisible, and tenacious poison, possessing a vitality and latency which permit its transportation to great distances. The further fact that the contagion of such sporadic cases will always reproduce the disease in unprotected persons seems conclusive against their spontaneous origin.

The nature of the contagion has not been determined. Experimental and clinical observation point to the existence of a scarlatinal microbe, but such an organism has not been isolated or demonstrated. That it is a contagium vivum[1] seems indisputable; for it is inconceivable that a poison of spontaneous origin could possess such special and peculiar properties, capable of reproducing a disease which would afford immunity from subsequent invasion and infection.[2]

Its volatility is established by its minute divisibility and diffusibility in the atmosphere, its rapid transference from person to person without direct contact or close proximity, and its wide-spread prevalence among unprotected persons in the same or neighboring communities.

The evidence in favor of its portability is conclusive. It may be conveyed by the clothing, furniture, toys, flowers, letters, locks of hair, and food from the sick-room. Any article of wearing-apparel, either of the sick person, physician, or nurse in attendance, or of any other person who may be exposed to the direct contagion, may convey it. During such conveyance its latent vitality will remain unimpaired for a considerable time. The wearing of the clothing of the sick, occupancy of the sick-room, dusting, beating, and cleaning the clothing of the sick, and even a visit to the house of a scarlatinous patient, have frequently infected susceptible persons. It may also be communicated by domestic animals. It is even believed that the horse, dog, cat, cow, and swine may contract the disease, and thus become sources of direct contagion. It may be disseminated by contaminated drinking-water, and in later years has been quite frequently communicated by infected milk. Three kinds of milk epidemics are recognized by Klein, of which examples may be found in the literature of recent epidemics in England. In one kind the infective material is communicated to the milk by the exposure of either the milk or the milk-cans to a patient during the desquamative stage; in the second kind, by the conveyance of the poison to the milk from an infected cow; and in the third kind, by the direct poisoning of the milk of a cow suffering with the disease. The most common mode of infection is, however, from direct contact with a scarlatinous patient.

---

[1] It appears to have been established that a virus may retain its pathogenetic power after being deprived of living micro-organisms. Chauveau supposed he had proved this in 1880; but Pasteur claims that it has only been recently demonstrated in his laboratory. The fact is, however, that Salmon and Smith in 1885 made the decisive demonstration which settled the question.

[2] Richardson believes "that the disease is caused by the development of a chemical body, which by its presence gives rise to the symptoms, and by its ultimate elimination frees the system from them all except those which are secondary."

The "Hendon outbreak" of scarlet fever in London, in December, 1885, was associated with the distribution of the milk from a herd in Hendon that was affected with an infectious and contagious disease communicable by inoculation to healthy cows and to man. Klein discovered in the discharge from the ulcers on the udders of the affected cows a micrococcus which he believes is identical with a micro-organism which he has found in the blood of human scarlet-fever patients. Edington, Thin, and others, who have pursued a similar line of experiment and investigation, have failed to verify the conclusion of Klein that the Hendon disease was scarlet fever. The streptococcus isolated and cultivated by Klein is associated with an eruptive disease of the udders and teats of cattle, but it has not been proved that this disease is scarlet fever.

The extraordinary tenacity and vitality of the scarlatinal contagion are shown by the various modes of conveyance, propagation, and infection, but the duration of its vitality has not been ascertained. Richardson reports that five months after the first case occurred children became infected when they occupied a room under the infected straw roof of a house; and it is stated that Hildenbrand's coat retained its contagiousness for one year and a half. Numerous recorded observations indicate the inertness of the ordinary means of purification and cleanliness, and that continuous and energetic ventilation but imperfectly accomplishes the removal of the poison.

The contagiousness of scarlatina is conclusively proved by the communicability of the disease to a healthy person by inoculation. Various experiments have been performed with the view of producing an attenuated virus which might be employed to secure immunity from the more fatal forms, but the generated disease has proved even more dangerous than that contracted in the ordinary mode. The disease has been produced by inoculation with the blood, epidermic scales, and serum from cutaneous vesicles, and the persons so inoculated have not contracted the disease subsequently when exposed to the infection. It may also be communicated by the pulmonary and cutaneous exhalations, by the nasal and pharyngeal secretions, and by the urine. These experiments and clinical observations show that the blood is the essential seat of the contagion, and that it is disseminated throughout the tissues, secretions, and excretions of the body.

The contagion is so volatile and intense that the briefest contact with a scarlatinous patient or exposure to the atmosphere of the sick-room may be sufficient for infection. In fact, the receipt of a letter from a long distance, written in the room of such a patient, may communicate the disease to susceptible children. A third person pausing for a moment in the room may convey the contagion for a distance and infect susceptible persons for weeks after. The poison may, however, be diluted and rendered innocuous by thorough and persistent ventilation of the sick-room. Well-directed ventilation, and isolation of the patient, may limit the spread of the disease in a household. Some maintain that the spreading of the disease may be effectually arrested by isolation of the patient, disinfection of the sick-room and clothing of the patient, inunction of the body with some disinfecting material, and scrupulous cleanliness. Whether or not the poison can be destroyed by such means is questionable, but that its intensity and diffusibility can be thus diminished is beyond dispute. The accepted belief is that the poison can be destroyed only by heat, and that a temperature nearly up to 212° F. is necessary.

The period of most intense contagiousness and the duration of capacity for infection have not been positively settled. Some contend that the stage of desquamation, others that during the bloom of the eruption, is the most contagious period. The only fact universally admitted is that with restoration to health the contagiousness declines; but it certainly does not cease until desquamation has been completed, and it is known to have remained attached to dropsical patients. The period of infectivity must then necessarily vary with the patient. Some will desquamate more rapidly than others; some will suffer from dropsy or other sequelæ, while others will escape. With the termination of desquamation, completion of convalescence, and restoration to health, infectivity will certainly have ceased.

The varying predisposition, susceptibility, and immunity of individuals and families are as remarkable as they are inexplicable. In a family of children one or more may escape or suffer but a mild attack, whilst the remaining members may, without apparent cause, exhibit the most intense susceptibility. Families residing in close proximity, perhaps in adjoining houses, and subject alike to the infection, may be very differently influenced. In one the most aggravated form of the disease may prevail, and the other will be protected by a special immunity. This absence of susceptibility may continue throughout life, or only during the prevailing epidemic, or it may continue during residence in one locality and disappear upon removal to another in the same city or to some distant village or city. Predisposition may be increased or diminished by locality. Rapid and fatal cases indicate extraordinary susceptibility.

A population long exempt from scarlet fever does not necessarily exhibit very marked susceptibility when invaded by an epidemic. If such invasion is long continued, the susceptibility seems to increase, and individuals and families that have escaped previous epidemics may exhibit susceptibility in its most aggravated forms.

It quite frequently occurs that persons who have escaped the disease, or who have had a mild or severe attack, will upon every subsequent exposure to the direct contagion suffer more or less from a sore throat. This form of angina may recur with every exposure, or it may cease after one or more attacks.

Social position and external circumstances influence the mortality, but do not seem to affect the predisposition. The death-rate increases with poverty and diminishes with affluence. This difference is undoubtedly due to the condition of life rather than to any difference in constitution. Among the well-to-do, the modifying influence of ventilation, diffusion and dilution of the poison, more efficient care, nursing, feeding, and medical attendance is very markedly shown in the diminished mortality.

Age exerts a very decided influence. No age is exempt. Children have been born with scarlet fever, and newly-born infants are occasionally attacked, but during the first year the susceptibility is not very marked. It is increased during the second year, but between two and seven years it is most intense.[1] After the tenth year the liability is greatly diminished, and more so after the fifteenth. Only about 1.75 per cent. of cases occur in adults over twenty-five years of age. These facts point very clearly to the value of isolation. If children can be protected during the first ten years of life, the chances of escape are greatly enhanced, and the danger is greatly lessened.

Sex and race do not influence the predisposition. Previous condition of health is a doubtful factor. Conditions of soil seem occasionally to favor the prevalence of the disease, but residence in the country or in cities does not show any marked difference. Occupation is a doubtful element. Very many unprotected persons whose employment brings them in contact with affected persons contract the disease, but this cannot be ascribed to the occupation except in so far as it may expose them to the contagion.

Epidemics of scarlet fever occur most often in the autumn, and in successive order of frequency during winter, spring, and summer. The condition of the weather does not influence the epidemics. Changeable, cold, and moist weather does not aggravate them. In fact, they seem to be independent of atmospheric conditions. Altitude is without influence.

Pregnancy increases the predisposition of the unprotected, but not so markedly as the lying-in and the nursing. The wounded and those who have undergone operation seem to acquire increased susceptibility.

---

[1] The rarity of the disease under two years is denied by some. Others assert that the most common age is about the third or fourth year.

The prevalence of epidemics of scarlet fever is mainly dependent upon personal intercourse. All other conditions and elements are of minor importance. Locality, condition of the subsoil, season, density of population, and circumstances of life may increase or lessen the predisposition, but without personal intercourse and the direct conveyance of the contagion from the affected to the unprotected the spread of the disease in any community would be circumscribed, and in most instances limited to comparatively few of those susceptible to it. In view of this fact, immediate isolation of the sick should be imperative, and non-intercourse should be established and maintained until the period of desquamation has been completed and health restored. Epidemics of scarlet fever usually begin with a few scattered cases, and not infrequently, such cases being of a mild type, no restrictions are imposed beyond those voluntarily assumed by the patient. Intercourse with other children is permitted, and the patient is usually returned to the school to disseminate the disease throughout the community. A mild and apparently sporadic case is no guarantee against a wide-spread, virulent, and fatal epidemic. The susceptible in any community cannot be considered safe until the epidemic has entirely disappeared. Its subsidence and disappearance are usually very slow. The epidemic may have lost its force and virulence, but the single cases occurring at localities distant from one another may continue and prolong the infectious influence until the subjects are exhausted.

A study of the history of the epidemics of scarlet fever during the past two and a half centuries has suggested the theory of periodic recurrence. Whilst this is apparently true in some localities, the exceptions have been too numerous to permit the acceptance of the law of periodicity. In some cities it has become endemic. In fact, in many populous cities the disease is probably always present. Occasionally it has become pandemic, spreading over vast areas of country and continuing through a protracted prevalence.

Epidemics of scarlet fever frequently follow in the wake of epidemics of measles. This fact does not establish any relation of cause and effect between the two diseases, but favors the theory that measles in some inexplicable manner increases the susceptibility of unprotected individuals and thus prepares the field for the rapid dissemination of the poison of scarlet fever.

Prevention.—There is no effectual method of protecting the susceptible from the contagion of scarlet fever. Much can be accomplished in limiting the prevalence of the disease by prompt and efficient isolation of the sick, and by non-intercourse. The intensity of the poison may be diminished by ventilation, diffusion, and dilution. It is also highly probable that its mortality may be greatly diminished by the rigid enforcement of hygiene, both domiciliary and personal.

All experiments to secure protection by the internal administration of drugs have failed. For a time many, especially laymen, harbored the conceit that the administration of belladonna in minute doses would afford protection; but the theory has long since been exploded. Of late years the employment of antiseptic gargles and inunctions has been advocated by some. Jamieson maintains that the exhalations from the mouth and throat and the particles of cast-off cuticle are the sources of infection, and insists that the contagion may be destroyed by frequent applications to the mouth and throat of a strong solution of boracic acid in glycerin. The skin, including the scalp, should be thoroughly bathed daily with warm water, and twice daily the entire surface of the body should be anointed with an ointment composed of carbolic acid gr. xxx, thymol gr. x, vaseline ʒi, and simple ointment ʒi; or the following salve: "resorcin 1, lanoline 6, and ol. sesame 2 parts. This is rubbed into the skin to hasten desquamation and to destroy the specific organism." Brown uses a five-per-cent. carbolized oil inunction all over the body, except on the face, where olive oil is used. This is done daily for six or eight weeks, followed by a warm bath. He claims that sequelæ are averted. Long anoints with a carbolized inunction, and gives daily baths as soon as the patient's condition will permit. Wigglesworth employs carbolic acid internally, rendered liquid by ten per cent. of water. Three to six minims are given every two hours day and night during the first three days and continued at longer intervals during the four or five succeeding days until the urine is deeply discolored. He gives one minim three times a day to everybody in the dwelling, and claims complete protection. The reports seem to establish the value of these methods of prophylaxis. It is undoubtedly true that frequent bathing and inunction during the period of desquamation will effectually prevent the diffusion of the cast-off particles of the epidermis in the surrounding atmosphere, and thereby limit to a very considerable extent the dissemination of the poison; but it is not believed that the vitality of the poison is lessened. Walford claims that arsenic given during the incubative stage will either prevent or greatly modify the disease. He employs the liquor arsenicalis in as large dose as the age of the child will permit, in combination with sulphurous acid and syrup of poppy. The dose should be given daily for several days, and then less frequently.

Separation and disinfection are the most effectual prophylaxes. The discharges of the patient and all vessels employed in the sick-room should be thoroughly disinfected. The clothing worn by the patient, the bed-linen, and other clothing should either be destroyed or be submitted to some certain disinfecting process. The mattress should be burned. After the patient is well and has returned to the family circle, the room should be subjected to an equally effective process of cleansing and disinfection. No unprotected person should be permitted to occupy it for a reasonable period thereafter.

The prevalence and mortality of scarlet fever demand the rigid enforcement of every practicable method of prevention.

Incubation.—The period of incubation varies. In the vast majority of cases it varies from two to eight days. There are, however, many exceptions to this general law. In occasional instances the disease has developed a few hours after the first and only exposure; in other instances, far more numerous than those of brief incubation, it has been delayed for several weeks. It is never safe or wise to pronounce a child who may have been exposed to the contagion free from the danger of an attack until several weeks, at least three, have elapsed after the date of last exposure. In many of the cases of delayed incubation it may be that a second or a third exposure has occurred, and the period of incubation in such cases would date from the last. In this as in many other particulars scarlet fever presents very many anomalous variations, which demand unusual alertness on the part of the medical adviser.

Immunity.—Reference has been made to the peculiarities of constitution and variations in the susceptibility of individuals and families, through which some seem to have inherited and others acquired an immunity which continued throughout life or vanished under changed conditions of life. Immunity may be acquired by age. The disease rarely occurs more than once in the same individual. Occasional instances of a second, a third, and even a fourth attack have been reported. It is a common but a mistaken belief among laymen that second and third attacks are frequent. Physicians sometimes pronounce cases of roseola and erythema to be mild attacks of scarlatina, and, when the mistake is recognized, fail to correct the diagnosis. Then, again, mistakes are made, and parents are greatly surprised when another physician, usually in a different city, ascribes the illness of their child to an attack of scarlet fever. The writer during a considerable experience has never seen a second or subsequent attack, but he has very frequently encountered the assertion of second and third attacks. Why the susceptibility is not completely destroyed by a first attack is as inexplicable as the fact that a single attack usually affords complete immunity for life. By a second attack is meant a new case occurring after a shorter or longer interval, without any connection with the first attack, and after a subsequent exposure to the contagion. Such cases must be distinguished from the cases of relapse which follow immediately in the wake of the first attack, and also from the cases of pseudo-relapse, which are characterized by the recurrence of the exanthem during the second or third week of the disease.

The survival of susceptibility is most frequently a family inheritance. In some cases it seems to be due to the incompleteness of the first attack, and in other and rarer instances changed conditions of life seem to have revived the susceptibility.

Pathology.—The special characteristics of the contagion of scarlet fever have been set forth in the preceding sections. Beyond this but little is known in regard to the nature of the disease. Recent experiments and investigations by Klein, Edington, Jamieson, and others point very distinctly to the existence of an organism peculiar to scarlet fever. A number of organisms have been discovered in the blood and desquamation, and some of them have been isolated and cultivated. The indications are that the bacillus scarlatinæ, which consists of "rods measuring 0.4 m. in thickness and 1.2 m. to 1.4 m. in length, most usually forming excessively long-pointed and curved filaments," is the specific cause of scarlet fever.[1] But control experiments of sufficient magnitude have not been made to establish the fact.

Pathological Anatomy.—The morbid anatomy in scarlet fever consists mainly in the changes which take place in the integument, subcutaneous connective tissue, and mucous membrane of the oral and nasal cavities and throat. The changes which are found in the viscera refer more particularly to the complications and sequelæ. The skin is hyperæmic, and the surface is more or less covered with an exanthem, which consists of numerous and closely-aggregated points, slightly red in the beginning, but rapidly increasing in redness, sometimes to a brilliant scarlet color. They are seldom larger than a pin's head, and may be separated by pale points of skin. They may be, and most usually are, so crowded together as to present to the naked eye a uniform scarlet redness of marked intensity. These points may be flat or slightly elevated, are usually circular in form, but may be elongated. Marked confluence with vivid redness denotes increased hyperæmia. In mild cases with moderate hyperæmia the points remain isolated and disappear with desquamation.

The exanthem usually maintains its maximum development for one or two days, rarely less than one day, and then gradually fades, to disappear with the beginning of desquamation.

The subcutaneous cellular tissue is but slightly affected in mild cases. The infiltration and exudation are limited to the superficial layers, but in severe cases they may be greatly increased and extend to the subcutaneous cellular tissue. Exudations of blood may also take place in the layers of the skin and in the sub-integumental tissue. In consequence of the hyperæmia of the skin and the exudation into the rete Malpighii, there takes place a rapid new formation of epidermis and consequent exfoliation. The desquamation may be either branny or lamellar, and may recur several times, dependent upon the nature of the epidermis and the intensity of the exanthem. After the completion of the desquamation the skin returns to its normal condition, except in very rare instances. After a relapse the process of desquamation may be repeated.

The changes in the organs of the throat and the mucous membrane of the oral cavity and pharynx vary. It may be a simple turgescence, with moderate swelling of the uvula, palatine arches, and tonsils, and increased secretion; or it may be a much more extensive inflammation, involving the posterior pharyngeal wall and the structure of the tonsils, and extending throughout the mucous membrane of the mouth and the lining membrane of the nasal cavity. The tonsils may be greatly enlarged; sometimes abscesses form in them. Follicular abscesses are quite often observed on different parts of the inflamed mucous membrane, most frequently on the soft palate. In some cases the turgescence is so intense as to impart a livid color to the parts, and the œdema of the soft parts is so great as to interfere with deglutition. In more severe forms of scarlatinous angina the submucous cellular tissue may become involved in the inflammatory process. This may be limited to small areas, or it may extend to the post-pharyngeal and laryngeal regions, or externally to the neck and to the parotid and submaxillary glands. The swelling outside about the neck, jaws, and temples is sometimes very considerable, and may result in very extensive abscesses. In such cases the parenchymatous inflammation of the tonsils progresses very rapidly to the formation of large abscesses. Gangrene with extensive sloughing may follow. This destruction may be limited to the tonsils, or may extend in any direction to the parts involved in the inflammation.

---

[1] Shakespeare, Ann. Univ. Med. Sci., vol. v. p. 465 et seq.

There is no longer any doubt that scarlet fever may be associated with the formation of a membrane in the throat and upper air-passages similar to diphtheritic exudation. In some cases the membrane is diphtheritic; in others it is a scarlatinal affection. In the latter class of cases it appears between the third and sixth days, rarely invades the larynx, but may extend into the posterior nares. The cervical glands may enlarge and suppurate, but this form is never followed by paralysis. The exudation may be a soft, white, pultaceous, easily-detached deposit, may consist of layers, or may be a well-formed membrane, attended with dysphagia, swelling of the cervical glands, and infiltration of the cellular tissue of the neck, and may invade the nasal cavities and ears. True diphtheria does not usually appear before the second week, and is a much more serious complication.[1] It may complicate the mildest as well as the severe forms of scarlatina, and may assume the form and extent either of an ordinary case of diphtheria, or of the most malignant type. In all cases the course of the fever is aggravated by this complication. It is probably due to a secondary infection with the germs of true diphtheria.

In rare instances the brain and its membranes are congested. The intestinal and mesenteric glands may be enlarged. Some have contended that the gastro-intestinal mucous membrane is inflamed and subjected to an exfoliation of epithelial cells. The spleen may be enlarged and softened. In fatal cases complicated with catarrh of the stomach and alimentary tract, bronchitis, pneumonia, pleurisy, peritonitis, endocarditis, or pericarditis, the ordinary changes incident to such inflammations will be found.

The kidneys quite often present evidences of derangement. Some recent authors consider renal catarrh a necessary accompaniment of scarlet fever;

---

[1] The duality of scarlatinal diphtheria and true diphtheria is not admitted by all observers. Some maintain their identity. Weigert and Heubner claim to have demonstrated the anatomical identity of scarlatinal and ordinary diphtheria; but the etiological and clinical differences have not been established.

but this view has not been generally accepted. The changes are not uniform. The organs may be simply congested, and changes more or less marked may be found in the glomeruli, arterioles, and convoluted tubes. There may be proliferation of the nuclei in the Malpighian tufts, degeneration of the intima of the capillaries, swelling and fatty degeneration of the epithelium of the convoluted tubes, infiltration of the interstitial tissue, and filling of the tubes with hyaline casts. In other but rarer instances the morbid appearances are more marked, and indicate later stages of the inflammatory process. The tissue-changes usually found are those of renal catarrh in its primary stage. In fatal cases of scarlatinal nephritis of long duration the tissue-alterations are more pronounced.

Forms.—Scarlet fever occurs in such a variety of forms, and the complications are so numerous, that it is impossible to arrange a classification sufficiently comprehensive to present them in a definite manner. The earlier authors described the disease under the subdivisions of scarlatina simplex, scarlatina anginosa, and scarlatina maligna; but later writers have generally adopted the classification of regular, irregular, and malignant, which will be followed in this chapter.

Symptomatology.—*Regular Form.* The regular form is characterized by a well-marked exanthem, angina, and more or less fever. It may begin suddenly, or be preceded by a day or two of indefinite indisposition, during which time the patient will complain of headache, with general malaise and loss of appetite. The tongue will be slightly coated. The bowels are usually constipated; occasionally there may be some looseness. In some cases there will be marked sluggishness, and in others fretfulness with loss of sleep. Most frequently the disease begins suddenly with a chill, vomiting, a convulsion, or a high fever associated with the usual phenomena of high febrile action,—headache, frequent pulse, flushed face, thirst, sparkling eyes, anorexia, twitching and starting, and perhaps delirium or stupor. The vomiting may be persistent, but usually it is not troublesome except in serious cases. A slight diarrhœa may supervene. These symptoms continue without abatement, and sometimes are increased, until the appearance of the eruption, which may occur within a few hours or be delayed one, two, or three days,—not often, however, later than twenty-four hours. The rash appears first about the neck, chest, and shoulders in indistinct points, increases rapidly in redness, and extends over the trunk and extremities, reaching its maximum development in rare cases during the first day, but most usually during the second, and in some cases not before the third or fourth day. When the development is slow the confluence is more marked. When at its maximum the entire body is covered, the skin being uniformly red, hot, and dry, and sometimes tense, swollen, very sensitive and œdematous, especially about the face and eyes. Sudamina may also appear, and in some localities extravasations may take place. The rash does not always become confluent, but frequently remains distinctly punctate. The degree of confluence and intensity of color are very variable. In some epidemics these conditions are much more marked than in others, and in different cases in the same epidemic there are great variations. In some cases the burning and intensely red and hot skin denote an unfavorable prognosis. During the development of the exanthem the other symptoms remain unchanged or increase, and during the period of its full development the accompanying symptoms and conditions usually reach their maximum intensity, to subside with its gradual disappearance.

The throat-symptoms begin with the onset of the disease and progress with the development of the exanthem, but it does not follow that an intense exanthem or a very high fever, or both combined, necessarily indicate a severe angina. In some cases the throat-affection, in others the constitutional conditions, predominate. A moderate exanthem may exist with a severe angina, and an intense and confluent exanthem may exist with a moderate angina. But as a rule the throat-affection, whether mild or severe, will increase during the periods of development and maximum intensity of the exanthem. With the fading and gradual disappearance of the rash the throat-affection usually subsides. In many cases, and in some epidemics, the throat-affection is insignificant, but as a rule the changes in the mucous membrane of the oral cavity and pharynx are sufficiently characteristic to be readily distinguished. In the beginning the tongue is reddened at the tip and coated white. The enlarged papillæ project through the coating. In a few days the coating disappears and the surface assumes the characteristic strawberry appearance, deeply red, with thickly-studded, enlarged, and shining papillæ. The lips are dry and crack at the angles. Niven says the breath is peculiarly sweet, almost aromatic, in the early stage of the disease. When the angina is severe, and more especially when ulceration occurs, the breath is foul, and fetid if suppuration and sloughing take place.

Sudden and marked elevation of the temperature, with corresponding rapidity of the pulse, is one of the most common initial and characteristic phenomena of scarlet fever. At the onset the fever may reach 102° F. and rapidly rise during the first day to 105° or 106° F. In some cases it may reach this and even a higher elevation in a few hours. In a majority of cases it will either continue during the period of development and maximum intensity of the exanthem to range between 102° and 104° F., or gradually rise during each succeeding day until the exanthem has reached its maturity, and then lessen daily with the gradual disappearance of the rash, until the normal is reached with the beginning of desquamation. The course of the fever is marked by remissions and exacerbations. In very mild cases there may be distinct intermissions. In this form the temperature does not often exceed 106° F., and this highest point is usually reached during the period of maximum intensity of the rash. With the rise and fall of the fever the scarlet color of the rash varies, increasing with the elevations and lessening with the remissions. During the period of high fever there is usually active delirium, in some cases stupor, and in others twitching, jerking, jactitation, tossing about the bed, moaning, and occasionally screaming as if in pain. The effects of high temperature are very different. Some patients will bear continuous high temperature without any cerebral or nervous disturbances, while others will exhibit the most alarming perturbations under a much lower temperature. The amount and intensity of the poison and power of resistance may have much to do with the degree and extent of the pyrexial phenomena. Defervescence is slow and gradual, with increasing remissions and shortening exacerbations. If the normal is not reached with the beginning of desquamation, some complication may be suspected.

The pulse ranges high from the beginning, and continues so with corresponding increase in frequency with the rise of the temperature, sometimes reaching 160, or more, per minute. It diminishes in rapidity, but not correspondingly, with the fall of the temperature. Usually it continues fast until convalescence is established.

During the continuance of the fever the urine is scanty and high-colored. Total suppression is rare, but in high grades of pyrexia it may occur. Marked diminution of the quantity is ominous, and indicates renal complication. Careful observation will frequently detect evidences of renal catarrh in the early stages of the disease, and sometimes at the very beginning. Epithelial débris, mucous casts, and blood-corpuscles, with or without albumen, may indicate an insignificant catarrh, but if such conditions be overlooked or neglected the graver forms of scarlatinal nephritis may rapidly develop and assume a threatening aspect.

The eruption fades and disappears on the different parts of the body in the order of its appearance. Desquamation may begin as early as the third, but usually not before the fifth, day; it sometimes is delayed for a week, and in very rare cases for a longer period. It usually begins about the neck or between the fingers in furfuraceous scales; on other parts of the body it peels off in lamellæ. It is peeled off the palmar and plantar surfaces in long strips. The character and extent of the exfoliations depend greatly upon the intensity of the exanthem and the nature of the skin. When the rash is mild and the skin soft and delicate, the exfoliation is branny and moderate; when severe, it is lamellar and very abundant. In some a second, and in occasional instances a third, desquamation takes place. The process continues for a week or ten days, and with its completion convalescence is usually established. The skin is soft and clean, the throat-affection has subsided, the tongue has resumed its normal appearance, the bowels and urinary secretion are healthy, the appetite has returned, and the patient solicits release from confinement and isolation. The patient is not, however, free from danger even when convalescence has been established. Some one of its numerous complications or sequelæ may unexpectedly interrupt the progress of recovery. It is not safe to discharge a patient from observation and control until six or eight weeks have elapsed.

Such is a brief description of the ordinary course of the regular form of the disease. But there are very many exceptions to this general rul Trivial complications aggravate the symptoms, prolong the course, and dela and protract convalescence. The graver complications increase the sufferin and endanger the life of the patient. The course of the disease is so var able and so easily influenced by intercurrent conditions and irregularities o the usual phenomena that it is not possible always to anticipate unfavorab changes or complications. A guarded prognosis should be the rule.

Irregular Form. The irregular form of scarlet fever is most frequentl caused by some pre-existing or coexisting disease, and the irregularities ma refer to the febrile phenomena, extent and intensity of the exanthem, natu and degree of the local affections, constitutional peculiarity, or circumstanc of life. The febrile phenomena are very variable. Total absence is ve rare, but moderate fever throughout the course of the disease is qui common. In many cases the fever continues moderate until some compl cation supervenes, and then a dangerous explosion ensues; in other cas the fever persists without apparent cause after the disappearance of even moderate rash and complete recovery of the throat-affection; and in othe its course is irregular, with marked remissions, and sometimes intermission succeeded by exaggerated exacerbations. In fact, the irregularities are times as surprising as they are inexplicable, but usually the sudden exace bations may be traced to some local affection which has just begun, or t some intercurrent affection which has been aggravated. Oftentimes the are due to some indiscretion of the patient or nurse. Sometimes the irreg ular course of the fever is due to irregularities in the development an progress of the throat-affection; in other cases, to the intensity of the infe tion and to blood-poisoning. Intercurrent nervous attacks are quite ofte due to irregularities in the course of the fever.

Variations in extent, intensity, and duration of the exanthem are commo in the irregular form. It may be so indistinct as to be scarcely recognizabl or limited to circumscribed regions, as in the joint-flexures, or appear an disappear in a brief period, or not appear at all, or be delayed in appea ance, and may vary in extent and intensity from a mild and discrete pun tate rash to an intensely hyperæmic and confluent eruption, attended wit a tense, swollen, hot, and burning skin, exudations, and extravasation Delayed eruption with high fever is a very grave condition. When th course of the disease is modified by a pre-existing entero-colitis the ras is delayed; and it is usually modified by intercurrent attacks of intestin disturbances. The danger of these irregularities mainly refers to the sup pression and intensity of the exanthem.

Irregularities of the angina are very frequent. Instead of declinin with the disappearance of the rash, it may become worse. Suppuration an gangrene may take place. Diphtheria may set in at any time, either durin the continuance of the angina or after it has subsided. The implication o the lymphatic and glandular structures in close proximity may persist an progress to the formation of abscesses.

The regular may be transformed into the irregular form at any stage by the development or aggravation of a pre-existing local affection. It quite often happens that a case will pursue a regular course for a time and then suddenly assume an irregular and graver form. In rare instances this will occur independently of any local affection, and is probably due to some constitutional peculiarity.

Malignant Form. This form of scarlet fever is, fortunately, not so common as the others. In some epidemics more cases occur than in others. It refers especially to the combination of dangerous nervous phenomena with hyperpyrexia. Its beginning is explosive. The initial symptoms are intense headache, high fever, delirium, sometimes coma, which continue without abatement for one, two, or perhaps four days, when death takes place. A case may begin with a convulsion, followed by coma and death within twenty-four hours, before the appearance of the rash. In some cases the type of malignancy is due to early and grave renal complications. Delirium, excitement, mania, jactitation, convulsions, coma, are the ordinary phenomena, and death the usual result, of this form. In some cases it is ascribed to the delay of the exanthem; in many, to some pre-existing disease; in others, to the virulence of the contagion; and in some, probably, to an intensified predisposition.

Complications and Sequelæ.—The complications and sequelæ of scarlet fever are too numerous to be considered here *in extenso*. To some only a brief reference can be made. The more common and graver conditions will be more fully discussed. Complications may occur in any form and in any case. They are more frequent and serious in the malignant form.

In the section on pathological anatomy reference has been made to the graver forms of angina and tonsillar inflammation and to the suppurative and gangrenous conditions which may ensue. Aggravated angina, attended with ulceration and sloughing, is a very frequent, serious, and sometimes fatal complication. In such cases the inflammation usually extends to the glandular structures and connective tissue about the neck, which, as a rule, progresses to suppuration, and perhaps to sloughing, with extensive destruction of tissue. When the external inflammation is not directly connected with the throat-affection, or is independent of any angina, it usually terminates by resolution, unless associated with a low vitality and great exhaustion. In rare cases cancrum oris occurs.

Few cases escape a mild coryza. Occasionally it proves serious and extends along the Eustachian tube into the tympanum and sets up a painful and troublesome otitis. If the nasal discharge should be fetid, diphtheria may be suspected. Diphtheria is usually a fatal complication. When it occurs as a sequel, recovery may be anticipated.

Bronchitis and pneumonia are not so frequent as inflammations of the serous membranes. Pleurisy, peritonitis, pericarditis, and endocarditis are rare but grave complications. They usually set in during the second week of the disease. Pleurisy and pericarditis are generally associated with joint-inflammations, which ordinarily follow the course of rheumatism. Purulent arthritis is rare.

Disturbances of the alimentary tract are quite common. Vomiting in the beginning of the disease is not serious, but when persistent or recurring during the progress of the disease produces rapid exhaustion and emaciation. Diarrhœa is not often severe, and is usually associated with vomiting. Follicular inflammation and entero-colitis are much more serious but not frequent complications. When the stools are frequent, large, and bloody, with colicky pains and tenesmus, collapse may speedily occur.

A mild conjunctivitis is quite common during the eruptive stage. More serious and protracted ophthalmias occur during the later stages, and sometimes lead to grave corneal troubles.

Otitis may be a complication or a sequel. The lining membrane of the Eustachian tubes is in direct continuity with the mucous membrane of the pharynx and continuously exposed to an extension of the inflammatory process present in the latter region. Ear-troubles usually begin during the later stages or convalescence of the disease, with earache. The lining membrane of one or both tubes becomes inflamed and swollen, so that the products of the inflamed membrane are pent up. The effusion increases, becomes purulent, fills the tympanic cavity, distends the drum, and may penetrate into the mastoid cells. The pain produced by the accumulation and distention is very acute. After a variable period of intense suffering, the drum bursts, and a copious discharge of pus takes place through the external meatus. In many cases recovery follows the perforation of the drum and discharge of the accumulated pus. The aperture heals without any serious impairment of hearing. Not so, however, in a few unfortunate cases. Ulceration of the mucous membrane and necrosis of the bony structure may follow. The aperture may be enlarged by complete destruction of the drum, and the ossicles, becoming detached, may be discharged. In such cases the hearing will be seriously impaired, if not totally destroyed. The inflammation may extend to the meninges, followed by convulsions and death. In other rarer instances the ear-complication becomes chronic, terminating finally in total deafness, and perhaps in death.

Convulsions occurring at the outset of the disease are not necessarily an alarming symptom, but when recurring or occurring during the progress of the disease are a very fatal complication. J. Lewis Smith asserts that convulsions occurring after the complete development of the exanthem are uniformly fatal. This may be true when caused by cerebral congestion or the scarlatinous poison; but uræmic convulsions are not necessarily fatal.

Nervous disturbances of variable character, motor and sensory, are not infrequent. Hemiplegia, paraplegia, and paralyses of single nerves have been observed. Neuralgiæ, hyperæsthetic and anæsthetic conditions, epilepsy and hysteria, and a variety of mental disturbances, have followed scarlet fever.

Nephritis.—Nephritis may appear as either a complication or a sequel of scarlet fever, and is as often associated with the mild as with the severe forms. Some recent authors maintain that renal derangement is present in every case; but this extreme view cannot be admitted. It is, however, far more frequent than is generally believed. It is more constantly present in some epidemics than in others, and may begin with the beginning or at any stage of the disease, but most frequently the first symptoms are not observed before the latter half of the first or during the second week. As a sequel it may not appear for several weeks after convalescence. No patient ought to be considered safe until six weeks have elapsed. In some cases it is a febrile phenomenon, but in most cases it is an effect of the scarlatinal poison, and in other instances it is probably the result of indiscretion in diet or of improper exposure. It may appear as a mild albuminuria with moderate diminution of the amount of urine and gradually increase from day to day, or it may set in suddenly with intense headache, stupor, and convulsions, followed by coma and death. In fact, it may be said to be as insidious in its onset, as irregular in its course, and as uncertain in its result as the disease which it so frequently complicates and aggravates.

Scarlatinous nephritis may or may not be a dangerous complication. The danger lies mainly in the failure to discover its presence until grave symptoms appear. Careful observation of the quantity and specific gravity of the urine should be made daily, and chemical and microscopical examination sufficiently often to discover the early symptoms of simple renal catarrh. A diminution in the quantity of urine is usually the first symptom. The albumen may be slight. Dropsy is usually later, but may be one of the early symptoms. It may begin as a slight œdema about the eyes and quickly invade the serous cavities. If the early symptoms of a mild renal catarrh be overlooked or neglected, the complication may very speedily become more serious than the original disease. The urinary secretion may be totally suppressed or very scanty, highly albuminous, with an abundant deposit containing hyaline, epithelial, and granular casts, with epithelial cells and blood-corpuscles in varying quantities. In such cases the course will be marked by rapid increase of the dropsy, more or less headache, sudden elevation or accession of fever, vomiting more or less persistent, and, finally, convulsions, either partial or unilateral, clonic or tonic, and perhaps recurring in rapid succession, followed by coma and death.

Scarlatinal nephritis is not necessarily, even in its graver forms, a fatal complication. If recognized in the beginning it usually yields to prompt and appropriate treatment, but when not discovered until late it becomes a very grave disorder; but no case ought to be dismissed as hopeless until the evidences of renal degeneration beyond restoration are manifest.

Diagnosis.—In a majority of cases, when first seen by the physician the diagnosis may be made at a glance. A characteristic exanthem and angina, or either separately, with a moderate or a high fever, will be sufficient. The prevalence of the disease, or the fact of exposure of a susceptible person to the contagion, is always suggestive, and frequently, in cases of doubt, is sufficient to establish the nature of the malady. Failures in diagnosis do not often occur in the regular form. The difficulties mainly refer to the irregular and malignant forms, and in such cases the circumstances of prevalence and exposure to the contagion constitute important elements. If either or both of these facts are established, a provisional diagnosis may be made under a great variety of symptomatic conditions before the appearance of the exanthem or the development of the characteristic angina. The subsequent history would speedily verify its correctness. Previous exposure of a susceptible person within a reasonable period of time would establish the diagnosis, before the appearance of the rash, in a person taken suddenly, or after several days of indisposition, with a chill or a convulsion, with more or less fever and a mild angina, which might be nothing more than a slight tenderness and redness of the throat. The circumstance of prevalence or exposure would be even more important in cases beginning with a chill, a convulsion, or severe vomiting, followed by more or less fever; in cases beginning with a slight angina with moderate fever; or in cases beginning with an indistinct rash, without either fever or angina, or with a high or moderate fever without any of the usual accompanying phenomena. In all such cases the appearance of the characteristic exanthem or the development of the anginose conditions would establish the diagnosis. In some such cases a roseola or erythema, due to intestinal troubles, may complicate the diagnosis, but the delay of a day or two will usually remove the doubt. In scarlet fever the symptoms are more serious and persistent, and the peculiar condition of the tongue, more or less marked, is present. Erythema is not so widely diffused, spreads in an irregular manner, and is absent from the extremities, neck, and portions of the chest. Roseola is usually unattended with fever or acceleration of the pulse. There are no joint or glandular swellings, and the rash is more likely to appear in circumscribed spots, and resembles measles.

When the doubt arises from scantiness of the exanthem, the angina will rarely be absent, and the tongue will present its usual characteristic appearance. In most cases there will be more or less swelling of the cervical glands. In some such cases the diagnosis cannot be made until desquamation occurs, or, perhaps, symptoms of nephritis appear.

The condition of the tongue, with an eruption, and the occurrence of cervical lymphadenitis or a mild form of nephritis, may occasionally constitute the entire picture. The absence of the scarlatinal eruption from the region of the mouth is often a valuable differential symptom.

Rheumatic conditions or a nephritis with a scarlatinal tongue, desquamation, and swelling of the cervical glands will establish the diagnosis.

A miliary eruption, with or without sudamina, may be differentiated by the absence of the angina, glandular enlargements, and characteristic tongue. It is never so generally diffused over the whole body.

Erysipelas is distinguished by the punctate character of the scarlatinal rash and the vesicular formations with œdema of the connective tissue in erysipelas. In scarlet fever desquamation may occur in places where there was no eruption; in erysipelas it is always limited to the affected part.

It is sometimes very difficult to distinguish scarlet fever from rubeola and measles. In rubeola the rash appears as early on the face as on the neck and trunk, and is short in duration. The scarlet tongue is absent, and the inflammation, if present, is more generally diffused throughout the oral cavity and pharynx.

Measles must be distinguished by the history, the prodromal stage, the coryza, the later appearance of the eruption, the course and character of the fever, peculiarities of the exanthem, and the absence of the special characteristics of scarlet fever.

In malignant forms without an eruption, the diagnosis can be made only by the intensity of the fever and coexisting severe nervous disturbances without other assignable cause. The prevalence of an epidemic or the fact of exposure of the patient to the contagion will be an important aid.

In very many cases of the irregular forms of scarlet fever the diagnosis will be perplexing, but a careful study of the history of the case and close and intelligent observation of the symptoms will almost always enable the physician to reach a correct diagnosis. Sometimes there may be a delay of a day or two; but few, if any, cases occur which do not during their course present phenomena sufficiently characteristic to settle the diagnosis definitely.

Scarlatinous dropsy is easily distinguished by its occurrence in children during or subsequent to an attack of the fever, its acute course, and its usual beginning about the face and subsequent effusion into the serous cavities. When occurring late during convalescence there will be present the evidence or history of desquamation, and probably existing glandular enlargements.

Prognosis.—The prognosis of scarlet fever is as indefinable as the general course and progress of the disease. In the ordinary regular form it is favorable, but the variations are so numerous and the liability to complications is so constant that the mildest case may be suddenly transformed into one of maximum gravity. The most trivial complication may quickly change the whole picture. Constitutional peculiarities, epidemic influences, circumstances of life, intensity of the poison, season, location, density of population, pre-existing disease, age, nursing and care, and diet, are important considerations, and often factors decisively determining a favorable or an unfavorable prognosis. Hyperpyrexia, with a temperature rapidly rising to or continuing above 105° F., with or without the usual nervous disturbances, a fever persisting after the beginning of desquamation, or failing to decline with the fading of the exanthem, or marked by acute exacerbations, is unfavorable. All nervous disturbances and all inflammatory complications increase the danger of the disease. Some are more serious than others. Gangrenous angina very often, and diphtheritic conditions more frequently, prove fatal. Death is almost invariably the result when the diphtheria extends to the larynx and nose. Intense coryza, extensive suppuration of the structures about the neck, typhoid conditions, early appearance of nephritic disturbances, intense albuminuria, scanty urine, early appearance and persistence of dropsical conditions, effusion into the serous cavities, severe and continuous vomiting, dysentery, severe eye- and ear-affections, and intense infection, are unfavorable, and the more so in proportion to the degree and extent, and to the strength and condition of the patient. Pyæmia and septicæmia are usually fatal. Continuous delirium and recurring convulsions are very grave symptoms, and coma is ordinarily the sign of speedy death. Paralytic complications are serious, but not necessarily fatal. An intense exanthem, with a high fever and a rapid pulse, is very grave, and more so when associated with extravasation of blood. Abundant hemorrhagic extravasations, hæmaturia, and evidences of the hemorrhagic diathesis, however mild the associated phenomena may be, must be accepted as signs of an unfavorable prognosis.

No one can assert, either at the beginning or during the progress of any case of scarlet fever, that it will run a regular course and terminate in complete restoration of health. So long as the regular form pursues the ordinary course, and the stages of invasion, development, maximum intensity, and decline of the exanthem, desquamation, and convalescence, are uninterrupted, the prognosis will continuously improve from period to period, and one can, as these successive stages run their course favorably, more confidently, though always guardedly, give assurance of ultimate and complete recovery. But even then it must not be forgotten that sequelæ are not infrequent, and that the patient must at least be held as an invalid until sufficient time has elapsed to justify the positive assurance of cure.

Mortality.—Scarlet fever is the most fatal of the exanthematous diseases. The mortality varies with epidemics and the circumstances of life. In some epidemics it has reached ten per cent. It is more fatal in cities than in the country, and in hospitals than in private practice. In the regular form death is usually due to some complication.

Treatment.—The expectant and symptomatic methods of treatment yield the best results. Various attempts have been made to formulate a specific treatment for scarlet fever, but as yet they have proved to be of little or no value. Recently Illingworth has stated that biniodide of mercury is a specific. Clement Duke gives it in doses varying from one-half to one-twenty-fourth of a grain, and maintains that it arrests fever and prevents desquamation. Shakhovsky asserts that salicylic acid will prevent all complications, such as uræmia, dropsy, diphtheria, anginas, and lymphadenitis, and will remove them when present. He employs the following formula: ℞ Acid. salicylic., gr. xv; aquæ destill. ferrid., ʒii; syrup. aurantii, ʒi. From one to four teaspoonfuls every hour during the daytime, and every two hours by night. To prevent relapses, the mixture must be continued at longer intervals for several days after defervescence. He says that he has succeeded with this method in a large number of cases of malignant type. It is, however, useless when resorted to late, or when there is present severe chronic disease. Moderate cases of the regular form need but little medicine, and the very mild cases none. Isolation, confinement to bed in a properly-heated and well-ventilated room, a simple or fluid diet, and proper care of the bowels may be all that is needed. When two or more are sick in the same house, separate apartments should be provided, and cleanliness and disinfection should be rigidly enforced. The person of the patient should be kept clean, the bed-linen should be changed daily, and all unnecessary hangings and furniture should be removed from the sick-room. Every care should be taken to prevent the dissemination of the contagion. It is as much the duty of the physician to direct and supervise the methods of prevention as it is the duty of the nurse to execute his orders.

If constipation exists, an enema or some mild aperient may be given. Irritating and drastic cathartics should be avoided. Moderate diarrhœa may need nothing more than a restricted diet. If attended with intestinal inflammation and colicky pain, cold abdominal compresses may be found very useful, with such internal medication as is usually applicable in entero-colitis and dysenteric conditions. Vomiting in the beginning will usually cease with complete evacuation of the stomach; but when persistent, pellets of ice, carbonic-acid water, lime-water, either alone or with milk in varying proportions, in small quantities at short intervals, will prove serviceable. Lime-water with creasote is very valuable in irritable conditions of the stomach. Vomiting recurring during the progress of the disease is usually associated with some grave complication, and is best relieved by such treatment as the intercurrent affection may demand.

Restlessness, sleeplessness, and other mild nervous disturbances will frequently yield promptly to bromide of potassium. Convulsions, especially in the early stage, may also be controlled by it. The cerebral and nervous disturbances are so generally associated with the febrile condition that their proper treatment refers to the management of the temperature, and such measures as will reduce the fever will effectually control them.

Fever is the most constant symptom demanding treatment. When the temperature ranges below 102° F., and is unattended with nervous perturbation, but little more than a placebo of the liquid acetate of ammonium is needed. Water should be allowed in reasonable quantity; unnecessary bed-clothing should be removed; and the temperature of the room should not, as a rule, exceed 65° F. When the patient's temperature ranges above 102° F., and especially when associated with other symptoms resulting therefrom, more effective measures should be resorted to. Quinine is a very valuable drug in all grades of fever demanding energetic treatment, and may be given in such doses at short or long intervals as the age of the patient and the range of the temperature will suggest. In hyperpyrexia larger doses at shorter intervals will be required. Quinine, preferably the hydrochlorate, in tonic doses, is also very valuable in all conditions of exhaustion, especially so in suppurative complications and in slow and protracted convalescence.

It may be given either by the mouth or in the form of rectal suppositories. Antifebrin and antipyrin are very efficient and certain agents to produce rapid and decided lessening of the temperature, but are not always safe. Antipyrin is a very popular antipyretic, and is especially indicated in those cases of hyperpyrexia where the temperature is above 105° F. and attended with grave cerebral and nervous symptoms. It should be given with great care and not in large doses. It is better to repeat several small doses than to give one large dose; and when given, the physician should make a timely visit to observe the effect, and not allow any dose to be repeated without such observation as may enable him to decide upon its advisability. If collapse should follow or be threatened, some alcoholic stimulant will be necessary. Small doses of antipyrin are sometimes admissible in cases of moderate fever with high nervous excitement and delirium, but usually in such cases the fever can be controlled by some milder diaphoretic, as quinine or salicylate of sodium.

The reduction of the temperature by the abstraction of heat is a very popular and efficient method of treating scarlet fever. This is accomplished by the employment of cold water, in the form of cold affusion, sponging, cold or graduated bath, bathing of the head, face, and extremities, the cold pack of Currie, or the iced coil to the head. When the temperature does not exceed 102° F., it is not necessary to resort to this method, but in many cases of moderate fever great relief and comfort will be obtained by frequent sponging of the head and face, the effect of which may be increased by the addition of bay rum to the water, which promotes evaporation, and, consequently, the rapid dissipation of heat. When the temperature rises above 103° F., and more especially in the hyperpyretic conditions, with a temperature above 104° and perhaps reaching 106° or 107° F., some one of the more efficient methods of the application of cold water must be employed, such as cold sponging of the entire body frequently repeated, Ziemssen's graduated bath, by immersing the patient in water at 90° F. and gradually reducing it to 80°, the cold pack of Currie, or the application of the ice-coil to the head. The writer has found the last the most efficient and rapid method of reducing very high temperatures. It is perfectly manageable, and easily regulated by adjusting the coil of rubber tubing about the head, with the reservoir filled with water at such temperature as may be deemed necessary, and at such a height above the patient as will produce a rapid or slower current as may be desired. The colder the water and the more rapid the current through the tubing, the more rapid the abstraction of heat. This method has accomplished satisfactory results in cases where antipyrin had proved dangerous because of the collapse following its use. It may be necessary in some cases to associate these methods of rapid reduction of temperature with alcoholic stimulants to avoid exhaustion. If heart-failure is threatened, alcohol, digitalis, quinine, and the carbonate of ammonium may be demanded.

In all conditions of exhaustion, whether caused by continuous high temperature or by other grave complications,—and a protracted convalescence must be considered a complication,—stimulants are necessary. In addition, quinine in tonic doses and digitalis are valuable adjuvants. Digitalis is oftentimes not only necessary but imperatively demanded, and may be given in very decided doses. The carbonate of ammonium would be contra-indicated in cases complicated with stomachal or intestinal disturbances. Alcohol in some form and digitalis are the most available remedies in conditions indicating the employment of cardiac stimulants and tonics.

The condition of the throat demands special attention. Very mild anginæ need but little treatment, but it is never safe to neglect even the mildest forms of throat-affections. In such cases pellets of ice, frequent draughts of carbonic-acid water, gargles of the chlorate of potassium, or powders of the chlorate and white sugar placed on the tongue and allowed to be dissolved in the saliva, may be all that will be needed. In the severer and graver forms spraying will prove the most effective method of treatment. The fluid may be either a simple solution of the chlorate of potassium, or such solution to which carbolic acid, glycerin, and tincture of the chloride of iron have been added. A spraying fluid composed of water, glycerin, chlorate of potassium, and tincture of the chloride of iron, in such proportions as the case may demand, has proved the most generally applicable and efficient treatment. It is necessary to repeat the spraying every one, two, or three hours, according to the condition of the throat. Gangrenous and ulcerative anginæ are to be treated with disinfecting and deodorizing gargles and sprays. Sometimes cauterization of the ulcerated surfaces will be required. When complicated with infiltration and swelling of the glands and connective tissue about the neck, cold compresses will be serviceable during the early, and warm fomentations or poultices during the later, stages. As soon as suppuration can be detected, it should be evacuated by a free incision. In diphtheritic and nasal complications the treatment should conform to the plan ordinarily in use in those diseases.

The internal administration of the chlorate of potassium, either alone or in combination with the tincture of the chloride of iron, is a routine practice of undoubted value in very many cases of scarlet fever. The potassium salt must not, however, be pushed too far, or given in too large quantity, for fear of its injurious effect upon the kidneys.

Eye- and ear-troubles must be treated as similar affections would be under ordinary circumstances.

The inflammatory complications do not require any special treatment beyond that which similar conditions would require as original or primary diseases. It must always be borne in mind, however, that such affections occurring as complications of scarlet fever are always more serious than when attacking a subject otherwise healthy. He is most successful in the management of such cases who is most alert in the recognition of the earliest symptoms of such intercurrent maladies and equally prompt in the application of such treatment as the extent of the disease may require.

In bronchial and pulmonary complications the air of the room should be kept pure.

Rheumatism is usually mild, and needs nothing more than complete rest of the affected joint and the application of some anodyne liniment. Synovitis will demand the treatment usual in such cases.

As a rule, the exanthem needs no further attention than some application to allay the itching. Sponging with tepid water will frequently meet this indication and afford great relief to the patient, while at the same time it will, in a measure, lessen the fever. Some one of the inunctions before referred to may be employed. Some maintain that inunctions diminish the temperature of the body, and with this view, in former years, the daily anointing of the entire body with lard was advised and highly extolled. Vaseline, cacao butter, or pure glycerin is much neater, and equally effective in relieving the burning and itching. In cases where the exanthem is delayed, partially develops, or fades too soon, a warm bath, hot or warm douches, hot poultices containing mustard, or cold affusion followed by warm wrappings, may be employed. If such irregularities of the exanthem are attended with grave symptoms, the most energetic applications will be necessary, together with the internal administration of ammonia and antifebrile medicines.

Nephritis demands special care. It cannot be said that every death attributable to this complication is due to the neglect of the medical attendant, but it is true in much the larger number of cases. All cases in which marked diminution in the quantity of urine occurs, whether or not attended with any other symptom of renal disturbance, demand prompt attention. If simply a febrile phenomenon, the reduction of the temperature and increased allowance of fluid may suffice. Flushing of the kidney by increasing the consumption of some pure drinking-water, such as the Poland, is not only valuable as a remedial measure, but will also to some extent prove preventive of renal complication by dilution of the concentrated urinary filtration.

The danger of scarlatinal nephritis lies mainly in the failure or arrest of the emunctory function of the kidneys, and, consequently, in uræmic toxæmia. The indications, therefore, for treatment must refer to the promotion of elimination by diaphoresis, catharsis, and diuresis. Diaphoresis may be promoted by steam, hot-water, or hot-air baths. The first may be accomplished by enveloping the patient under cover with steam conveyed by rubber tubing from a generator, or by placing around the patient hot bottles or bricks wrapped in moist cloths; the hot-water bath, by immersing the patient in water at the temperature of 90° F. and gradually raising it to 105° or 110° F.; and the hot-air bath, by packing about the patient, under cover, bottles filled with hot water, care being taken to avoid direct contact of the bottles with the skin. These baths may be repeated at longer or shorter intervals, as circumstances may require. Free diaphoresis is the most effective method of elimination in cases of imperative urgency, when the renal function is nearly or totally suppressed. In such cases the hypodermatic use of the hydrochlorate of pilocarpine, or the internal employment of the fluid or solid extract of jaborandi, becomes a valuable and often necessary adjuvant. In addition to the diaphoretic measure, prompt attention should be given to the bowels, and, unless some contra-indication is present, free catharsis should be induced. A saline purgative may be sufficient, but usually the compound jalap powder, in decided doses, will be preferable and more effective. In cases of great urgency, podophyllin, croton oil, or elaterium may be given. Loomis insists that a full dose of calomel will prove very valuable in cases attended with considerable effusion. It not infrequently happens that after a free sweating and purgation the quantity of urine increases very rapidly: nevertheless, it is not wise to omit such measures as may aid in speedy restoration of the renal function. Hot poultices may be applied to the back. Common experience points to acetate of potassium and digitalis as the most efficient diuretics,—the latter especially so when there is present any condition of the heart indicating its use. They may or may not be given in combination, and should be given in doses at such intervals as the urgency of the symptoms may demand. The infusion of digitalis is the most reliable preparation. If the effusion in the pleural or abdominal cavity is serious, paracentesis must be performed. The withdrawal of a portion of the liquid will usually promote the rapid absorption of the remaining portion. In the later stages of scarlatinal nephritis iron and quinine become most valuable remedies. The tincture of the chloride of iron, in the form either of Basham's mixture or the mistura ferri et ammonii acetatis, is especially beneficial in the later and anæmic condition. Uræmic convulsions should be treated by brisk purging and copious sweating, together with the employment of efficient diuretics. The bromide of potassium, chloral hydrate, chloroform by inhalation, or the sulphate of morphia hypodermatically, may be employed to control the convulsions for the time being. Illingworth advises venesection. In dropsical conditions jalap is the best purgative, and iron is the essential tonic.

The diet should be fluid, preferably milk, light broths, and soups. Farinaceous foods may be allowed in limited quantity after the acute stage has passed. A rigid but proper dietary should be adhered to until the disappearance of the renal complication. The patient should be confined to the house, and perhaps to the sick-chamber, and all exposure of the person to sudden and inclement changes of weather should be avoided. Chronic cases should be treated as cases of Bright's disease.

The general management of scarlet fever after the disease has run its ordinary course, terminating with the completion of desquamation, refers to the treatment of the complications and sequelæ, the nutrition of the patient, and the employment of such tonics, especially iron and quinine, and perhaps alcoholic stimulants, as may be necessary to support the patient and obviate fatal exhaustion.

DIPHTHERIA.

By J. LEWIS SMITH, M.D.

Diphtheria is one of the most dreaded, one of the most fatal, and unfortunately one of the most common maladies of childhood. It is believed to be produced by a micro-organism. It is characterized by the occurrence of a grayish-white pellicle upon the mucous surface, or the skin deprived of its protecting epithelium. The specific principle is ordinarily received by the inspiration of infected air, but it is sometimes received by direct contact of infected matter with one of the surfaces not lying in the respiratory tract.

Diphtheria is a disease of antiquity. M. Sanné mentions the following names by which it has been known in different countries and at different periods: ulcus Syriacum, ulcus Ægyptiacum, garrotillo, morbus suffocans, affectus strangulatorius, pestilentis gutturis affectio, pedancho maligna, angina maligna, anginosa passio, mal de gorge gangréneux, ulcère gangréneux, angina polyposa, angine maligne, croup, diphtheritis, diphtheria. These terms expressing the prominent characteristics of diphtheria render it probable that this was the disease alluded to.

It is impossible to state or form a probable conjecture in regard to the time when diphtheria originated, but its origin probably antedated the Christian era. According to Aurelianus, Asclepiades, who lived one hundred years before Christ, scarified the tonsils, and performed laryngotomy for the relief of respiration, and it is supposed that he treated cases of membranous croup, and probably diphtheria. Aretæus, a Greek physician of Cappadocia at the commencement of the Christian era, gave in writings still extant a clear and accurate description of mild and severe diphtheria. After describing what he designates ulcers upon the tonsils "covered with a white, livid, or black concrete product," he adds, "If the malady invades the chest by the trachea, it causes suffocation on the same day. Children up to the age of puberty are most exposed to this disease." He gives, also, a graphic and truthful description of the suffering of the child when the disease extends to the larynx and croup results. Galen, in the second century, apparently alludes to diphtheria, when he describes a fatal disease prevalent at his time, in which fragments of "membranous tunic" are expelled. He states that he is able to determine by the manner in which the fragments are expelled, by coughing or spitting (hawking), whether they are detached from the larynx or the pharynx. Cœlius Aurelianus, a Latin physician, who is supposed by some to have lived in the second century and by others as late as the fifth century, describes a grave angina in which the symptoms which sometimes arise correspond with those in diphtheritic croup and diphtheritic paralysis as observed at the present time. In the fifth century Aëtius of Amida described a disease accompanied by "crusty and pestilential ulcers" sometimes having a whitish and in other instances an ashy or rusty color, and not preceded by a discharge. Aëtius alludes to the hoarseness which he says sometimes supervenes, and is a source of danger up to the seventh day.

From the close of the fifth century until the sixteenth the record of diphtheria is broken. It is probable that during the long period embraced in the Dark Ages, every decade witnessed epidemics of this fatal disease, but if they were observed and recorded the records were lost, the literature of diphtheria sharing the fate of general literature during this time of intellectual darkness.

In the sixteenth century epidemics of diphtheria occurred in various parts of Europe, and clear and unmistakable descriptions of them have been preserved. From the sixteenth century until the present time, diphtheria has continued to be one of the most frequent and fatal of the epidemic diseases upon the European continent, and it is apparently permanently established in its great cities.

It is a remarkable fact that those pestilential diseases which desolate families and communities in modern times originated in the Eastern hemisphere, chiefly in Asia or Africa, and extended to the Western nations through commerce or navigation. The aborigines of America had in their primitive state no ailments, so far as we can ascertain, except such as occurred from vicissitudes of temperature or were incident to age and their wild and exposed nomadic life. Pernicious to them was the discovery of America by Europeans for various reasons, but especially because it led to the introduction of the contagious and pestilential maladies. The cruel and rapacious gold-hunters under Cortez introduced small-pox into Mexico, and for ages afterwards throughout Central America heaps of skeletons of those who perished of this disease were found in shaded and out-of-the-way localities where they had been taken by their friends. Adventurers from the Old World introduced the eruptive fevers, and the loathsome contagious diseases of vice and immorality, into the islands and upon the continent of North America. The medicine-men of the Indians had by their incantations gained great repute in the management of the diseases with which they were familiar in their wild life in the forests, but they were unable to cope with the new diseases which the vessels of the foreigner had brought to this Western world.

Of all the diseases which America has received from Europe, the one most dreaded, because of its highly contagious character, the great mortality which attends it, and the extreme suffering which certain forms of it produce, is diphtheria. It is to be, from appearance, above all other maladies the scourge of America in the future. It is probable that the first cases of diphtheria in America occurred in or near Boston. Josselyn made two voyages to New England in 1638 and 1663, remaining eight years in this country after his second arrival. He states that the Europeans residing in New England are greatly afflicted by a disease "which hath proved mortal to some in a very short time, quinsies and impostumations of the almonds, with great distempers of colds."[1] At Roxbury, Massachusetts, in 1659, three children in a family were attacked by the "malady of bladders in the windpipe," all dying within two weeks.[2]

At the close of the seventeenth century and in the first half of the eighteenth century epidemics of diphtheria occurred in various parts of New England. At Kingston, New Hampshire, in March, 1735, a child died of three days' sickness of a throat-affection. A week subsequently, three children in another family, four miles distant from the first case, also died of a three days' sickness. The malady continued to spread, and the first forty cases all perished. They died of a disease located in the throat, neck, and air-passages, attended in many of them by swelling of the cheek or neck. The disease from Kingston spread to other townships, but in its subsequent course it was milder than at first. We recognize in this nameless disease the characteristics of diphtheria.

In August, 1735, in Boston, a child had a disease of the fauces attended by white spots. In the following month several similar cases occurred in different parts of Boston. In October of the same year a young man, lately arrived from Exeter, New Hampshire, where a brother had died of this new disease, himself sickened with it, in a more severe form than had yet occurred in Boston. Diphtheria thus established in Boston was epidemic during the following winter and spring months. At the height of the epidemic, in the second week of March, 1736, the burials increased from an average of ten to twenty-four, through the prevalence and severity of the new disease. Two years later,—1738,—a monograph appeared from the pen of I. Dickinson, A.M., Boston, bearing the title, "Observations on that Terrible Disease vulgarly called the Throat Distemper, with Advices as to the Method of Cure, in a letter to a friend." The writer of this epistle, though a clergyman, appears to have been a close observer. He probably, as was not unusual at that period, practised both as physician and clergyman. Dickinson's graphic description shows that the disease in his day presented the same characteristics as at present.

Diphtheria thus established in eastern New England spread westward through the intercourse of the inhabitants, reaching New York in about two years. Dr. Cadwallader Colden, writing in 1753, had already carefully observed diphtheria. He remarks, "When the disease first appeared it was treated in the usual way for a common angina, and no plague was more destructive. . . . The orifices made by the lancet in bleeding, and the adjacent parts, were apt to become diseased; so likewise the places where blisters were applied." He recognized the fact, now well known, that in rare instances the throat remains unaffected, while the diphtheritic inflammation and exudate appear upon other surfaces: "A girl about ten years of age, while the throat-distemper was prevailing, had sores on her private parts, like those on the tonsils of others, but no symptom of the disorder appeared in her throat." Dr. Jacob Ogden, writing from Jamaica, Long Island, in 1769, and again in 1774,[1] described diphtheria as it occurred in his practice and in the adjacent townships. He recommended the use of senega and calomel. But the American physician of this period whose writings contributed most to a correct understanding of diphtheria was Samuel Bard, of New York (1771). He possessed a mind admirably qualified for scientific investigations, and especially for study of an obscure disease, basing his opinions upon accurate clinical examinations. A recent appreciative reviewer, Dr. John C. Peters, says, "Bard's article is among the calmest, wisest, and most accurate that has ever been written on diphtheria, both before and since his time." He recognized the fact that the various forms of diphtheritic inflammation were identical in nature, and, however differing in appearances, had the same underlying cause.

---

[1] Wm. Veazie, Boston, 1865.

[2] Historical Researches of Dr. Elsworth Eliot.

In the first half of the present century diphtheria was regarded as a very important disease in Europe, and was made the subject of investigation by the most renowned clinical teachers, among whom we may mention Jurine (1807), Bretonneau (1821), Bourgeoise (1823), Gendron (1825), Billard (1826), Bland (1827), Blanquin (1828), Broussais (1829), Trousseau (1830), Cheyne (1833), Fricout and Burley (1836), Boudet (1842), Guersant and Blache (1844), Moland (1845), Damot (1846), and Heine (1849). During this half-century, ending with 1850, which witnessed such an augmentation of the literature of diphtheria in Europe, this disease attracted but little attention in America. It appears to have been much less prevalent on this continent than in the Old World. It may have occurred in small epidemics in various localities from the time of Dr. Bard until 1850, but they attracted so little notice from American physicians that no monograph or communication to medical journals relating to diphtheria, which was worthy of preservation, appeared during this long period.

Since 1850, epidemics of diphtheria have occurred in numerous localities in North America, not only in the cities with their sewers and crowded tenement-houses rendering the air impure, but also in the sparsely-settled and mountainous sections, where no impurities in the air exist. But diphtheria is most prevalent and fatal in the cities. During the last quarter-century it has become established in most of the larger cities in the Northern and Western States from the Atlantic to the Pacific coast, along the line of commerce and travel. The permanent establishment of diphtheria in the centres of trade and travel, and the fact that many have this subtle malady in so mild a form that they are not aware of it and mingle with others in places of resort, inevitably tend to disseminate the disease throughout the country. Hence in rural localities intervening between the cities outbreaks of diphtheria of unknown origin are common, in at least all the eastern, northern, central, and western portions of the United States and in Canada. Consequently in the last two decades in America diphtheria has been the subject of discussion at numerous meetings of medical societies, many cases of interest have been reported, and histories of epidemics and statistics of treatment have been published in the medical journals. Therefore the American literature of diphtheria is abundant and rapidly accumulating, and to the genius and perseverance of an American (O'Dwyer) the world is indebted for the means of combating more successfully than in former times the most painful, most dreaded, and most fatal form of diphtheritic inflammation.

---

[1] See Medical Report, vol. v., 1802.

In Europe, diphtheria is established in the centres of medical education, as Paris, Berlin, London, and more recently Vienna. It has in these cities and in smaller cities and towns, where it has occurred, been the subject of much discussion and investigation. In Europe, therefore, as well as in America, the literature of diphtheria has been greatly increased during the last decade, by reports of cases, histories of epidemics, and statistics of treatment. In six consecutive months in 1888 the deaths from diphtheria in ten of the principal cities of Europe were as follows:

	DEATHS.	POPULATION.		DEATHS.	POPULATION.
Paris	1047	2,260,945	Buda-Pesth	207	442,787
London	852	4,282,921	Copenhagen	210	300,000
Berlin	523	1,414,980	Christiania	196	135,600
St. Petersburg	341	928,016	Prague	161	300,828
Vienna	251	1,212,232	Amsterdam	136	390,016

Bull. Gén. de Thér., October 30, 1888.

In Madrid, with a population increasing from 136,663 in 1880 to 157,965 in 1885, the deaths from diphtheria during the six years ending with 1885 were as follows:

In 1880	242	In 1883	1027
" 1881	799	" 1884	1079
" 1882	587	" 1885	1350

La Higiene, October, 1888.

Among the American physicians who have recently advanced our knowledge of diphtheria are Drs. Curtis and Satterthwaite, of New York, in their Report on the Pathology of Diphtheria made to the New York Board of Health, Drs. Wood and Formad, of Philadelphia, in their Memoir on the Nature of Diphtheria, prepared for and published by the National Board of Health in 1882, and Dr. A. Jacobi, in his treatise on diphtheria (W. Wood

& Co., 1880). In Europe during the same period interesting and instructive monographs have been published by Peters, Birch-Hirschfeld, Rosenbach, Leyden, Wagner, Fürbringer, Fischl, Weigert, Meyer, and others.

Etiology.—During the last twenty years numerous experiments and microscopic examinations have been made in order to elucidate the cause and nature of diphtheria. Each year of investigation has strengthened the belief that the cause is a microbe, but it is still a matter of doubt which microbe is the causal agent, or whether there may not be more than one species of bacteria which by their action upon and in the tissues produce diphtheria.

Between the years 1868 and 1873, many of the leading pathologists of Europe believed that the cause of diphtheria had been discovered,—that it was the micrococcus or spherical bacterium. During the decade commencing with 1868 no subject in pathology attracted so much attention as the relation of the micrococcus to diphtheria. Oertel (1868) discovered micrococci in the diphtheritic pseudo-membrane and in the blood, lymphatic vessels, and kidneys in severe diphtheria, appearing as "point-like, dark-contoured, round or oval little bodies, isolated and in zoogloea." In later investigations (1874) he found a larger or smaller number of the bacterium termo accompanying the micrococcus, and he expresses more firmly the belief that micrococci lodging on the mucous surface cause the diphtheritic inflammation. He produced croup in rabbits by applying ammonia, and found few or no micrococci in the false membrane, and never in the blood or internal organs. He inoculated the trachea of rabbits, pigeons, and chickens with the diphtheritic membrane, and produced local lesions apparently identical with those of diphtheria in man, and the blood of the animals subjected to the experiment contained micrococci in abundance. Nassilof[1] states as the result of his observations that fungi, not designating the species, are always present in diphtheritic membranes and precede their development, and that they penetrate the tissues by the blood-vessels and lymphatics before any observable change occurs in the tissues. Therefore he believes that they cause the diphtheritic inflammation. Hueter and Tommasi inserted particles of the diphtheritic membrane in the back of the rabbit. Death occurred in forty hours. Micrococci were found at the seat of the injury, and, before death, in the blood of the animal. Similar experiments and observations made by other pathologists of renown strengthened the belief that the cause of diphtheria had at last been discovered in the micrococcus. Cohn (1872 and 1873) classified this organism, which had now assumed great importance, with the schizophytes, tribe sphæro-bacteria, and he designated it *micrococcus diphtheriticus*.

On the other hand, Eberth (1872) and Klebs (1871) expressed the opinion that the diphtheritic micrococci are the same as septic micrococci. Senator (1874) states that other diseases of the mouth and pharynx are accompanied by the same micrococci as those in diphtheria. They are also, he says, found in the mucus between the teeth, and in normal urine, and the micrococci of diphtheria do not differ in cultures from those occurring in other conditions. Billroth (1874) also dissented from the opinion that micrococci caused diphtheria. He made the broad statement that "the so-called pathogenic bacteria of diseases are positively identical with those found in putrefying dead tissues." Therefore the theory that micrococci alighting upon one of the surfaces caused diphtheria met with strong opposition soon after it was announced, and, as time went on, facts and observations which militated against it multiplied.

---

[1] Virchow's Archiv, 1870.

In 1877, Drs. Curtis and Satterthwaite were employed by the New York Health Board to investigate the etiology and pathology of diphtheria. After many experiments, they reported "that the bacteria of diphtheritic membranes do not differ in optical or chemical behavior from those found in putrescent but non-diphtheritic animal material." They also found that "scrapings from the upper surface of a somewhat furred tongue from a healthy person" cause, when inserted in the cellular tissue of the rabbit, an effect exactly similar to that produced by inoculations with diphtheritic membrane. Putrid Cohn's fluid (an aqueous solution of ammonic tartrate, potassic and calcic phosphates, and magnesic sulphate) also caused the same result. They were enabled, after many carefully-conducted experiments, to enunciate the following propositions: "Thorough trituration of proven virulent diphtheritic membrane and tongue-scrapings with a high percentage of salicylic acid fails not only to remove, but even markedly to modify, the intensity of the infectious quality of those substances. Hence, since salicylic acid in even a minute percentage is capable of permanently suspending the vital activity of bacteria, the inference is that the infectious quality of diphtheritic membrane upon the system of the rabbit is not correlated to the vital activity of the bacteria present in such membrane." Therefore if, as is probable, the agent in the pseudo-membrane which causes the noxious effects in the inoculated rabbit be the same as that which causes diphtheria in man, it follows "that *there is no theoretical ground for assuming that preventing the bacteria of a diphtheritic patch from making their way through the underlying mucous membrane will, per se, prevent general diphtheritic infection of the system.*"

These important observations and opinions, expressed by Curtis and Satterthwaite in 1877, evidently prepared the way for the theory that the bacteria themselves are not the cause or the infectious principle of diphtheria, but chemical substances or ptomaines, produced by the agency of the bacteria, maybe.

In 1882, Drs. Wood and Formad, employed by the National Board of Health to investigate the nature of diphtheria, after many microscopic examinations and experiments, declared their belief that the micrococcus diphtheriticus and m. septicus, inasmuch as they responded alike to optical, chemical, and vital tests, are identical. They found the same micrococcus in the unhealthy pus of erysipelatous cellulitis, and in twenty-one instances in which death resulted from inoculations with this pus they found the same micrococci in the blood of the victims. The blood of twenty-two cases of erysipelas was examined for micrococci, with the following result: "In thirteen of these the organisms were found in the blood, whilst in the other nine there were none. Of measles, twenty-nine cases were studied: in six only were micrococci detected, whilst in eight cases of rötheln or German measles there were no organisms. We have also investigated four cases of malignant fatal scarlet fever, in all of which we found the blood a few hours before death loaded with micrococci, both free attacking the white corpuscles, and in zooglœa masses, and in one of which micrococci emboli were abundant in the kidneys. We have also studied four cases of 'puerperal fever,' probably septic metritis, in all of which micrococci existed in the blood before death."

It soon became apparent to pathologists, from experiments and observations like the above, that the so-called micrococcus diphtheriticus is not peculiar to diphtheria, that it occurs in all pestilential and putrid diseases, in decomposing animal tissues in various diseases, and even upon the tongue and gums in health. Hence it was necessary to look elsewhere for the cause of diphtheria.

In 1883, Klebs made extended and thorough examinations of the microbes of diphtheria, and formed the opinion that a bacillus which he had observed in the pseudo-membrane and upon the inflamed tissue merited special attention. Subsequently Loeffler pursued the investigation, and the organism known as the Klebs-Loeffler bacillus became a prominent object of study as perhaps the causal agent in diphtheria. Loeffler, in the published statement of his investigations, remarks that all observers have found bacteria in the diphtheritic exudate, micrococci most frequently, existing in colonies, and especially abundant in superficial portions of the pseudo-membrane. At times bacteria have been found in the lymphatics in the vicinity of the inflamed tissues. Every diphtheritic patch contains many species of bacteria which have been cultivated; but, as they have not been isolated, the specific germ of diphtheria has not been determined. The rejection of the theory that micrococci are the causal agent of diphtheria, on the ground that they occur, presenting the same optical, chemical, and vital characteristics, in other distinct diseases and conditions, led to a more careful examination of other bacteria present in the diphtheritic exudate and upon and in the underlying tissues. The bacillus described by Klebs and later by Loeffler is motionless, partly straight, partly curved, of the length of the tubercle-bacillus, but double its thickness. It is abundant in the pseudo-membrane, but is not found in the blood-vessels, lymphatics, or internal organs: so that its pathogenic action must be localized on the surface. If it be the specific principle or germ of diphtheria, it must act by producing a ptomaine or chemical poison where it is lodged, which poison entering the lymphatics and blood-vessels causes systemic infection. In some typical cases of diphtheria Loeffler was unable to find the bacillus, which of course militates against the theory that it is the specific germ; but he suggests that it might have died and been eliminated before the death of the patients. Such an explanation seems very improbable; it is making a stubborn antagonistic fact yield to a theory; and yet without such an explanation we must look for some other cause of diphtheria. The Klebs-Loeffler bacillus was found by Loeffler in the exudate in thirteen cases of diphtheria, and cultures to the twenty-fifth generation inoculated in guinea-pigs and birds caused a whitish exudation at the point of inoculation.

W. Watson Cheyne[1] recognizes the importance of Klebs and Loeffler's researches, and thinks it probable that the micro-organism which causes diphtheria is a bacillus, which lodging upon the surface of the throat is propagated there. Having upon the mucous membrane a favorable nidus, it not only lies upon but penetrates the superficial portion of the mucous layer, and causes the exudation of fibrin. The pseudo-membrane thus produced consists, according to Cheyne, of the fibrinous exudate and dead epithelial cells. As the bacilli multiply and extend, the exudate enlarges. Cheyne believes it probable, though demonstration is lacking, that the bacilli cause very poisonous ptomaines, which, entering the lymphatics and the blood, give rise to systemic infection and render the disease constitutional.

But since the observations of Klebs, Loeffler, and Cheyne, the bacillus which they consider the specific principle of diphtheria has been subjected to a more thorough examination, with the result of apparently demonstrating that the same bacillus occurs in non-diphtheritic cases, and even in healthy persons, as well as in diphtheria. Thus, Von Hofman-Wellenhof[2] detected this bacillus in twenty-six of forty-five cases in various conditions of the buccal and faucial surfaces. He discovered it in seven cases of diphtheria, in three of measles, in six of nineteen cases of scarlet fever, and in four of eleven normal cases. In cultures and experiments, the bacilli from different sources appeared to be identical. Therefore in the light of recent investigations the Klebs-Loeffler bacillus has no more significance as a cause of diphtheria than the micrococcus of Oertel.

Prof. Oertel, who was one of the earliest advocates of the theory of the microbic origin of diphtheria, and whose monograph, in 1868, published in "Ziemssen's Cyclopædia," led to the belief in the profession that the micrococcus was the cause, now admits that the theory that diphtheria is due to the action of bacteria, though plausible, is not proved. He has endeavored to elucidate the pathogeny of the disease by a careful and minute study of its anatomical characters.[3] After an elaborate study of its histology, he remarks, "In the earliest-formed membranes many varieties of microbes can be isolated; but practically there are two chief kinds,—chain-forming cocci (streptococcus) and rod-shaped bacteria with rounded extremities (bacilli)."[1]

---

[1] Brit. Med. Jour.

[2] Wiener Med. Wochenschr., 1888, Nr. 3 und 4.

[3] Die Pathogenese d. epidemischen Diphtherie, nach ihrer histologischen Begründung, Leipzig, 1887.

Oertel remarks that in the septic form of diphtheria the cocci are abundant. In a pseudo-membrane of twelve hours' continuance, micrococci abounded mostly on the surface, but in the fibrinous net-work the bacilli, often in colonies, preponderated. In a specimen of twenty-four hours' duration, the upper surface was full of cocci, and between them were bacilli. In another specimen of membrane detached after six days, these two forms of microbes were also intermixed. As regards the tissues and organs, the micrococci and bacilli occurred upon the mucous membranes, not penetrating them to any great depth. They were not found in the "necrobiotic foci," nor were they observed in any of the sections of the kidneys, which were examined. This is a noteworthy fact, because in the examinations made between 1865 and 1871, the results of which were published in Oertel's article in "Ziemssen's Cyclopædia," micrococci were found in the kidneys. He attributes their presence in the kidneys during this period to the fact that the cases under observation were septic, whereas in those recently examined septic infection was not common, on account, he thinks, of the employment of disinfecting and antiseptic measures in place of the escharotic treatment, and forcible detachment of the membrane, in use during the time of his former observations.

The purpose of Oertel in his recent investigations has been to ascertain, if possible, the nature of the diphtheritic virus by a close and minute study of the lesions, or anatomical changes, which it produces. It appears from his examinations that the primary lesion is cell-change. "Necrobiotic processes" and "necrobiotic areas" commencing in the cells are observed in the tonsils, the mucous membrane of the fauces, uvula, epiglottis, larynx, trachea, in the cervical, submaxillary, bronchial, and mesenteric glands, in the spleen, and in the follicles and agminate glands in the intestines. In different cases these structural changes vary, according to the intensity of the virus and the duration of its action. The morbific process extends by propagation through an organ, or from one part to another, the virus being carried by the lymph-stream or blood, disintegrating products being the carrier.

The following is a summary of Oertel's views in regard to the virus of diphtheria. They express all that is at present known of the etiology of this disease. The nature of the virus, says Oertel, is still obscure. It acts upon cells, causing their death and disintegration, and the infected particles convey the virus to other cells. The virus causes hyaline degeneration in the tissues. The hyaline degeneration in the walls of the blood-vessels causes them to rupture, producing hemorrhages. The unequal amount of hyaline change in different parts of the vascular apparatus may be attributed to difference in resisting power, or unequal exposure to the infected blood. Secondary inflammatory processes in the lungs, heart, liver, kidneys, and in the central and peripheral nerve-tissues, must arise from the infectious property of the blood circulating in them. After enumerating at length and with much detail the results of his examinations, Oertel expresses the opinion that bacterial organisms cause diphtheria, and that they produce this result not by their direct action, but by producing a ptomaine which infects the system and causes the disease to be constitutional. The microbe itself is mostly confined to the surface, whereas the action of the virus is "wide-spread and deep." The most eminent pathologists of the present time do not express any more positive opinions in reference to the specific principle or germ of diphtheria than is contained in the above summary of Oertel's views.

---

[1] London Lancet, March 31, 1888.

Dr. Prudden has recently made systematic studies on a series of cases of diphtheria which would seem to indicate that a streptococcus which is almost constantly present in the pseudo-membrane probably stands in a causative relation to the disease.[1]

At a recent meeting of the London Epidemiological Society, Dr. M. W. Taylor[2] expressed the opinion that common mould might sustain a causal relation to diphtheria. The walls of a sleeping-apartment became wet and sodden on July 12. On the 22d a fungus appeared on the plaster, and in the beginning of August the three children who occupied the room, and who had not been exposed in any other way, so far as could be ascertained, sickened with diphtheria. The aspergillus and coprinus grew abundantly in the mould. In another instance, in which the child died in three days, there was a great development of penicillum moulds. A young man had diphtheria severely four days after cleaning out a pigeon-loft where the exuviæ, débris, and rotten wood were covered with mould. But the theory that organisms which are commonly present in ordinary mould can produce diphtheria is improbable, for mould is common in all damp localities, where there is no diphtheria as well as where diphtheria is present. We shall see in our remarks on the propagation of diphtheria that there can be little doubt that pigeons and other feathered animals frequently have this disease, and in the instance referred to by Dr. Taylor it is probable that the exuviæ and débris in the pigeon-loft had been infected by sick pigeons. The specific principle must be introduced from without, but if it obtain a lodgement upon the wet and mouldy surface of any filthy accumulation it may find there a nidus favorable for its development. We shall see that the fact appears to be fully established that the diphtheritic virus is frequently propagated in foul and damp localities, apart from the animal tissues and independently of the sick. We repeat, therefore, that the theory in reference to the causation of diphtheria which is gaining acceptance throughout the world is that it is produced by a microbe or microbes, whose action is chiefly on the surface or at no great depth, and that blood-poisoning occurs mainly from a ptomaine or ptomaines produced by microbic agency. In order to obtain a knowledge of the ptomaine, chemistry must aid microscopical investigation.

---

[1] See Amer. Jour. Med. Sci., 1889.

[2] Brit. Med. Jour.

Mode of Propagation.—No fact is better established than that diphtheria does not originate *de novo*. Like the eruptive fevers, it is produced by the reception in or upon some part of the system of the pre-existing specific poison. The extreme contagiousness of diphtheria from person to person is well known : a moment's exposure to the breath of a patient, or in the infected room where he is under treatment or has been weeks or perhaps months previously, has in numberless instances communicated the disease. The virus adheres tenaciously to objects on which it happens to alight. The clothing of a patient, even when the disease is in its mildest form, his bedding, the furniture of his room, and the objects which he handles, may for weeks afterwards communicate the disease, and even when transported to a distance. A child with malignant diphtheria seen by me in consultation apparently contracted it by embracing a school-mate who was in the street for the first time after an attack of diphtheria. In another instance a child was for a brief period in a room where diphtheria had occurred two months previously, and after the usual incubative period sickened with the disease.

Although diphtheria is often contracted in the manner mentioned above, —that is, by exposure to a diphtheritic patient, or to a room or some object infected by such patient,—there is another mode of infection common in the cities. Dr. Sternberg, in his recent Lomb Prize Essay, published by the American Public Health Association, refers to the fact that damp, foul places, such as sewers, cellars, ill-ventilated spaces under floors, where the sun never enters and where refuse collects, afford conditions favorable for the development and propagation of the diphtheritic virus. The virus, whatever its nature, once received may be propagated in such a place for an indefinite time, and, ascending in the vapors which arise from this culture-bed, it is liable to communicate the disease to any one who inhales it. Thus, in New York City prior to 1850, although foul sewers and insanitary conditions existed, there was no diphtheria, but in the decade following 1850 diphtheria was introduced. Its germ made its way into the sewers underground ; and now wherever sewer-gas escapes into the domiciles of this city it carries with it the diphtheritic virus. The amazing vitality and power of propagation of the diphtheritic poison are apparent when we reflect that it permanently infects the filthy but constantly-flowing and constantly-renewed currents of the sewers of a great city. In all the wards and apparently in every street in New York City children are constantly falling sick with this disease, contracted by inhaling sewer-gas, which, notwithstanding "sanitary plumbing," often escapes from unsuspected sources, even in houses constructed with all the modern improvements. It is chiefly by exposure of children to infected sewer-gas which ascends from this widely-extending underground culture-bed, and to walking cases often so mild that there is little or no complaint of the throat or impairment of the general health, that this disease is so prevalent. Most of the contagious diseases of children are quickly detected by characteristic symptoms or appearances, which the most ignorant families are to a certain extent familiar with, but mild diphtheria possesses so few subjective symptoms that it is often not detected or suspected, even in intelligent families who are watchful of their children. Children with mild diphtheria sit among other children in the schools, in the city conveyances, in the churches and dispensaries, and frequently communicate to those who are near them a malignant form of the disease, from which the unfortunate victims quickly perish. The diphtheritic virus is so subtle, and its vitality and power of propagation so great, that when it is established in a sewered city it can probably never be stamped out, as cholera and yellow fever may be, by measures, however stringent and active, employed by Health Boards or by legislative enactments.

Commonly diphtheria is communicated by the inhalation of infected air, the inhalation of air containing the specific principle, whether derived directly from a patient or from some infected inanimate object, as the walls of a room, furniture, apparel, an article of merchandise, or sewer-gas. More rarely diphtheria is communicated by direct contact with some infected solid substance, as a particle of the diphtheritic exudate, muco-purulent secretion from an infected surface, or the blood of a patient. A considerable number of instances has been reported in which instruments infected by use upon a patient, and not properly cleaned and disinfected subsequently, have been the means of communicating the disease. In these instances of communication by direct contact, the poison is received either upon one of the mucous surfaces or upon the skin denuded of its protecting epidermis.

Diphtheria contracted from Animals.—Observations are accumulating which show that diphtheria, or a disease closely resembling it, occurs among animals and is sometimes communicated from them to man. The feathered tribe especially appear to be susceptible to this disease. On the island of Skiathos, off the northeastern coast of Greece, no diphtheria had occurred during at least thirty years previously to 1884, according to Dr. Bild, the medical practitioner of the island. In that year a dozen turkeys were introduced from Salonica. Two of them were sick at the time, and died soon afterwards; the others became affected subsequently, and of the whole number seven died, three recovered, and two were sick at the time of the inquiry. The two had a pseudo-membrane upon the larynx, difficult breathing, and swelling of the glands of the neck. As further evidence that the disease was true diphtheria, one of the turkeys that had survived had paralysis of the feet. The turkeys were in a garden on the north side of the town, and the prevailing winds upon the island are from the north. When this sickness was occurring among the turkeys, an epidemic of diphtheria commenced in the houses in proximity to the garden, and spread through the town. It lasted five months, and of one hundred and twenty-five cases in a population of four thousand, thirty-six died. Diphtheria was from this time established upon the island, and frequent epidemics of it have occurred since.[1] M. Menzies[2] states that diphtheria is common among the poultry in Italy, in which country the flat roofs of the houses afford a resting-place for turkeys, fowls, pigeons, and rabbits, and their evacuations are carried by the rain into the cisterns and wells. A physician at Posilippo, near Naples, had directed his servant not to obtain drinking-water from the well next to his house, but from a well at a distance. As long as he obeyed the instruction his family was well; but, yielding to his indolence, he finally disobeyed the command and obtained water from the infected well. Four of the children, who drank this water, soon took diphtheria and died. The fifth child, who did not drink the water, escaped. In 1878–79, Nicati, of Marseilles, observed cases which seemed to show that diphtheria is sometimes contracted from fowls.[3] The *Journal de Médecine de Paris*, February 19, 1888, contains an instructive paper by Dr. Delthil on the transmission of diphtheria from animals to man, in which a considerable number of apparent instances is related. Dr. F. T. Wheeler[4] states that while in a nesting of wild pigeons he found many sick with a pseudo-membranous sore throat. He dissected many with his pocket-knife, which he was obliged to throw away on account of the offensive odor. There were millions of pigeons in the nesting, and they were hunted and eaten by the inhabitants. The same year diphtheria broke out in a most malignant form, causing many deaths among the children. Several years previously, pigeons nested in the same locality, or near by, and fully half of the children living in the vicinity had diphtheria. Dr. George Turner[5] states that a pigeon was brought to him for dissection. The whole of its windpipe was covered by pseudo-membrane, as in the croup of a child. Pigeons were inoculated in the fauces with this membrane, and a similar disease was produced, which extended to their eyes through the nostrils. An epidemic of diphtheria occurred in the village of Braughing, Hertfordshire, England, the first cases appearing on a farm where the fowls were dying of a disease similar to that in the pigeon; and on other farms where diphtheria appeared it was preceded by a similar disease in the fowls. Dr. Turner also mentions several other epidemics of diphtheria in different localities, where the poultry, turkeys, pigeons, and in one locality the pheasants, perished of a disease attended by this membranous exudation. At Tougham a man bought a chicken at a low price, as it was affected by the prevailing disease, and cared for it at his home. Soon after diphtheria broke out in his family, and this case was the first in the village. Instances are also cited by Dr. Turner showing that cats, sheep, and pigs have suffered from a severe disease of the throat, probably diphtheritic, in several localities where diphtheria was prevailing among children.

According to the observations of various experimenters, diphtheria can be transmitted from man to animals; and, if this be true, it seems probable that it can likewise be transmitted from animals to man. Trendelenburg inoculated sixty-eight rabbits, introducing diphtheritic pseudo-membrane into the trachea through an artificial opening. Eleven of the rabbits died with the symptoms and appearances of diphtheria. In control experiments he introduced various foreign bodies into the larynx of rabbits and was unable to produce any results or lesions resembling those in diphtheria. Oertel performed twelve similar experiments, and five of the rabbits died after the production of pseudo-membranes. Zahn, Gerhard, Labadie-Lagrave, Francotte, and Vulpian obtained similar results from their experiments. Such observations and experiments render it probable that genuine diphtheria, equally fatal and attended by the same anatomical characters and symptoms as in man, does occur in birds, whether wild or domesticated, and in certain quadrupeds, as the rabbit. Neverthless, we should add that certain eminent pathologists, among whom we may mention the honored name of Virchow, have doubted the identity of animal and human diphtheria. With our present light upon the subject, it is evident that, since our relations to the domestic animals are so close, if they are sick with any disease resembling diphtheria the same precautionary measures should be taken to prevent infection of the family as in human diphtheria.

---

[1] Bulletin Méd., January 22, 1888.

[2] Thesis, Paris, 1881.

[3] Marseille Méd., 1879, p. 105.

[4] American Practitioner and News

[5] Journal of Laryngology and Rhinology.

Mr. Cole, a veterinary surgeon of Hinckley, Australia, published in the *Australian Veterinary Journal*, February, 1882, copied into the *New York Medical Record*, the account of an epidemic of diphtheria that was apparently traced to the milk obtained from a diseased cow. In 1879, Mr. W. H. Power, Health Inspector, investigated an outbreak of diphtheria, and believed that he traced it to the milk-supply. The cows which furnished the milk that seemed to communicate the disease had what the veterinary surgeons designate "garget," or "infectious mammitis."[1] Another similar history of an epidemic is related by the same journal that published Mr. Power's report. Prof. Damman, of the Hanover Veterinary School, reported in the *Deutsche Zeitschrift für Thiermedicin*, 1877, an epidemic of what seemed to be diphtheria in calves. He directed the attendant to make applications to the mouths and throats of the affected calves. This was on April 29. On May 5 the attendant became sick, complained of his throat, and was confined to bed. A pseudo-membrane appeared on his tonsils, which were highly inflamed; he had high fever, and enlargement of both the submaxillary and cervical glands. A dairy-maid who now took charge of the calves also had a similar but less severe attack. Milk is a culture-medium of various microbes, and that it may be the medium of communication of diphtheria as well as of scarlet fever seems probable.

The fact that the diphtheritic virus may be conveyed long distances without losing its virulence is now admitted from the many observations that have been made. Prof. C. W. Earle, of Chicago, read before the Ninth International Medical Congress an interesting statistical paper on the occurrence of diphtheria, often severe and fatal, in salubrious rural localities, free from sewage-gas and water-pollution, in the newly-settled and mountainous States and Territories of the Northwest. Dr. Earle's statistics render it probable that the diphtheritic infection is transported long distances to these localities, being carried in articles of clothing and merchandise. The well-known tenacious adherence of the virus to objects renders it highly important that thorough disinfection should be employed before articles are removed from an infected room.

---

[1] Med. Times and Gaz., Jan. 1879.

Age.—Trousseau has said that diphtheria does not spare any age, but is most common between the ages of two and five or six years. Guersant believes that the age of greatest frequency is between the second and seventh years, and Barthez and Rilliet agree with Guersant. Bouillon-Lagrange in sixty-three cases occurring in one epidemic treated—

Under 2 years	14 cases.	From 18 to 30 years	15 cases.
From 2 to 6 "	18 "	" 30 to 40 "	4 "
" 6 to 12 "	10 "	" 40 to 50 "	1 case.
" 12 to 18 "	9 "	Above 50 "	2 cases.

According to M. Barthez, in Sainte-Eugénie Hospital during twenty years the ages of the diphtheritic patients were as follows, adults being excluded from this institution:

Under 1 year	81 cases.	From 6 to 7 years	59 cases.
From 1 to 2 years	314 "	" 7 to 8 "	36 "
" 2 to 3 "	319 "	" 8 to 9 "	24 "
" 3 to 4 "	292 "	" 9 to 15 "	82 "
" 4 to 5 "	200 "	" 15 to 17 "	2 "
" 5 to 6 "	103 "		

Louis has shown that diphtheria may occur at an advanced age; but its occurrence is rare over the age of forty years, and very rare after the age of sixty years.

Oertel says, "In the first half-year the infant organism seems to be not at all susceptible to the disease." As in scarlet fever, so in diphtheria, cases are infrequent under the age of six months; but a considerable number of cases are on record showing that it does occur even in the newly-born. Dr. A. Jacobi has collated the following cases: a child of fourteen days treated by Tigri, one of fifteen days by Bretonneau, one of seventeen days by Bednar, one of eight days by Bouchut, one of seven days by Weikert, several cases by Parrot, and eighteen cases observed by Sirédey in the Hôpital Lariboisière in the spring of 1877. Dr. Jacobi adds, "I have met with three cases of diphtheria of the pharynx and larynx in the newly-born myself. One of these became sick on the ninth day after birth, and died on the thirteenth day; the other died on the sixteenth day after birth; the third was taken when seven days old, and died on the ninth day."[1]

Certain physicians having charge of maternity wards have observed a disease occurring in newly-born infants which bears some resemblance to diphtheria, but which, if it be true diphtheria, presents anomalous features. Thus, Dr. W. S. Bigelow reports in the *Boston Medical and Surgical Journal* for March 11, 1875, ten cases occurring between September and December, 1873, in the Boston Lying-in Asylum, all fatal but two. The prominent symptoms and anatomical characters were dark hue of skin, hæmaturia, pseudo-membranous exudation upon certain mucous surfaces, dark-green stools, spleen enlarged and dark, kidneys engorged, in some of the cases effusion of blood into the pelves of the kidneys and along the urinary tract. Dr. Bigelow refers to what appear to have been similar cases in one of the European asylums. The presence of pseudo-membranous exudations on the mucous surfaces, and renal casts, raises the suspicion that the disease which gave such strong evidence of infectiousness was diphtheria. That, so far as appears from the records, the mothers remained well, does not preclude the belief that the disease of these infants had a diphtheritic origin; for in cases which we will presently relate the mothers with one exception remained well, although their infants a few days old undoubtedly had diphtheria.

---

[1] Treatise on Diphtheria, W. Wood & Co., New York, 1880.

A case in some respects similar to those observed by Dr. Bigelow came under my notice. Malignant diphtheria occurred in a family in West Fifty-Third Street, New York, in October, 1880. The patient, a boy of ten years, died, and the remaining two children, as soon as the nature of the malady was apparent, were sent from the house. Nevertheless, one of these, precisely seven days after the removal, was attacked by diphtheria of the hemorrhagic form, and died in less than a week. Blood escaped from the nostrils, fauces, under the skin in numerous places, causing purpuric spots, and from the kidneys or urinary tract, causing hæmaturia. The mother, who was at this time in the sixth month of pregnancy, continued greatly depressed by the occurrence, although her general health seemed to be good. She had been in constant attendance upon her children. Her infant born three months subsequently to the occurrence of diphtheria in her family (February 6, 1881) was well developed, but it presented a similar hemorrhagic cachexia to that in the second case of diphtheria. Blood escaped from the vessels under the skin, causing blotches and prominences, and from the mucous surfaces. The bleeding was persistent and copious from the umbilicus, so that death occurred in less than a week. The diphtheritic virus is subtle and penetrating, causing the specific inflammation in the uterine walls of the parturient woman, even when her fauces are not affected. Nevertheless, whether diphtheria sustains a causal relation to cases like the above is uncertain, and can be determined only by more numerous observations.

The admitted infrequency of diphtheria in the newly-born, and the statement by some writers that the newly-born have an immunity from it, induce me to relate the following cases, in which the diagnosis of diphtheria was established beyond doubt by carefully-conducted necropsies and microscopic examinations:

The New York Foundling Asylum at Sixty-First Street and Tenth Avenue has during the twenty-three years of its existence been remarkably free from contagious and infectious maladies, but from September 1, 1887, to April, 1888, an epidemic of diphtheria occurred in the institution. During this time five new-born infants had diphtheria, the pseudo-membrane appearing in its usual situation on the pharyngeal, nasal, and laryngo-tracheal surfaces, and in one of the cases also lining the œsophagus.

Case I.—Violet M. was born after normal labor on January 5, 1888, and the umbilicus was dressed with borated cotton. The mother did well, and was able to leave her bed on the seventh or eighth day. Nothing unusual was noticed in the infant until January 11, when a little suppuration was observed in the umbilical fossa, at or around the point of attachment of the cord, and on examination the walls of the umbilicus were found thickened and indurated. The appearance indicated the commencement of an umbilical phlegmon, and the skin covering it was red as in erysipelas. The phlegmon increased until January 14, when the thickening and infiltration extended to the distance of about one and a half inches in every direction from the umbilicus, so that the form of the phlegmon was circular or wheel-shape. The pulse on the 13th varied from 132 to 144, and the rectal temperature was 101.8° F.

On January 14, when the patient was nine days old, we observed for the first time the grayish-white exudate of diphtheria on each side of the fauces, and a day or two later also on the Schneiderian surface, so closing the nostrils that respiration through them was impossible. The baby on attempting to draw the nipple became cyanotic and was obliged to relinquish the hold. During the 14th and 15th the temperature fell to 98.5° and 98° F., the pulse was very feeble and too rapid to be counted accurately, and the respiration varied from 24 to 48. Death occurred on the 15th, at the age of ten days.

The autopsy revealed a diphtheritic pseudo-membrane upon the faucial surface on both sides, extending downward so as to cover both surfaces of the epiglottis, the entrance of the larynx, and the laryngeal surface, completely concealing the vocal cords and the portion of the larynx above them. The trachea and bronchial tubes were free from the exudate. The lungs in every part were thickly mottled with points of extravasated blood. The weather at the time was very cold, and the body in the dead-house was frozen soon after death. Prof. Prudden, of the laboratory of the College of Physicians and Surgeons, discovered and cultivated two forms of microbes in the phlegmon, and in the thrombus that plugged the vein, to wit, the staphylococcus pyogenes aureus and the streptococcus pyogenes.

Case II.—Hilda M., born February 28, 1888, plump and robust, weighed eight pounds and seven ounces. The mother seemed to be well until March 3, when she had fever, apparently due to pelvic cellulitis, probably of septic origin. The infant was fretful on March 3 and 4, and on the 5th a small ulcer was observed in the umbilical fossa. The skin surrounding the umbilicus over an area the size of a silver dollar had a deep-red color, and the tissues underneath constituting the abdominal walls were infiltrated and thickened. The phlegmon extended so that on March 6 it nearly reached the ensiform cartilage above and the pelvis below. The fauces had been daily inspected, and at five P.M. on March 6 the characteristic diphtheritic pellicle was observed for the first time covering the tonsil on each side. On March 7 the exudate had increased, the cry was hoarse, the fingers livid at times, and fluid regurgitated through the nostrils. The phlegmon occupied nearly the entire abdominal wall anteriorly. March 8, surface cyanotic, respiration labored and at times accompanied by the expiratory moan; a diphtheritic pseudo-membrane in the right nostril. Death occurred at 6.30 A.M., March 9, at the age of ten days, on the fifth day of the phlegmon and the third day of the diphtheritic pellicle on the fauces.

Prof. Prudden immediately took charge of the body, and made the autopsy with sterilized instruments, and with all possible precautions to prevent the access of adventitious germs. His report states, "The pharynx, larynx, and trachea showed soft reddish friable patches of diphtheritic membrane, partially covering their free surfaces." This membrane did not extend into the bronchial tubes. Both lower lobes of the lungs were inflamed, broncho-pneumonia with considerable consolidation being present. In examining the umbilicus and the adjacent walls of the hypogastric arteries and the umbilical vein he found them infiltrated with spheroidal bacteria, and in a small pus-cavity at the site of the umbilicus were not only spheroidal bacteria, but a few rod-like microbes. The most abundant species was the staphylococcus pyogenes aureus. Dr. Prudden adds, "The anatomical diagnosis, then, is diphtheria of the pharynx, larynx, and trachea, with double bronchopneumonia, localized septic inflammation of the umbilical vein and hypogastric arteries, and the abdominal wall surrounding them."

Case III.—Olivia G., born January 8, and wet-nursed by its mother, was apparently well until January 14, when she became restless. On the 15th, when she was seven days old, she was carefully examined, and diphtheritic patches were observed on the faucial surface; rectal temperature 100° F., respiration 36, pulse 120. She had commencing nasal catarrh, with the usual infiltration and muco-purulent discharge, which so obstructed the nostrils that she could not take the breast, and she was fed with the mother's milk from a spoon. Probably patches of pseudo-membrane were present in the nostrils, but none were observed upon the visible parts until the 17th, when the characteristic pellicle occluded the right nostril. Daily notes of the case have been preserved, and the symptoms as regards temperature, respiration, pulse, and the cyanosis bore a close resemblance to those in the above cases. Death occurred on the 18th. At the autopsy, in addition to the diphtheritic patches already mentioned occurring upon the faucial and nasal surfaces, a pseudo-membrane was found covering the larynx, trachea, and œsophagus to within one inch of the stomach. No notable change was observed in the appearance of the internal organs, with the exception of numerous points of extravasation in the lungs.

Case IV.—Victor K., born December 7, 1887, appeared to be in usual health until January 13, when at the age of thirty-seven days the mother called the attention of the resident physician, Dr. Davis, to him, as he appeared to be seriously sick. His temperature was 103.2° F., and his breathing indicated acute nasal catarrh. On the following day, the 14th, the grayish-white exudate of diphtheria was observed covering the left side of the uvula. The inability to remove it by the brush or washing demonstrated its diphtheritic nature. His subsequent history resembled those given above. Death occurred on the 15th. At the autopsy no pseudo-membrane was observed except that already described.

Case V.—Vincent B., born December 31, 1887, was well until January 17, 1888, when symptoms of a catarrhal nature attracted attention. The nostrils seemed to be unaffected, but upon the posterior portion of the fauces was a grayish-white patch of the usual diphtheritic appearance. By antiseptic and solvent inhalation this pellicle became smaller, and on the 21st had disappeared. The infant recovered.

Diphtheria of the newly-born is sometimes wrongly diagnosticated. Thus, in the New York Foundling Asylum, where diphtheria was occurring, the tonsils of an infant a few days after birth presented a grayish-white appearance, suspected to be diphtheritic. After its death, the curator, Dr. Northrup, discovered a pultaceous state of the surface of the tonsils, but no pseudo-membrane. The disease was apparently not diphtheritic; but, as regards the cases related above, diphtheria was undoubtedly present in the first three, and there can be little doubt that this was also the disease in the remaining two. The occurrence of these cases in so short a time in a small maternity service shows that under certain circumstances the newly-born infant exhibits considerable susceptibility to diphtheria.

Incubation.—The duration of the incubative stage in experimental inoculation is short, varying from twelve hours to three days. In Trendelenburg's experiments the incubation was mostly from one to three days; in Lagrave's, about twenty hours. In Duchamp's inoculations the animals died after forty-eight hours with the larynx and trachea, upon which the infectious material was applied, covered with pseudo-membrane. Oertel says that the rabbits upon which he experimented by inoculation of the muscles perished in from thirty to thirty-six hours, rarely after forty-two hours, the disease-process extending rapidly to neighboring tissues. When diphtheria is contracted by a child upon an excoriated or wounded surface, as after circumcision, ablation of the tonsils, or upon a leech-bite or a burn, the incubative period is short, but it may be as long as four days. Thus, the *British Medical Journal* and subsequently the *Archives of Pediatrics* published the following interesting case, contributed by Mr. Phillips. A few hours after the performance of tracheotomy for diphtheritic croup, the same instruments were employed for performing circumcision in a child of eighteen months. Four days later a false membrane appeared upon the wound of the prepuce, which by the following day had extended over the glans, with much œdema of the prepuce and retention of urine.

When diphtheria is contracted in the usual manner,—that is, by the inspiration of air containing the specific principle,—the period of incubation appears to be on the average somewhat longer than when it is communicated by direct contact. My observations lead me to believe that when the incubative period is short the disease is likely to be severe, and mild when the incubative period is long. I was enabled to ascertain very nearly the incubative period in the following cases. A boy of nine years was in the same room about one hour on Saturday with a child who had fatal diphtheria. On the following Tuesday, without any other exposure, he sickened with a severe and fatal form of the disease. Mrs. E. assisted in nursing a severe case of diphtheria from November 11 to 13, 1874, after which she returned home, several blocks away. On the evening of the 15th she complained of sore throat, and on the following day the diphtheritic pseudo-membrane was observed upon her tonsils. On the 19th the exudation had disappeared, and she was convalescent. On the 20th her sister, who resided with her, and who had not been elsewhere exposed, was also attacked. The only other case in the family, a boy, sickened with diphtheria on December 2. In the first of these cases the incubative period seems to have been from two to four days, while in the last it was longer. In April, 1876, a little girl died of malignant diphtheria in West Forty-First Street, New York City. Her sister, aged one year, remained with her from April 14 to 17, when she was removed to a distant part of the city and placed in a family where there had been no diphtheria. On April 24, seven days after her removal, this infant was observed to be feverish, and on the following day, when I was called to examine her, the characteristic diphtheritic patch had begun to form over the left tonsil. In April, 1875, two sisters, aged five and seven years, resided with their parents in a boarding-house in West Twenty-Second Street. A playmate in the same house had symptoms which were supposed to be due to a cold, but which were diphtheritic, when one night severe laryngitis occurred, and ended fatally the following day. The physician who had been summoned diagnosticated diphtheria, and the two sisters were immediately removed to a hotel. Seven days subsequently diphtheria commenced in the older child. The younger sister was then removed to a distant part of the same hotel, but six or seven days later she also was attacked. Sanné says that in ninety-eight cases the incubative period appears to have been as follows:

From 1 to 2 days	7 cases.
" 2 to 8 "	48 "
" 8 to 13 "	23 "
From 13 to 15 days	6 cases.
" 15 to 20 "	14 "

But diphtheria is so insidious and the modes of exposure so many that in some of the cases of an apparently long incubation there may have been a second exposure. The above statistics show that the incubative period varies, but is most frequently from two to eight days.

Nature.—Diphtheria resembles scarlet fever in certain particulars: in its incubative period varying from two to eight days, with occasional cases outside these limits, in its variability of type from a very mild to a malignant form, in the common seat of its inflammations,—to wit, upon the fauces and nasal passages,—in the profound prostration and blood-poisoning in the graver cases, and in the frequent occurrence of nephritis as a complication or sequel. It resembles both scarlet fever and small-pox in the fact that it has the twofold mode of communication through the air and by contact or inoculation. It resembles erysipelas in the variableness of its duration, and in the fact that one attack does not prevent the occurrence of another. In its etiology it resembles typhoid fever; for it is not only communicable from person to person, but it is communicated by foul exhalations, as sewer-gas, in which the poison lurks. But, while there are certain resemblances, it is distinguished from all these infectious diseases by marked peculiarities.

Diphtheria is primary or secondary. The secondary form most frequently occurs during epidemics of the other infectious diseases and as a complication of them. Those infectious maladies which are accompanied by inflammation of the fauces and air-passages are most liable to this complication, if they occur in a locality where diphtheria prevails. In these instances of secondary diphtheria the diphtheritic inflammation supervenes upon the inflammations which pertain to the primary diseases. Scarlet fever beyond any other malady appears to furnish the conditions which are most favorable for the occurrence of diphtheria, in the latter part of the first week or in the second week of its continuance. If scarlet fever and diphtheria be epidemic in the same locality, not infrequently towards the close of the first week of the former disease a sudden aggravation of symptoms occurs, and the cause is soon rendered apparent by the appearance of the diphtheritic exudate upon the faucial surface, usually upon its tonsillar portion. The discrimination under these circumstances of the diphtheritic inflammation from a severe scarlatinous angina is to be carefully made, and is sometimes not easy, for the scarlatinous inflammation, if intense, occasionally becomes gangrenous, so as to present an appearance resembling that of a pseudo-membrane. The other infectious maladies which are most liable to the diphtheritic complication are measles, variola, whooping-cough, and typhoid fever, the catarrhal inflammation of these diseases changing to a pseudo-membranous inflammation.

It is an interesting and important fact that when diphtheria is contracted by a person having inflammation of one of the surfaces the specific inflammation with the pseudo-membrane usually occurs upon the part which is already inflamed. A catarrhal inflammation, however produced, is liable under the influence of the virus to become diphtheritic and pseudo-membranous. Thus, at one time diphtheria entered the eye-ward of the New York Foundling Asylum, and three children, who were under treatment for inflammation of the eyelids, were attacked by diphtheritic conjunctivitis, exemplifying the remark by Billroth that "catarrhal conjunctivitis, which is so very common, may become diphtheritic."[1] Catarrhal inflammation from abrasions, burns, wounds however produced, are liable to be attacked by the diphtheritic inflammation and become covered with the pseudo-membrane. In Paris, where diphtheria is very prevalent, the circumcised prepuce has so often become the seat of the diphtheritic exudate that the distinguished surgeon Saint-Germain considers this fact a strong argument in favor of stretching, which he practises instead of circumcision. He also for the same reason among others recommends the treatment of enlarged tonsils by galvano-cautery instead of excision. However, in one instance in which I was employing dilatation of the prepuce, and in which the mucous membrane may have been injured by the operation, a severe diphtheritic inflammation set in on the following day, and extended from the tip of the prepuce to the body, with intense redness and swelling. The tonsils at the same time were inflamed and covered with the membranous exudation. Although severely sick, the patient recovered in a few days.

This general fact in regard to the nature of diphtheria and its mode of manifestation, to wit, that in one affected by it the diphtheritic inflammations appear by preference upon such surfaces as are already inflamed, has an important practical bearing. In frequent instances during epidemics of diphtheria, inflammations which physicians of experience believe to be simple or catarrhal, and have diagnosticated as such to the friends, are seen in a few days to be diphtheritic. The most serious error of this kind, if it be one, is to diagnosticate and treat diphtheritic croup as a simple or catarrhal laryngitis, until the increasing dyspnœa reveals the true nature of the disease. This experience always places the physician in an unfavorable light. But is it not probable that in a certain proportion of such cases the disease was at first a simple catarrhal inflammation, and that it became diphtheritic during its progress, just as scarlatinous angina or rubeolous laryngitis becomes a diphtheritic inflammation in those who contract diphtheria ?

The frequent occurrence of diphtheria in all civilized countries during the last thirty years, and the great mortality which attends it, have awakened an interest in this malady which has led to a careful study of its causes and nature. At first these inquiries were chiefly clinical, but in later years microscopic examinations and experiments on animals have furnished important aid in elucidating the nature of the disease. The importance of these microscopic examinations and experiments cannot be overestimated. In connection with clinical observations, they render highly probable the theory which has been stated above, that diphtheria is produced by micro-organisms, which coming in contact with the mucous membrane, or the cuticle deprived of its epidermis, adhere to it, and, multiplying rapidly, act as an irritant and produce the characteristic inflammation; and the fact that since antiseptic treatment has come into general use, microbes, in at least many instances, have not been found in the blood-vessels, lymphatics, or internal organs, in those who have died of diphtheria, has led to the belief, as we have already remarked under the head of etiology, that the systemic poisoning occurs through the agency of chemical products, or ptomaines, which, produced by microbic action, are absorbed into the system. Whether this theory be entirely true or not will be determined by future investigations. If true, it of course establishes the fact that diphtheria is primarily a local disease. Whether it is primarily local or constitutional has been and is still much discussed. It is sufficient for the wants or purposes of the practising physician to be assured that in all cases, unless of the mildest type, diphtheria, if not primarily constitutional, is attended by systemic blood-poisoning very early, even on the first day, so that in all cases of average severity constitutional as well as local treatment is required. The following facts indicate the early blood-poisoning in diphtheria.

---

[1] Encyc. Pathol., translated, p. 267.

1. It is a law in pathology that those diseases which have or may have a long incubative period—say of a week or more—are constitutional.

2. Another fact, which indicates primary blood-poisoning in diphtheria, is observed in certain cases, namely, the *occurrence of severe constitutional symptoms for a longer or shorter time, perhaps for half a day, before the appearance of the usual inflammation.* Thus, a girl of five years, having malignant diphtheria, whom I saw in consultation, was carefully examined on the first day of her sickness by the attending physician, and, although he closely inspected the fauces, there was no appearance which indicated the nature of the malady till the subsequent day. In such cases, a sufficient number of which I have observed, there is likely to be complaint of soreness of the throat, or difficulty in swallowing, almost from the beginning of the general symptoms; but the pain and tenderness seem to be in the deeper tissues of the neck.

Again, treatment of the inflammations by the most reliable and efficient antiseptics and disinfectants which we possess, commenced at the earliest possible moment and repeated at short intervals, does not prevent the occurrence of indubitable symptoms of blood-poisoning in cases of a severe type. Thus, I have treated every portion of the inflamed surface, so far as it was accessible, every second or third hour, with carbolic acid and other disinfectants, almost from the very commencement of diphtheria, and so thoroughly that any vegetable or animal poison with which the remedies had come in contact would probably have been destroyed or rendered inert, and yet, except in mild cases, symptoms of diphtheritic blood-poisoning have occurred, and as early and uniformly as if less energetic local measures had been employed. While, therefore, I do not fail to recommend local treatment as calculated to diminish septic poisoning and relieve the inflammations, I have lost confidence in it as a means of preventing the entrance of the diphtheritic poison into the blood. Its powerlessness to prevent contamination of the blood by the diphtheritic virus is an additional evidence that this contamination occurs early.

3. *The quick succumbing of the system in certain malignant cases* is evidently due to diphtheritic toxæmia. We sometimes observe a fatal result on the second, third, or fourth day, without any dyspnœa or sufficient laryngitis to compromise life. Cases of this kind, terminating fatally even in the first day, have been reported. The system is suddenly overpowered by the poison, struck down, as it were, by the profound blood-change, while the inflammations are still in their incipiency.

4. Important evidence of the constitutional nature of diphtheria is afforded also by the *state of the kidneys.* No internal organs are so often affected in diphtheria as the kidneys, and, on account of their location and anatomical relation, it is evident that the poison first passes through the system before it reaches them. Any clinical or anatomical fact, therefore, which indicates that the diphtheritic virus has reached and affected the kidneys affords proof that it has penetrated the system and poisoned the blood. Now, the occurrence of albumen, with granular or hyaline casts, in the urine, in cases unattended by dyspnœa, affords proof of nephritis, caused by the action of the poison on the kidneys.

Sir John Rose Cormack, of Paris, in a series of interesting and useful papers relating to diphtheria, published in the *Edinburgh Medical Journal* during 1876, states that albuminuria, and of course the nephritis on which it depends, sometimes begin as early as the first day. My observations confirm this statement, as in the following cases:

Case I.—L. McD., aged three years, was first visited by me on February 29, 1876. I learned from the parents that she had been feverish during the preceding forty-eight hours, and her urine very scanty. A moment's examination was sufficient to show that the case was one of malignant diphtheria, for the fauces were already nearly covered by the diphtheritic pellicle, the temperature was 103¼° F., and the pulse 140. The skin was hot and dry, and there was moderate swelling under the ears, and a muco-purulent discharge from the nostrils. On account of the scantiness of the urine, the amount not exceeding f℥iv–v daily, it was impossible to obtain sufficient for examination till the following day. It was then found to have a specific gravity of 1032, to contain a deposit of urates and hyaline and granular casts, a diminished amount of urea, and a large quantity of albumen. It can hardly be doubted, from the scantiness of the urine, and the large amount of albumen found when the urine was first examined, that albuminuria had been present on the first day.

Case II.—The following was a similar case. K., aged four years, living in West Thirty-

Sixth Street, was visited by me in consultation on January 29, 1875. Her sickness had also continued forty-eight hours; her fauces were swollen, and covered with the diphtheritic pellicle, which was dark and offensive; respiration guttural; pulse 120; temperature 101° F.; she had a free discharge from each nostril; urine scanty, its specific gravity 1030; it contained a small amount of albumen, with casts, and a large amount of urates, with no apparent diminution of the urea. Death occurred on the fourth day.

In such severe cases, in which albumen and casts are found in the urine at the first visit of the physician, there can be little doubt that the nephritis begins nearly or quite as early as the pharyngitis; and therefore, since poisoning of the blood must antedate the renal disease, diphtheria affects the system very early, probably from the occurrence of the first symptoms.

Again, there are cases, though not frequent,—three I can recall to mind during the last two years in my practice,—in which the external manifestations of diphtheria are very mild, even insignificant, and quickly cured, but in which the kidneys are early and severely affected. The occurrence of such cases is best explained on the supposition of an early and profound blood-change. The following are histories of two of the cases alluded to:

The house 229 West Nineteenth Street, New York, is an old wooden structure, and the family which has occupied it during the last five years has been three times visited by diphtheria, the first case, that of the oldest child, proving fatal. In February, 1876, one of the children had diphtheria in a moderately severe form. He recovered, and, after my visits had been discontinued, his sister, aged six years, who had had scarlet fever when eighteen months old, became feverish, and complained of her throat. No rash appeared on her skin, and there was apparently no coryza. Inspection of the fauces by the parents revealed a small diphtheritic patch over each tonsil. Although diphtheria was so frightful a malady to this family from their past experience, the case seemed so mild that the parents treated it without medical attendance, by the remedies which had been employed for the boy. A mixture of carbolic acid, subsulphate of iron, and glycerin was applied to the fauces every third hour, sufficiently often, apparently, to destroy all bacteria or other vegetable or animal organisms with which it might have come in contact, and within two or three days the inflammation of the throat seemed to the parents to be cured. Nevertheless, with this insignificant inflammation of the fauces, so quickly subdued, and with no other apparent inflammation of the mucous surfaces, there was severe internal disease going on as the result of the general infection. The child did not regain her former appetite; she had increasing pallor, although able to play about the house; and finally, in the third week, when I was called to see her, slight œdema of the face and limbs was observed. Her urine, which was scanty, was found to contain pus and blood-corpuscles, albumen, and granular casts, and nearly two months elapsed before, under treatment, it became normal and her health was restored.

The second case occurred in January, 1878, in West Fifty-First Street. A boy, aged six years, in a family in which diphtheria was occurring, had slight sore throat, which abated in two or three days. It was attended by little or no exudation, and would not have been considered diphtheritic except for the circumstances in which it occurred, and the subsequent history. Still, the boy remained ill and fretful, and four days subsequently his urine was found to be very scanty and very albuminous; and three days later death occurred, preceded by total suppression of urine. The last urine passed, which was not more than a teaspoonful, became nearly semi-solid by heat. There had been no scarlet fever in the family.

Cases like the above, in which there is an early and profound systemic infection, with but slight evidence of lodgement of the virus upon the faucial or other exposed surface, are interesting as showing the constitutional nature of the malady, even when the symptoms and visible lesions have extreme mildness. Certain clinical observations, therefore, lend support to the theory that diphtheria, even if it be in most instances local at first, is in some cases systemic from its commencement, and seem to justify the remark made by Dr. A. Jacobi, that probably in some instances the diphtheritic virus enters the system through the lungs,—a supposition which demands consideration, notwithstanding the fact that many pathologists now believe that the specific germ acts only upon the surface. Whether diphtheria be always local in its commencement, or sometimes systemic, it answers the wants of the practitioner to be assured that, in cases of a severe type, diphtheria is systemic at so early a period that constitutional remedies are required at the first visit. He will be the most successful practitioner who fully recognizes the fact that he has to deal with a malady which has both a local and a systemic character.

Diagnosis.—It is very important that the diagnosis of a case of diphtheria be early made, so that proper remedial measures may be employed at the beginning, as well as measures designed to prevent propagation. In a large proportion of cases the diagnosis is easy after diphtheria has continued twenty-four hours, since, in addition to the fever, and pain in swallowing, the characteristic grayish-white pellicle has begun to form on one or both tonsils. Under such circumstances the nature of the malady is apparent on inspecting the fauces. But many cases are not so quickly and readily diagnosticated, even by experienced physicians. The diagnosis is uncertain, and is postponed until two or more days have elapsed. One reason of failure to diagnosticate early is the fact that many patients, even those old enough to express their sensations, do not complain of the throat. I have many times been informed by parents or nurses that there was no need of examining the fauces, as there was no complaint of pain in the throat, and yet on examination have observed unequivocal evidence of diphtheria. A physician practising in a locality where diphtheria is prevailing should at his first visit inspect the fauces of a child to whom he is summoned, especially if there be fever, and he will often discover evidences of diphtheria which without such examination would not have been detected.

When diphtheria has continued from twelve to twenty-four hours, external examination of the neck usually reveals some tenderness as well as fulness in the tonsillar regions, and the enlargement of the tonsils can be readily detected on palpation; but in some instances the tenderness and swelling are so slight as to be scarcely appreciable. In not a few cases it is impossible to make a positive diagnosis until the disease has been under observation some days and its progress and character have been carefully noted, the difficulty in diagnosis arising from the fact that the membranous exudate is concealed from view. Thus, in nasal diphtheria, the pseudomembrane may be located upon the superior and posterior portions of the Schneiderian membrane, and therefore be invisible, while the anterior and visible portions of the nares, and the faucial surface, are hyperæmic and secreting muco-pus in abundance, but are free from the pseudo-membranous exudate. The pseudo-membrane may and probably will appear upon visible parts before the disease terminates, but not early enough to establish a diagnosis in the first days of the sickness. Occasionally in the milder forms of pharyngeal diphtheria membranous patches occur in the depressions of the faucial surface, and are not visible on cursory inspection. They are brought into view when the patient coughs, or by firm external pressure upon the side of the neck, which elevates the depressed surfaces.

In laryngo-tracheal diphtheria, diagnosis is not infrequently delayed in a similar manner. The child, without known exposure to the diphtheritic virus, becomes hoarse, and the hoarseness with fever increases. The fauces show the characteristics of catarrhal inflammation, and the nostrils are not affected, or are affected but slightly. The diagnosis between catarrhal croup, non-specific membranous croup, and diphtheritic croup is uncertain. The patient may die without any visible pseudo-membrane, unless the laryngoscope be used, and without a diagnosis except the general one of croup. The occurrence of albuminuria with casts may enable us to make the probable diagnosis of diphtheria, and this opinion may be confirmed by the contemporaneous or subsequent occurrence of diphtheria in other members of the family; but in other instances no such aid is obtained, and the nature of the attack continues to be a matter of probability only. Such are some of the hinderances in the way of accurate diagnosis.

The following is a résumé of the characteristics of the white, grayish, or grayish-white products of disease which occur on the faucial surface and which are liable to be mistaken for the pseudo-membrane of diphtheria. Let us first consider the characteristics of the diphtheritic exudate. It is deeply set in the mucous membrane, penetrating it and being incorporated with it. It consists of necrosed mucous tissue and firm fibrinous material exuded from the minute vessels, and it cannot be detached from the faucial surface, except at an advanced stage of the disease, without producing hemorrhage. It is surrounded by inflamed and swollen mucous membrane as the crystal of a watch is surrounded by the rim. Compare these characteristics of the diphtheritic pseudo-membrane with the products of other and distinct diseases of the pharynx. First, *follicular tonsillitis*. This is a common disease. In New York, and probably elsewhere, it frequently extends through families as if contagious, all or most of the children being affected by it. It is attended by fever and dysphagia. It has no marked premonitory symptoms, unless of very brief duration, and commences suddenly, like diphtheria, with headache, chilliness, heat of surface, the temperature often rising to 103° F., languor, and frequently pain in the back and extremities. The dysphagia attracts attention to the fauces, the surface of which is seen to be hyperæmic, especially its tonsillar portion. In a few hours a whitish material exudes from the crypts of the tonsils, consisting of the secretion of the crypts and epithelial cells, and forming rounded masses of the size of a small pin's head. The secretion, occurring as small, rounded, salient masses, distinct from one another, is distinguished by its appearance from the diphtheritic pseudo-membrane, which at first is a thin, pellucid film, becoming thicker subsequently. Consisting simply of epithelial cells held together by the secretion, these small rounded masses are quickly detached by the swab or brush, when they are found to be friable, readily crushed between the thumb and fingers, and having a fetid odor. If two or more of them happen to unite, forming an appearance like that of the diphtheritic membrane, they still present the same physical characters, and are readily detached from the tonsillar surface without hemorrhage. This peculiar secretion of follicular tonsillitis is usually limited to the tonsillar portion of the pharynx, and is of short duration, ceasing to appear after two or three days. The inflammation abates soon. In a large number of cases which I have observed, the clinical history of this disease has been as mentioned above, except in one instance, when death occurred apparently from a sudden extension of the inflammation to the larynx and the occurrence of œdema glottidis. The diagnosis of follicular tonsillitis from diphtheria is easily made except as regards the mildest form of diphtheria.

Pultaceous Pharyngitis.—This form of pharyngitis usually occurs in low or debilitated states of the system. It occurs most frequently in the old and feeble, and in such exhausting diseases as scarlatina and typhoid fever. As the term "pultaceous" indicates, the inflammatory product is soft and friable, coming away in fragments, when touched by the brush or sponge, without bleeding or any injury to the mucous membrane. Under the microscope it is found to consist of epithelial cells, often in fragments, nuclei and nucleoli, but no fibrin. When this substance is removed, as it readily can be, the mucous membrane underneath is entire, hyperæmic, and covered by a newly-formed epithelial layer. The appearance of the pultaceous product to the naked eye may closely resemble that in diphtheria, but its friable character, its epithelial nature, and the absence of fibrin which the microscope reveals, render the diagnosis certain.

Scarlatinous Pharyngitis.—The frequency of scarlet fever and diphtheria, and the facts that epidemics of the two are not uncommon at the same time, and that diphtheria often attacks a scarlatinous patient, render important the differentiation of scarlatinous pharyngitis from diphtheritic pharyngitis supervening upon and complicating the scarlatinous. Very commonly when the pharyngitis of scarlet fever is severe, an abundant desquamation of epithelial cells occurs, which, aggregating, produce the pultaceous pseudomembrane described above. This membrane resembles the diphtheritic in appearance, but its anatomical character, consisting as it does of epithelial cells as stated above, suffices to show that it is not a diphtheritic exudate. The grayish-white or brown product of scarlatinal inflammation seldom appears upon other parts than the tonsillar or lateral pharyngeal surfaces, whereas the diphtheritic membrane often appears upon the uvula, upon the posterior faucial surface, and in the nares, in addition to the tonsillar surface.

Gangrenous Pharyngitis.—This variety of pharyngitis occurs oftener in connection with scarlet fever than with any other malady unless diphtheria, and when it complicates scarlet fever the appearance resembles very closely that in advanced cases of malignant diphtheria. The diagnosis is not difficult if the case be observed from the beginning. The diphtheritic pseudo-membrane is in the commencement white or grayish-white. It presents the dark-gray color of gangrene only at an advanced stage, by imbibition of blood and commencing disintegration. Gangrenous sore throat is from the first of a dark-gray, brownish, or even dark color. Gangrene produces a fetid breath, malignant diphtheria does not produce fetor to such an extent until decomposition begins or gangrene supervenes. Gangrene not infrequently complicates the later stages of severe diphtheria.

Herpetic Pharyngitis.—No one can mistake herpes of the fauces in its commencement for diphtheria, the minute vesicles of the former disease are so unlike the diphtheritic exudate. But when the vesicles have disappeared, and are replaced by minute ulcerations, covered by a white and adherent exudate, the differentiation of herpes from benign diphtheria is not easy. The presence of herpes labialis affords presumptive evidence that the pharyngitis is herpetic, but not conclusive, for it is sometimes also present in diphtheria. Immediately after the disappearance of the vesicles, small rounded concretions distinct from one another occupy their place, presenting an appearance entirely unlike that of diphtheria, which exhibits at first a film, soon becoming a thick and firm patch. It is when the concretions unite, forming a patch, that the diagnosis is difficult. I need not state that herpetic pharyngitis, like follicular tonsillitis, is often mistaken for benign diphtheria, and *vice versa*.

Ulcero-Membranous Pharyngitis.—This is an extension of ulcero-membranous stomatitis. It is characterized by a necrosis, limited in extent, and superficial, of the mucous membrane. The presence of ulcero-membranous stomatitis as the important part of the disease, predominating over the pharyngeal affection, aids to a correct diagnosis. Constitutional symptoms are slight or are wanting in this form of pharyngitis. Fever, albuminuria, and glandular swellings, which characterize diphtheritic pharyngitis, are absent or insignificant. The sphacelus over the tonsils, unlike that in diphtheria, is in patches isolated from one another. The microscope reveals epithelial cells, and bands of elastic fibres pertaining to the chorion, as the elements in the necrosed tissue.

Anatomical Characters.—The characteristic and diagnostic feature of diphtheria is the formation upon one of the mucous surfaces, usually the fauces, or upon the skin denuded of its cuticle, of a whitish or grayish-white pseudo-membrane. This membrane, occurring upon mucous surfaces lined by pavement-epithelium, penetrates and is incorporated with the mucous membrane, which undergoes necrosis. The mucous membrane when the pseudo-membrane is fully formed loses its vitality and becomes a part of the pseudo-membranous mass. It cannot, therefore, be detached without tearing the fibres of connective tissue and the vessels which unite the mucous membrane to the submucous tissues, until such time as it becomes detached by the sloughing process. Upon such mucous surfaces as are lined by columnar epithelium the pseudo-membrane does not form an integral connection with the mucous membrane, but lies over it, or lines it, so that it can be removed without injuring it. This form of pseudo-membrane occurs upon the respiratory tract below the superior vocal cord. Above this cord squamous epithelium lines the larynx, except in front, where columnar epithelium occurs as high as the middle of the epiglottis. If croup occur during the course of diphtheria and a pseudo-membrane form upon the laryngo-tracheal surface, in addition to that already existing upon the faucial surface, the patient has both forms of pseudo-membrane described above. Moreover, in the vicinity of these pseudo-membranous inflammations, and extending from them, we ordinarily find a catarrhal inflammation of greater or less extent, an inflammation characterized by redness and swelling of the mucous surface, and a muco-purulent secretion, but without the false membrane. Sometimes also when diphtheria is occurring in a family, one of the children has a simple catarrhal inflammation of the fauces of a few days' continuance. If he have a pseudo-membrane upon any of the surfaces, it is not visible. These three forms of inflammation, that in which the mucous membrane undergoing necrosis becomes incorporated with, and forms an integral part of, the pseudo-membrane, that in which the pseudo-membrane covers the mucous membrane but is anatomically distinct from it, and that in which no pseudo-membrane occurs, the catarrhal, we are in the habit of designating by the term diphtheritic, inasmuch as they occur from the irritating action of the diphtheritic poison. Unfortunately, the most renowned living pathologist, Virchow, restricts the use of the term diphtheritic to that form of inflammation in which mucous membrane undergoing necrosis forms part of the pseudo-membrane, while he does not apply the term diphtheritic, but the term croupous, to that form of inflammation, although occurring in a diphtheritic patient, in which the pseudo-membrane lies upon the mucous surface. This explanation seems to be necessary in order to avoid confusion in the use of the terms diphtheritic and croupous as employed by the school of Virchow.

Soon after diphtheria commences, as manifested by fever and the concomitant symptoms, we observe redness upon one of the surfaces which is to be the chief seat of the local manifestation of the disease. When the malady is contracted in the usual manner, this local manifestation is ordinarily upon the faucial surface, and primarily upon the tonsillar portion. If there be a pre-existing inflammation of one of the other mucous surfaces, or a portion of the cuticle denuded of its epidermis and inflamed, the specific inflammation is likely to appear primarily upon this part, as we have stated above, with or without its simultaneous appearance upon the faucial surface.

The inflammation varies greatly in severity and extent. In a mild attack it is often limited to a part of the fauces, and there are few exceptions to the rule that the tonsillar portion is affected, the redness gradually fading away in the healthy membrane beyond. But in the course of a few hours, in all except the mildest cases, the entire faucial surface presents the characteristic inflammatory redness and swelling, and its follicles are tumefied and actively secreting. In severe cases the uvula is elongated and enlarged from infiltration, and the inflammation even extends to the submucous connective tissue, which becomes hyperæmic and swollen, and the submucous lymphatic glands, especially the tonsils, also swell and are painful. The color of the inflamed surface is sometimes a deep bright red, almost like arterial blood; in other cases it is a dusky red, which indicates, if there be no croupal symptoms, an adynamic and dangerous type of the disease. The dusky-red hue is more common in secondary than in primary diphtheria.

Within a day and usually within a few hours from the commencement of the inflammation, a small, slightly-raised, whitish or grayish spot, or patch, is observed, usually upon the tonsillar portion of the inflamed surface, very significant as a diagnostic sign and as a forerunner of what is to happen. This patch, termed the pseudo-membrane, gradually becomes firmer, and at the same time thicker and broader from fresh exudations underneath. It retains for a time its grayish-white color, but it becomes brownish-white from age. In mild cases the pseudo-membrane is usually limited to the tonsillar surface, but in severe cases it covers the uvula, portions of the velum, the isthmus, and the walls of the pharynx, both lateral and posterior. It does not ordinarily attain a greater thickness than one-eighth to one-sixth of an inch. I have seen it, however, not far from one-third of an inch thick.

Briefly stated, the pseudo-membrane of diphtheria is found to consist of fibrin forming a delicate interlacing net-work, epithelial cells more or less altered by the inflammatory process, leucocytes, nuclei, mucus, and amorphous matter. It also contains, as has been remarked above, different species of bacteria, of which the micrococci are most abundant. The significance of the bacteria is fully dwelt upon elsewhere in this article. The same pseudo-membrane is often firmer in one part than in another, the outer and central portions being more compact and tough for a time than that underneath, which is more recent. After a few days, however, decomposition begins, and then that which was first formed becomes softer than the more recent production. When this occurs, the color of the exudation changes to a dirty brown, and its exposed surface is uneven and jagged, from the partial separation of shreds and fibres. Sometimes the diphtheritic patch has a reddish tinge, due to rupture of the capillaries and escape of blood-corpuscles. Its lower or attached surface may be blood-stained, while the exposed surface has the usual grayish-white hue.

The inflamed mucous membrane is not only hyperæmic and infiltrated with serum, but also contains numerous round white corpuscles (leucocytes), which may result in part from proliferation of connective-tissue corpuscles, but are believed by most pathologists, since Cohnheim's well-known discovery, to be in great part wandering white corpuscles of the blood, which have escaped through the walls of the blood-vessels along with the fibrin. In the commencement of the diphtheritic inflammation, before the pseudo-membrane forms, we often observe a grayish tinge of the mucous surface, which is due to the crowding of these cellular elements in and underneath the mucous membrane; for these newly-formed cells not only infiltrate the mucous membrane, but can also be traced into the submucous connective tissue. Even where the inflammation remains catarrhal, as it does over certain areas in all cases of diphtheria, this infiltration of the mucous and submucous tissues with cells is common.

During the active period of diphtheria, it is often astonishing to see with what rapidity the pseudo-membrane returns when removed by force. A few hours suffice to restore it as firm and extensive as before the interference. In the most favorable cases the membrane is detached in a few days, and is not reproduced. Its separation is promoted by the secretions underneath, especially by pus, which is secreted in abundance between it and the tissues underneath, which have preserved their integrity. In most instances it does not separate in mass, but disappears by progressive liquefaction. Occasionally, even in cases which do not present a severe type, the diphtheritic patch does not disappear until the lapse of four or five or even six weeks, or, if it softens and is detached, another appears in its place. In these instances of an unusual prolongation, diphtheria has been designated chronic.

Such are the appearances, character, and history of the pseudo-membrane in this malady. Although its common seat is upon the fauces, and in mild cases it is limited to them, nevertheless all the mucous surfaces are liable to be attacked by the inflammation, in consequence of infection of the blood, and therefore in severe cases, and even in cases of moderate severity, we often find the product elsewhere as well as upon the fauces, and in localities where from its mechanical effect it greatly increases the danger and even compromises life. The mucous membrane of the nostrils, mouth, larynx, trachea, bronchial tubes, Eustachian tube, conjunctiva, œsophagus, stomach, intestines, vagina, prepuce, and even the delicate lining membrane of the middle ear, are at times the seat of diphtheritic inflammation with the characteristic product. In a case which occurred in the Nursery and Child's Hospital of New York, the surface of the stomach was almost completely lined by the diphtheritic formation, so as apparently to abolish the function of that important organ. The occurrence of the pseudo-membrane in the nares is common, and is attended by the discharge from the nose of thin mucus and pus. Nasal diphtheria involves great danger, from the fact that it is likely to give rise to systemic infection of a grave type. In the nursing infant it is also dangerous, since by its mechanical effect it interferes with lactation. The thin, irritating discharge produces excoriations around the nostrils and upon the upper lip. I have met only one case of diphtheritic inflammation of the intestines in which the diagnosis was certain. A physician in whose family diphtheria was occurring became seriously sick with symptoms which closely resembled those of typhoid fever. After a long sickness, he expelled per rectum about one foot of pseudo-membrane of a cylindrical form, evidently derived from the surface of the intestines. In the subsequent months the patient suffered from constipation and severe abdominal pains, apparently due to contraction in healing of the large intestinal ulcer. Death finally occurred from this state of the intestines. The formation of the diphtheritic pellicle upon the vulva and vaginal walls is not infrequent, and in parturient women exposed to diphtheria it sometimes occurs upon the uterine walls, usually with a fatal result. A considerable number of cases are on record in which diphtheritic inflammation occurred upon the prepuce after circumcision, producing the usual pseudo-membrane, and in one instance in my practice, referred to above, it attacked the prepuce the day after I had dilated it with an instrument clean and free from infection.

In mild cases of diphtheria, in which the pellicle is small, superficial, and limited to the fauces, systemic infection is usually slight; and it is the belief of many that the disease, when of this mild type, not infrequently remains local. But in grave cases, in which the diphtheritic pellicle is extensive and deeply embedded, systemic infection commonly results, notwithstanding the most efficient local antiseptic treatment. The lymphatics and blood-vessels which are in immediate relation with the under surface of the pseudo-membrane take up poisonous ptomaines. Septic blood-poisoning as distinct from diphtheritic blood-poisoning is also likely to occur in those cases in which the pseudo-membrane has become dark-gray and friable from decomposition, producing an ichorous discharge and offensive breath.

The Blood.—The blood in cases of a severe type is usually darker than in health, and the clots soft. After death from diphtheritic croup it is also dark from the excess of carbonic acid in it. The chemical changes which the blood undergoes in diphtheria are little known. MM. Andral and Gavarret found a notable diminution of fibrin in grave infectious diseases, as typhoid fever, puerperal fever, etc., and it is not improbable that the same is true of diphtheritic blood, although the exudation of blood is so abundant. M. Bouchut and others have noted an excess of the white corpuscles in the blood in diphtheritic patients, so that, instead of three or four in the field of the microscope, as many as sixty have been counted. M. Sanné writes of diphtheria, "It is necessary to recognize in the dark-brown blood an abnormal accumulation of the débris of the red corpuscles, débris of little abundance in the normal state, augmented considerably under the noxious influence of the diphtheritic poison, which has rapidly produced destruction of a great number of globules."[1] Small extravasations of blood in the various organs are among the most constant lesions. They have been most frequently observed in the brain and its meninges, the lungs, spleen, and kidneys. In one case which I examined after death, in the

---

[1] Traité de la Diphthérie, p. 107, Paris, 1877.

New York Foundling Asylum, the extravasations in and under the gastric mucous membrane produced mottling as great as that of the skin in measles.

Brain and Spinal Cord.—The anatomical changes occurring in these organs are in a measure described in our remarks on diphtheritic paralysis. Oertel discovered, as the earliest anatomical change in the brain and spinal cord, as well as in the membranes, a venous hyperæmia, with small extravasations of blood "not larger than a pea" in the white medullary matter of the brain, while in the cortical layer and in the central parts no extravasation was found. In the most severe forms of the disease, small hemorrhages not larger than a pea were found not only in the cerebral meninges, but also in various parts of the brain. These produced some softening in their immediate neighborhood. These small hemorrhages have been found also in or upon the medulla oblongata and spinal cord, but with less softening. Buhl, in addition to the extravasations in and upon the brain and spinal cord, discovered in one case great enlargement of the anterior and posterior roots and the ganglionary swellings of the spinal nerves. The swelling was found to be due to the accumulation of cells and nuclei in the sheaths of the nerves and to extravasations of blood. These anatomical changes were most marked at the roots of the lumbar nerves. For further particulars relating to the pathology of the nervous system in diphtheria the reader is referred to our remarks on paralysis.

The most minute examinations of the organs in diphtheria yet published are those recently made by Oertel, and we will present a summary of them in the following pages.

Tonsils.—Covering these organs is the pseudo-membrane, consisting of the usual fibrillar mesh-work, enclosing leucocytes, changed epithelial cells, and amorphous matter: the older the exudation, the coarser is the net-work. The adenoid tissue and the septa have undergone hyperplasia. The follicles are crowded with cells which have undergone necrobiosis. As a result of the necrobiosis, masses are formed of various shapes and sizes, staining deeply. In consequence of the necrobiosis and degenerative changes, the follicles become a hyaline net-work infiltrated with leucocytes and granules. In advanced cases the adenoid and connective tissues undergo a similar necrobiotic change, and are so blended with the pseudo-membrane that it is difficult to determine where the latter ends and the tonsillar tissue begins. The vessels of the tonsils undergo a hyaline thickening of their walls; and if this occur chiefly in the intima, total occlusion may result. In the tissues immediately surrounding the tonsils, hyaline degeneration of the muscular fibres occurs (Zenker's degeneration), and the connective tissue between the muscular fibres is infiltrated with leucocytes.

Faucial Surface and Uvula.—These parts are often also covered with pseudo-membrane, and are more or less changed by the application of remedies. The line of separation of the exudate and underlying tissues cannot be readily distinguished. The upper portion of the diphtheritic pellicle is filled with bacteria and with leucocytes and other cells which have undergone necrobiosis. In the mucosa next to the pseudo-membrane, hyaline degeneration of the connective tissue occurs, and the mucosa is infiltrated with cells which have undergone marked changes. The nuclei of the connective-tissue cells exhibit various stages of degeneration and decay, though the cells may retain their form. The deeper layers of the mucosa, like the upper, are infiltrated with leucocytes.

The *uvula* in severe cases is usually swollen and œdematous, and sometimes entirely covered by the diphtheritic pellicle. When the uvula is involved in the general faucial inflammation, necrobiosis of the cells and nuclei occurs in every part of it. The cells in the arterial adventitia and in the perivascular tissue exhibit necrobiotic change, their nuclei being disintegrated. In the uvula, also, hyaline degeneration occurs in the walls of the vessels.

Epiglottis.—The epithelial cells covering the epiglottis undergo marked proliferation early in the disease, and are infiltrated with leucocytes. They soon begin to undergo degeneration, forming granular masses. Areas of necrobiosis occur, and finally hyaline degeneration of the net-work takes place. The leucocytes extend deeply into the mucous membrane, followed by degenerative and necrobiotic changes. In places the epithelium is thrown off, and a pseudo-membrane forms of exuded fibrin and necrobiotic leucocytes and epithelium. Bacteria, along with leucocytes and degenerated epithelial cells, occupy the meshes of the pseudo-membrane.

Lungs.—The anatomical characters of the air-passages are fully treated of in the article on diphtheritic croup. Catarrhal bronchitis is common in diphtheria. It is not often absent in croup, and one of the chief sources of danger in this disease is the extension of pseudo-membrane from the laryngo-tracheal surface to the bronchial, and the transformation of the catarrhal into a croupous inflammation. When bronchitis occurs, the inflammation creeps downward gradually from the laryngo-tracheal surface, and its severity is proportionate to the degree of extension. When there is a general bronchitis, and it is very liable to become croupous, the muco-purulent exudation is abundant. When pseudo-membranous bronchitis occurs, there are usually portions of the bronchial tree in which the inflammation remains catarrhal. One of the chief sources of danger in diphtheritic croup is the extension of the inflammation to the bronchial tubes, and the abundant secretion of muco-pus, which clogs the tubes and prevents proper decarbonization of the blood. When the bronchitis becomes croupous, a thin easily-detached film appears upon the intensely-red, hyperæmic, and swollen bronchial surface. It increases in thickness and firmness, and assumes a dull white color. Still later, it becomes thicker, firmer, and of a brownish-gray color. Whatever the stage of the inflammation, the pseudo-membrane can always be readily detached from the bronchial surface, since its relation to it is one of apposition, and not of integral connection as upon the pharyngeal surface. In the large tubes, and those of medium size, hollow cylinders, more or less complete, form; but in the smaller tubes, if the pseudo-membrane extend to them, solid cylinders are produced. Frequently in the bronchial croup of diphtheria, while the entire bronchial surface is intensely red and swollen, the pseudo-membrane is absent in certain parts; in other parts it forms cylinders, in other parts still, longitudinal bands of a ribbon shape are produced, and, in more or fewer of the minuter tubes, plugs which entirely fill the lumina and prevent the entrance of air. The alveoli beyond these plugs gradually collapse, and more or fewer of them return to the unexpanded fœtal state. From the tubes which are still pervious the muco-pus is with difficulty expectorated, on account of its viscidity, and the thick muco-pus which collects contains floating particles of pseudo-membrane. Pseudo-membranous bronchitis in diphtheria is in nearly all instances an extension of a laryngo-tracheal croup. It occurs, according to Sanné, most frequently between the second and sixth days.

Various forms of pulmonary disease occur in diphtheria, usually as a complication, and often as a final result of the downward extension of inflammation from the larynx, trachea, and bronchial tubes. Splenization, atelectasis, and broncho-pneumonia, the inflammation commencing upon the laryngo-tracheal surface and extending downward, are common complications of diphtheritic croup. Broncho-pneumonia, like pseudo-membranous laryngo-tracheitis and pseudo-membranous bronchitis, upon which it largely depends, occurs usually in the first week of diphtheria. In one hundred and twenty-one cases of broncho-pneumonia complicating diphtheria, observed by Sanné, the pneumonia commenced in two on the first day of diphtheria and in seventy-one between the second and sixth days inclusive.

Pulmonary congestion, occupying by preference the depending portions of the lungs, especially the posterior and inferior portions of the lower lobes, is also not infrequent. It occurs when respiration is obstructed in croup, and when the circulation is feeble in consequence of heart-failure. In the dyspnœa which accompanies paralysis of the pneumogastrics, venous congestion of the lungs commonly occurs.

The pneumonia which occurs in diphtheria usually results, as stated above, from extension of inflammation from the bronchial tubes, occurring largely in cases of pseudo-membranous bronchitis. It is a broncho-pneumonia. Simple pneumonia, or pneumonia occurring independently of bronchitis, is also sometimes met with. Peter cites a case, and Sanné states that he has observed forty-eight cases. In nine of thirty-two of these cases, verified by autopsies, he found gray hepatization.

Peter found the lesions of *pleurisy* nine times in one hundred and twenty-one autopsies in diphtheria, and Sanné observed them in twenty cases. The latter writer says, "All forms of diphtheria, but particularly croup and pseudo-membranous bronchitis, are to be found with pleurisy. Pleurisy always accompanies some other phlegmasia."

Vesicular emphysema commonly occurs during the progress of croup. Whenever, in consequence of occlusion of the tubes, a considerable part of a lung fails to receive air, its alveoli begin to retract and collapse, and the alveoli which receive air, which are principally those in the superior and anterior portions of the lung, are overdistended, since their function is compensatory. Vesicular emphysema consequently results, and in exceptional instances the vesicles rupture and the escaped air passes into the connective tissue, producing interstitial emphysema.

Pulmonary apoplexy occasionally occurs, the extravasations usually being of small size and disseminated through the lungs. It is most frequent in malignant cases,—in cases attended by profound blood-poisoning. It has been attributed, in some instances, to pulmonary emboli resulting from cardiac thrombosis, or microbic masses, intercepted in the capillaries. *Pulmonary œdema* also occasionally occurs, especially in cases of bronchial croup, pulmonary congestion, and broncho-pneumonia. Oertel in his recent microscopic examinations of the lungs noted subpleural hemorrhages and hemorrhages extending to the alveoli, which were compressed. "Leucocytes infiltrated the alveolar septa, and, in later stages, invaded the alveoli, the epithelium of which became detached and the characters of catarrhal pneumonia were thus produced." Some alveoli contained fibrinous exudation, and in one severe case the alveolar contents consisted of nuclei which exhibited disintegrating changes somewhat like those in necrobiosis.

Lymphatic Glands.—Enlargement of the cervical and submaxillary glands is of common occurrence in diphtheria, and it is a diagnostic symptom of some value. Hyperplasia of the cells of these glands occurs, with numerous hemorrhagic points in their capsules and in the periglandular tissue. Points of necrobiosis, staining faintly, occur in the glands, more in the cortical than in the central portions. The cells exhibit evidences of disintegration; and when this process is advanced, granular masses form in the affected foci. Hyaline degeneration is also observed in portions of the glandular tissue, a degeneration common in other organs in diphtheria. Where disintegration is not too far advanced, cells with polymorphous nuclei are observed,—evidence of an active hyperplasia. Hyperplasia with points of hemorrhagic extravasation takes place also in the bronchial glands, but fewer points of necrobiosis occur than in the cervical and submaxillary glands, and these chiefly in the follicles. The lymph-ducts may contain no normal cells, and only those with disintegrated nuclei along with other products of disintegration.

Heart.—The state of the heart will be in part described in our remarks relating to cardiac paralysis. Small extravasations of blood under the pericardial, and less frequently the endocardial, surface, have been observed. Oertel attributes these hemorrhages to changes in the walls of the vessels caused by the diphtheritic virus, and Buhl, to nuclear proliferation in the walls and mechanical obstruction. Leucocytes in masses often occur under the pericardium and endocardium and between the muscular fibres. Sometimes the muscle-nuclei have undergone segmentation and degenerative changes. These nuclear changes occur mostly in fibres under the endocardium and around the coronary arteries. The nuclei in the muscular coat of the arteries are increased in size, and slight proliferation and desquamation of the endothelia and infiltration of the adventitia also take place.

Mouth, Stomach, Intestines.—The diphtheritic pellicle sometimes forms in the cavity of the mouth, generally in small patches; but the buccal surface is usually only superficially involved, except upon the tongue, where the pellicle extends more deeply. I have elsewhere stated that the diphtheritic exudate sometimes occurs upon the surface of the stomach and portions of the intestines, producing more or less destruction of the mucous membrane. Necrobiotic foci have been observed by Bizzozero and Oertel in the intestinal follicles and agminate glands, but to a less extent than upon the respiratory surfaces. Active cell-proliferation and disintegration, and cleavage of nuclei, occur, but these altered cells are mixed with others which are normal. The epithelium is for the most part retained and normal, and hyaline changes have not been observed in the gastro-intestinal vessels. The mesenteric glands sometimes undergo enlargement from hyperplasia, especially when the intestines are affected, and points of necrobiosis occur in them. For the most part, however, the gastro-intestinal surface is less frequently affected than other mucous surfaces.

Spleen.—The diphtheritic virus reaches this organ through the blood-current. The spleen is swollen, so as to render its capsule tense. The pulp is soft, rising up through the cut surface of the capsule; the follicles are large and prominent; in the pulp are extravasations of blood and hæmatoidin masses, and the vessels are distended. Hyperplasia of the splenic corpuscles occurs, which is most marked around the bifurcations of the arteries, so that the reticulum is less prominent. The follicles are surrounded by a wide zone of the reticulated cells, among which we find lymphoid corpuscles, leucocytes, and large round cells. The nuclei in the cells undergo two changes: first, direct segmentation as in ordinary cell-division, and fragmentation, in which the chromatin is broken up in small, irregularly-disposed masses and the nuclear juice is susceptible of staining. In the Malpighian follicles either numerous epithelioid cells form, as mentioned by Stilling,[1] or large cells occur. The latter stain better by coloring reagents than the epithelioid cells, but less than the leucocytes. The epithelioid cells occur mostly in young patients. A wide zone of leucocytes surrounds and invades the follicles. The necrobiotic process also occurs as in other organs, beginning with nuclear disintegration, and when at its maximum the follicles are surrounded and loaded with the altered nuclei furnished by the round or epithelioid cells. Hemorrhages also occur in the follicles. In some protracted cases the vessels of the pulp exhibit the hyaline degeneration.

Liver.—Capillary hemorrhages take place within the capsule, and occasionally within the parenchyma. Leucocytes occur at certain points within the liver, infiltrating the tissue of the organ. They occupy the interlobular spaces, and do not exhibit nuclear changes. The hepatic cells are unchanged, or they become fatty.

---

[1] Virchow's Archiv, Bd. ciii.

Kidneys.—Albuminuria occurs from different causes, as we have stated elsewhere. Feeble heart-action, obstructed respiration, fever, and the direct irritating action of the diphtheritic virus upon the blood and the kidneys, are sufficient causes. The kidneys may be normal in cases of albuminuria, or exhibit different degrees of parenchymatous inflammation. Hemorrhages, glomerulitis, and disseminated nephritis are common lesions observed in the kidneys in those who have died having diphtheritic albuminuria. Hemorrhagic points occur not only under the capsule, but also in the glomeruli and in and between the tubules. Cell-infiltration takes place around the vessels, and the cells exhibit nuclear disintegration. On examining the glomeruli, thickening of Bowman's capsule is sometimes observed, with some albuminous exudation underneath it, and epithelial proliferation and desquamation. The nuclei and endothelia of the glomerular capillaries are increased, and the chromatin and nuclear juice have undergone disintegrating and degenerative changes, results of inflammation. The capillaries are therefore in a degree diseased through the action of the blood-poison. The epithelium of the convoluted and straight tubes is also diseased. The epithelial cells, undergoing cloudy swelling, become detached from the basement-membrane, fill the lumina with the necrosed product, and, some of them, escape, forming casts in the urine. Occasionally only the outer portion of the cell is necrosed and detached, the part adjacent to the basement-membrane, containing the nucleus, remaining *in situ*. Oertel says that when the entire cells are thrown off, granular casts are formed, but if only the outer portions are lost, hyaline casts are produced. The collecting tubes, filled with granular masses containing broken nuclei, cells, and epithelia, may be dilated.

The above description of the anatomical changes which occur in the various organs is for the most part a résumé of the recent investigations by Oertel. Whether his published statement will be fully sustained by subsequent microscopic examinations remains to be seen.

Symptoms.—Diphtheria, like scarlet fever, varies greatly in severity, from a form so mild that medical advice is not sought and the child is not even confined to his home, to a form so severe that the system is at once overpowered and the patient is in a critical state from the first. In general in the commencement of an epidemic the symptoms are more severe than when the epidemic influence is abating. During the continuance of the attack, the prominent symptoms, such as arrest attention, are often disproportionate to the gravity of the attack. Striking cases illustrative of this fact have occurred in my practice, the friends not supposing that there was any serious ailment, and not seeking medical advice until the fatal termination was near.

In *benign* diphtheria the initial symptoms are often slight, such as languor or lassitude, slight chilliness succeeded by fever of a light form, mild headache, pain or aching in the body or limbs, thirst, and impaired appetite. Usually some soreness of the throat is noticed in swallowing soon after the attack begins, and this continues. But the patient with mild diphtheria often continues to walk about, in the belief that he is affected with a slight and temporary ailment. Children with mild diphtheria, in the poorer families, are usually allowed to go abroad, and do great harm by propagating the disease. The symptoms in these mild cases so closely resemble those from a severe cold that the disease is liable to be mistaken for it. The slight tenderness or sensation of fulness in the fauces usually experienced by those old enough to express their sensations should always lead to an examination of the fauces,—when the character of the attack will frequently be apparent. A distinguished clergyman of the Pacific coast, who fell a victim to this disease, dreamed a few nights before he complained of his illness that his throat was cut. Doubtless the diphtheritic inflammation had already commenced, so that what seemed a forewarning had a natural explanation. So insidious was the commencement in this case that the disease had advanced beyond all hope of relief when medical advice was first sought.

Soon after the attack commences, inspection of the fauces reveals redness of the tonsillar surface, and this extends until the entire fauces present an infected appearance. After the lapse of twelve to thirty-six hours, or even as late as forty-eight hours, from the commencement of the disease, the diphtheritic exudate begins to form over the tonsils, producing the characteristic pellicle. Before it forms we often observe a grayish color of the prominent part of the tonsils, produced by the infiltration of the mucous membrane, and even of the surface of the tonsils, with newly-formed cells. The exudate may appear as points, which coalesce, forming a patch, or as a pellicle, which soon becomes thicker and at the same time firm. Its anatomical characters are described elsewhere.

But in most cases, in all except of the mildest type, the initial symptoms are more severe than we have delineated above. The attack in the *ordinary* as well as *severe* form of diphtheria commences abruptly, like scarlet fever, without a premonitory stage, and with pronounced symptoms from the first. The temperature rises to 102°, 103°, or even 104° F., with corresponding heat of surface, thirst, languor, loss or impairment of appetite, tenderness of throat, etc. Delirium as well as eclampsia may occur; but both are rare. The temperature ordinarily begins to fall after the second or third day in favorable cases, and often in those of a grave and fatal type. Subsequently to the third or fourth day the temperature is frequently but little elevated. The diphtheritic poison, when the system is fully under its influence, does not exhibit any marked tendency, like that of scarlet fever, to increase the animal heat. Even in profound and fatal diphtheritic blood-poisoning rapidly approaching an unfavorable termination, the thermometer often indicates nearly the normal temperature, so that the inexperienced practitioner may be deceived by this fact in his prognosis. A continued elevation of temperature considerably above the normal should lead the physician to examine for some complication, perhaps nephritis.

The tongue is moist and slightly furred. Many patients vomit in the commencement; and if this symptom cease, or be not repeated, it is not of grave import; but vomiting occurring often, so that a considerable part of the food is rejected, is common in grave cases, and is an unfavorable prognostic symptom. It frequently is due to uræmia. The appetite, in severe cases, is usually poor. Repugnance to food from loss of appetite, and pain in swallowing, characterize severe forms of the disease. There are no notable symptoms referable to the state of the intestines. The stools appear normal, except as they are changed by the medicines prescribed. In all cases except the mildest, a rapid destruction of red corpuscles occurs, and a relative increase of white corpuscles. Hence the anæmia which is soon manifested by pallor of the surface, and which rapidly increases as the disease advances. The early loss of the tendon reflex has recently been brought to the notice of the profession. It often occurs as early as the first, second, or third day. It is fully treated of in our remarks relating to diphtheritic paralysis in subsequent pages. It is a symptom of diagnostic value. Diphtheritic inflammations have a marked tendency to produce hyperplasia and consequent notable enlargement of the lymphatic glands in their immediate neighborhood. The poisonous and irritating products of the inflammation upon the surface taken up by the lymphatics, and deposited in the adjacent glands, produce in them tenderness, swelling, an increased afflux of arterial blood, and a rapid increase of the cellular elements. An inflammation both of the lymphatic ducts and glands arises, with more or less œdema and sometimes inflammation of the adjacent connective tissue. Suppuration of the glands and connective tissue, though it may occur, is much less frequent than in scarlet fever.

Temperature.—There is probably no other disease in which the thermometer furnishes so little aid to an understanding of the case as in this, since the degree of fever does not sustain any fixed relation to the amount of blood-poisoning. Malignant diphtheria with profound blood-poisoning and approaching a fatal termination may be almost apyretic, while a benign form of the disease with but little blood-poisoning may commence with considerable fever (102°, 103°, or 104° F.). Fever in diphtheria is rather a symptom of the inflammation than of the blood-poisoning. Considerable elevation of temperature in diphtheria usually indicates an active pharyngitis, tonsillitis, laryngo-tracheitis, bronchitis, pneumonia, or nephritis. Therefore, although the thermometer does not aid in determining the amount of blood-poisoning, it enables us to form an opinion in regard to the extent and severity of the inflammation which may be present. The thermometer is also useful when diphtheria occurs as a complication of another constitutional disease, as scarlet fever, measles, typhoid fever, since it indicates the severity of this disease.

Such is the clinical history of diphtheria as it usually occurs; its local manifestation being primarily upon the tonsillar portion of the fauces, and extending from the tonsils, when the case is severe, to the posterior surface of the fauces, over the anterior and posterior pillars, and to the uvula. The uvula, when it is involved, becomes greatly swollen, even two or three times its normal size, so as to lie upon the tongue, and, especially if it be covered by a pseudo-membrane, to fill up the space between the swollen tonsils and intercept the view of the posterior fauces. When the inflammation is intense and the pseudo-membrane has not yet formed or has been removed by solvent applications, the tonsillar portion of the fauces often presents a grayish appearance, from infiltration of leucocytes. This infiltration, if so great as to obstruct the circulation, leads to necrosis; but, as we have stated elsewhere, the necrosis of the mucous membrane is more likely to occur when it is still covered by the pseudo-membrane, the pseudo-membrane and mucous surface being incorporated with each other and being detached together. The color of the pseudo-membrane, at first whitish or a grayish white, becomes in a few days, in severe cases, a yellowish brown by the action of the atmosphere, and sometimes by extravasation of blood. If the membrane be abundant, it is likely to have in a few days a musty and offensive odor, due to commencing decomposition. The constant inhalation of the highly poisonous gases which result is detrimental to the patient, and they increase the danger of infection in others. However, with the use of disinfectants, now so commonly employed, the poisonous gaseous products of decomposition are not so common as in former times. Since the pseudo-membrane is incorporated with the mucous membrane, and capillaries penetrate its under surface, forcible detachment of the pellicle is likely to give rise to hemorrhage. Hemorrhage is always a bad prognostic sign. The duration of the pseudo-membrane is very variable. On the average, in favorable cases it is from one to two weeks. There are cases, however, in which the ulcerated surface is long in healing, and the ulcers are covered many days with the grayish-white diphtheritic exudate. In exceptional cases at the close of the third or even fourth week we occasionally observe on the faucial surface diphtheritic patches two or three lines in diameter, without surrounding inflammation, in those who consider themselves nearly well, and who would appear in the streets if they were allowed to do so. We will consider elsewhere how long enforced seclusion of the patient should be enjoined in order to prevent the propagation of the disease to others.

Nares.—Usually inflammation of the nostrils occurring in diphtheria is secondary to that of the pharynx. The pharyngitis has continued one or more days, when a discharge of a thin serous appearance occurs from the nostrils. This is attended by swelling of the Schneiderian membrane; and in proportion to the amount of swelling the respiration through the nostrils is embarrassed. As the inflammation continues, the swelling increases, and respiration is accompanied by a nasal snuffle, or the occlusion of the nostrils is so great that it is performed entirely through the mouth. The impediment to respiration in infants at the breast is often so great that spoon-feeding is necessary. The discharge is very acrid and irritating, causing excoriation around the entrance of the nostrils and even upon the cheeks. It soon becomes more viscid or less fluid than at first, and it presents a creamy appearance from the large proportion of pus-corpuscles. When the inflammation of the nares is severe, the glands around the articulation of the lower jaw usually undergo hyperplasia, becoming nodular and prominent, so as to be apparent not only to the touch but also to the sight.

Although commonly diphtheritic inflammation of the nasal surface is secondary to that of the fauces, it is sometimes the primary inflammation. It may exist for some days before the fauces become affected, and under such circumstances the diagnosis is frequently not made until the disease is in an advanced stage and profound blood-poisoning has occurred. In nasal diphtheria the pseudo-membrane probably occurs as early as in other forms of diphtheritic inflammation, but, being usually out of sight, it is not observed in the first days or until it has extended so that its anterior edge can be seen on inspecting the nasal fossa. From its concealed position it is easy to perceive why the disease is so frequently overlooked and a simple nasal catarrh is supposed to be present when there is no inflammation of the fauces to aid the diagnosis, or it is late in appearing.

Nasal diphtheria always involves great danger, since it is very liable to give rise to systemic infection from the large number of lymphatics lodged in the connective tissues of the nares. In certain severe cases accompanied by swelling of the face there is reason to think that the inflammation has entered the antrum of Highmore, a very serious extension. It sometimes extends up the tear-duct, producing its occlusion, and also along the Eustachian tube. Hemorrhage sometimes occurs in nasal diphtheria. In those who recover, the Schneiderian membrane returns slowly to its normal state.

The Eye.—We have stated above that the inflammation sometimes passes along the tear-duct to the conjunctiva, but in other instances the inflammation occurs independently of this mode of propagation. Thus, if a child with simple conjunctivitis contract diphtheria, the pre-existing inflammation is very liable to assume a diphtheritic character, in accordance with the law already stated, that diphtheria attacks by preference surfaces that are already inflamed. I have elsewhere stated that diphtheria at one time entered the ophthalmic wards of the New York Foundling Asylum, and three children, under treatment for granular lids, who contracted the disease, had diphtheritic inflammation of the lids, with the usual pseudo-membranous exudate. The result of diphtheritic conjunctivitis, even with prompt and appropriate treatment, is likely to be disastrous as regards the eye. The eyelids become red and greatly swollen from œdema, and their under surface is soon lined by a thick and firm pseudo-membrane. The eye itself is the seat of chemosis. The pseudo-membrane upon the ocular conjunctiva is less firm, not so thick, and more in flakes, than that upon the palpebral conjunctiva. The eye affected by this disease should be closely watched and promptly and efficiently treated; but, unfortunately, under the most judicious treatment the cornea is likely to become hazy, and sloughing or ulceration follow, with total destruction of sight, and perhaps prolapse of the iris.

The Ear.—The ear may become inflamed by extension of the inflammation along the Eustachian tube from the fauces. The opening of this tube upon the faucial surface is small and slit-like in the child, and moderate inflammation and exudation are sufficient to close it. When this occurs, the patient complains of pain in the site of the tube, and in the ear. The formation of a membrane plugging the tube and the extension of the inflammation to the ear, producing an otitis media, add very much to the gravity of the case. Perforation of the drum, caries of the bones of the ear, and that grave disease otitis interna, may occur, increasing very much the gravity of the case. Fortunately, this extension of the inflammation is not frequent. It does not often occur except in those malignant cases which are likely to be fatal from other causes. Sometimes, also, a diphtheritic otitis externa occurs. It is usually preceded by a catarrhal inflammation which has arisen from other causes and was present when the diphtheria commenced. Bezold described three cases of otitis externa with a diphtheritic pellicle upon the drum.[1] Moos and Callan have also narrated cases.

The Mouth.—During the progress of diphtheria any sore or abrasion of the mouth is likely to become the seat of the diphtheritic exudate. Usually the fauces and sometimes the nares are at the same time affected. The diphtheritic pellicle, commonly of small extent, may appear upon the inside of the cheek, the tongue, gums, and lips. Usually the inflammation of these parts is of secondary importance, but in malignant or highly septic cases it may be attended by considerable infiltration and thickening. Buccal diphtheria, if severe, is painful, and it may interfere with the proper nutrition. The clinical history of diphtheritic inflammation of the fauces and respiratory tract below the epiglottis is sufficiently presented elsewhere.

Œsophagus, Stomach, Intestines.—The upper part of the œsophagus not infrequently participates in the inflammation of the pharynx. Its walls are thickened, and the pseudo-membrane presents the same characters as upon the fauces. Occasionally nearly the entire œsophagus is the seat of diphtheritic inflammation, the œsophageal walls being greatly thickened from infiltration of cells, and very vascular. In one of the cases, related in a foregoing page, of diphtheria of the newly-born, the œsophagus was in nearly its entire length covered by the diphtheritic pseudo-membrane. In only one instance have I observed a severe diphtheritic gastritis. In this case nearly the entire surface of the stomach was covered by a thick pellicle. Probably the inflamed follicles did not secrete normal pepsin. A few cases are on record of diphtheritic inflammation of the intestines. Dr. A. Jacobi relates the case of a child of three years who had diphtheritic enteritis. Fever, moderate tenderness of the abdomen with but little tympanitis, constipation, and great prostration were the prominent symptoms. The autopsy revealed the presence of a diphtheritic inflammation in the jejunum and ileum, the membrane consisting of "a dense net-work with granular contents." The most marked case of diphtheritic intestinal inflammation which has come under my notice was that of a physician to whose case I have elsewhere referred. He lost his appetite, had fever, lost flesh and strength, had distress in the abdomen which raised the suspicion of a typhoid fever; but at the usual time for the termination of a self-limited fever no abatement of symptoms occurred. Finally, after weeks of suffering, he expelled a cast of the intestine several inches in length, probably from the colon. Obstinate constipation was the most prominent symptom during this time and subsequently, due, probably, to cicatrization and contraction of the intestine. The patient died from the effects of the disease several months subsequently, having suffered constantly from faulty digestion, abdominal pain, and constipation, which no treatment could relieve or benefit.

---

[1] Virchow's Archiv, lxx. 329.

Genito-Urinary Organs.—Diphtheria of the prepuce commonly occurs after some injury. It either arises by direct inoculation upon an abrasion or wound, or is contracted by exposure to an infected atmosphere. Many cases are on record. I have elsewhere stated that the eminent surgeon M. Germain Sée, whose practice is in a locality where diphtheria is endemic and very prevalent, now recommends stretching of the prepuce in nearly all cases of narrow and adherent prepuce rather than circumcision, for the reason, among others, that diphtheria is more liable to follow the latter operation. Diphtheria of the prepuce is contracted by the use of infected instruments, sponges, or fingers, in the operation of circumcision, or by the performance of the operation with clean instruments and hands but in an infected atmosphere. Thus, Dr. F. Langé saw a case of preputial and scrotal diphtheria in a child of three weeks who had been circumcised when diphtheria was occurring in the family.[1] Dr. Greves states that a boy who had been circumcised for phimosis was admitted into the Liverpool Infirmary with an unhealthy prepuce, which had never healed after the operation. Weak and anæmic when admitted, he continued to sink, and died of heart-failure. The wound and subjacent tissues were infiltrated with micrococci presenting the same characters as those in pharyngeal pseudo-membranes. In a preceding page I have alluded to a case, related by Mr. Phillips, of preputial diphtheria occurring after circumcision by infected instruments, and have related a case in my own practice of a severe diphtheria of the prepuce, and simultaneously of the fauces, occurring after instrumental dilation of the foreskin. Dr. A. Jacobi states that he incised the upper part of the prepuce in a healthy boy of three years, employed stitches, and applied carbolized dressing. On the following day diphtheria attacked the wound, with the usual swelling and erysipelatous appearance. The stitches were removed, but death occurred four days after the operation. Dr. A. Jacobi also relates the case of a boy of four years whom he circumcised and dressed the wound with antiseptic solutions. Diphtheria supervened, and in a few days the entire prepuce and a small portion of the penis became gangrenous. The boy eventually recovered, with deformity of the organ.

---

[1] Medical Record, July 10, 1880.

Billroth has called attention to the fact that diphtheria in localities where it is prevailing is likely to attack wounds produced by operations on the urinary apparatus, as after lithotomy or urethrotomy, and in cases of ectopia vesicæ and vesico-vaginal fistula. The inflammation under such circumstances is usually localized, but it may extend to the retroperitoneal connective tissue and produce a fatal peritonitis. The marked liability of the uterus, vagina, and vulva when wounded in any way, as in parturition, to become the seat of diphtheritic inflammation in case of exposure to the infection is well known to the profession, and no prudent obstetrician will attend an obstetrical case after visiting a diphtheritic patient, without change of apparel and personal disinfection. Some years ago I was summoned to a young lady who during the week following her confinement insisted on seeing her child, then in the commencement of diphtheria. The child was brought to her bedside for a moment. Within a day or two she was attacked with a violent form of metro-peritonitis, which was speedily fatal. In children diphtheritic vulvitis and vaginitis occasionally occur, associated or not with pharyngitis. I. Zit has records of thirteen cases of diphtheritic vulvitis, in some of which inflammation was the first manifestation of diphtheria. Diphtheritic inflammation of the vulva and vagina is believed to be rare without a pre-existing catarrhal inflammation.

Skin.—An efflorescence is sometimes observed upon the skin during the time in which the temperature is exalted. It is the *erythema fugax* of dermatologists, suddenly appearing and disappearing. This eruption, which is common in the febrile and inflammatory affections of childhood, does not seem to present any peculiar characters in diphtheria. But there is another eruption which I have not infrequently observed, and which is attributable to diphtheritic toxæmia or septicæmia. It appears after the sixth or seventh day, in the form of red points or spots not more than a line in diameter, and interspersed with patches of efflorescence with irregular margins, one to two inches in diameter. This roseolar eruption is slightly raised, like that of measles. Sometimes it is punctate. It disappears on pressure, and in my practice it has usually appeared in grave cases, in which there were other evidences of blood-poisoning. Occasionally extravasations of blood occur in and under the skin, like those in internal organs. The pallor of the skin which diphtheritic anæmia and toxæmia produce in and after the second week is known to all who have had experience with this disease.

The anatomical characters and symptoms pertaining to the nervous system and kidneys will be treated of at length in our remarks on albuminuria and paralysis. Albuminuria and paralysis, whether we regard them as symptoms, complications, or sequelæ, occur so frequently and are of such grave import that it is proper to treat of them at length. They are the most important of the phenomena pertaining to the symptomatology of diphtheria.

Albuminuria.—It is perhaps remarkable that numerous epidemics of diphtheria had been observed before it became known that albuminuria is a common accompaniment of it. The fact that the kidneys are affected so as to give rise to albuminous urine was discovered by Mr. Wade, of Birmingham, England, in 1857. The interesting paper communicating his discovery was published in *The Midland Quarterly Journal of Medicine*, 1857. Immediately after its appearance, the subject to which he drew attention was fully investigated in different countries, and in the same year Mr. James published his observations in the *Medical Times and Gazette*. In the following year—1858—two noteworthy papers appeared on the same subject, one by MM. Bouchut and Empis, read before the Parisian Academy of Sciences and published in the *Gazette des Hôpitaux*, and another by Germain Sée, read before the Société des Hôpitaux. Since 1858, monographs and reports of cases too numerous to mention have been published, so that the literature of diphtheritic albuminuria is quite full.

As to the frequency of albuminuria in diphtheria, Bouchut and Empis found it in two-thirds of their cases, Germain Sée in one-half of his, and Sanné in two hundred and twenty-four cases out of four hundred and ten. In New York City, where diphtheria has been many years naturalized or endemic, I made in the years 1875 and 1876 daily examinations of the urine in sixty-two consecutive cases, and found it present in twenty-four, while thirty-eight were recorded exempt. But the proportion of cases as stated in my statistics is probably below the truth, for the albuminuria is sometimes transient, and it often occurs as a mere trace and is liable to be overlooked. Its duration is frequently not more than from one to three days, and in the majority of instances it does not continue longer than ten days; but we are all familiar with cases in which it continues fifteen or twenty days, or even months.

The date of the commencement of albuminuria varies greatly in different cases. Perhaps the largest number of observations bearing on this point are those of Sanné. In two hundred and twenty-four cases albuminuria was detected on the first day of diphtheria in three, on the second day in ten, on the third day in thirty, on the fourth day in thirty, on the fifth day in twenty-two. From the sixth day to the eleventh the number on each day in whom albuminuria was present for the first time varied from ten to thirty-three. After the eleventh day there were only nine new cases, and after the fifteenth day only one new case. Hence from these statistics we infer that there is little danger that albuminuria will occur after the second week, if the patient have exhibited no symptoms of it previously.

The amount of albumen in the urine varies greatly in different patients, from a slight cloudiness, scarcely visible after boiling, to so large a quantity that it becomes semi-solid by the application of heat or nitric acid. When the proportion of albumen is very large there is also usually a notable diminution in the quantity of urine passed. In ordinary cases the percentage of albumen varies at different times. It sometimes disappears during one or two days, and we are led to think that the patient is rapidly recovering, but its reappearance in full quantity shows that the apparent improvement was due to some transient cause. "Nothing," says Sanné, "is more irregular than the course of diphtheritic albuminuria. At one time the precipitate is sudden, abundant, and flocculent; at another it commences with an opaque cloud, and continues with this characteristic till the time at which it disappears." Diphtheritic albuminuria differs in many respects from that in scarlet fever. The urine at first, when the renal disease is active, sometimes presents a pinkish tinge, and the microscope reveals the presence of red blood-corpuscles, but afterwards, and in mild cases from the first, the urine exhibits nearly the normal appearance, even when very albuminous, in contradistinction to its cloudy appearance in scarlet fever. The specific gravity is low, falling to 1010 or less, and casts, both granular and hyaline, are present. When the kidneys are seriously implicated, the quantity of urine is usually notably diminished. Great diminution is a serious symptom, and it often precedes the fatal issue.

In favorable cases the albuminuria does not in the average continue as long as in scarlet fever. The albumen may disappear from the urine in two or three days if its quantity has been small, and in a large proportion of cases it disappears within ten days; but cases occur in which albuminuria continues many months, with its final disappearance and the complete restoration of the health. Thus, a boy of six years treated by me had nephritis following a very mild attack of diphtheria. His urine in the first weeks was deeply tinged by the presence of red blood-corpuscles, but its quantity was normal, as determined by daily examinations, and it contained nearly or quite the normal amount of urea. Its specific gravity was at or under 1010. After a time the blood-corpuscles disappeared, the urine when not heated had its normal appearance, its specific gravity became normal, and the granular casts at first present disappeared. The patient was uniformly cheerful, was free from fever, his appetite was good, and no subjective symptoms occurred to indicate renal disease. Nevertheless, after the lapse of ten months, a little albumen was still present in the urine.

But the presence of albumen in the urine, if considerable, is an unfavorable prognostic sign. Sanné states that in two hundred and thirty-three cases of diphtheria accompanied by albuminuria one hundred and forty-two died and ninety-one recovered. In one hundred and sixty cases in which albuminuria was absent, sixty-three died and ninety-seven recovered. The statistics of others correspond with those of Sanné: so that the fact may be considered established that a larger proportion of cases of diphtheria with albuminuria perish than of those without albuminuria. It does not follow necessarily from this that the affection of the kidneys which produces the albuminuria contributes to the fatal result, for albuminuria is more frequent in grave cases than in those of a mild type. The gravity which leads to a fatal result may be due, and often is largely due, to other causes than the renal disease.

Although severe and so-called malignant forms of diphtheria are more likely to be complicated by albuminuria than are mild forms of the disease, yet, as in scarlet fever, severe and fatal renal disease giving rise to albuminuria sometimes occurs in very mild cases of diphtheria. Several years ago I attended a child of six years with the following history. He had mild pharyngitis, with scarcely appreciable exudation, and almost no constitutional disturbance. On the second day the patient seemed so nearly well that both the doctor and the intelligent grandmother who had charge of him did not think further medical attendance necessary. One week subsequently I was summoned to the child in haste on account of nearly complete suppression of urine. About one drachm was passed each time, and at long intervals. This when heated became semi-solid. The late Prof. Austin Flint, who saw the case in consultation, and myself notified the family of the extreme gravity of the case and its approaching fatal termination, a prediction which was verified in forty-eight hours. In such rare cases, while the diphtheritic poison acts with great power upon the kidneys, producing a fatal nephritis, its influence is feebly felt in those tissues which are the usual seat of diphtheritic inflammation. Diphtheritic albuminuria is rarely attended by anasarca or by symptoms of uræmic poisoning. In two hundred and twenty-four cases of diphtheritic albuminuria embraced in Sanné's statistics, dropsy occurred in only seven. Trousseau did not meet it oftener than in one case in twenty. Its infrequency has been attributed to the fact that only one kidney, or only portions of the kidneys, have been affected, the sound portions performing sufficiently the excretory function.

Oertel says, "The albuminuria of diphtheria is referable to many causes, of which the virus circulating in the blood is only one. Cardiac failure, respiratory difficulty, the febrile process, are adequate for the production of this symptom. The kidneys in cases where albuminuria has been present may be quite normal, or, on the other hand, they may exhibit varying degrees of parenchymatous inflammation."[1] The two common causes appear to be passive congestion of the kidneys, as of other organs, occurring during the dyspnœa of croup, or from heart-failure, the albumen escaping from the over-distended renal veins, and parenchymatous nephritis, in which the tubules contain detached and disintegrating epithelial cells. In parenchymatous nephritis granular casts are commonly present.

As regards prognosis, writers agree that diphtheritic albuminuria in itself does not tend to a fatal result in most cases, the unaffected portions of the kidneys, as stated above, being sufficient for the excretion of the deleterious products, especially the urea, whose retention in the system would involve danger. Therefore Sanné says "that diphtheritic albuminuria is an epiphenomenon which in the vast majority of cases remains without influence upon the course of the disease." But cases do occur, as we have seen by the history related above, in which fatal albuminuria, or fatal nephritis producing albuminuria, does take place as a complication or sequel of diphtheria.

---

[1] Synopsis of Oertel's monograph, London Lancet.

Unruh in 1881[1] expressed the opinion that the albuminuria of diphtheria results from a simple transudation. But more exact microscopic examinations show that it is only in cases of croupal asphyxia or heart-failure that that degree of passive renal congestion occurs which leads to a transudation of serum. When there is no obstructed respiration, and no marked weakness of the pulse, the albuminuria is a result and symptom of infectious nephritis. Prof. Bouchard[2] states that infectious nephritis, whatever the cause or source of the infection, is a parenchymatous nephritis. Says he, "The kidneys are sometimes augmented in volume and weight. Their capsule has the ordinary appearance and adherence. The cortical substance appears sometimes grayish, sometimes congested and sprinkled with whitish tracts. The medullary substance preserves its normal aspect. In kidneys thus changed microscopic pathological anatomy reveals integrity of the tubes of Henle, catarrhal change of the straight tubes, and to a considerable extent of the convoluted tubes. In the convoluted tubes the epithelial cells remaining in place are swollen and sodden together. The cellular mass is entirely granular. . . . Not only are the convoluted tubes obstructed by granular cells, but they are filled in some points by colloid matter or by blood. The glomeruli appear healthy; but we have seen the glomerular capsule distended with blood. In another case Renaut has seen it distended by colloid matter." Brault[3] has observed in diphtheritic albuminuria intense congestion of the capillaries of the tubules and glomeruli, altered epithelial cells, and transuded blood-elements indicative of parenchymatous inflammation.

Paralysis.—Another very important symptom and sequel of diphtheria is paralysis. It has diagnostic and prognostic value. Writers in medicine prior to the sixteenth century were either ignorant of diphtheritic paralysis or they vaguely alluded to it when they described the extreme debility which sometimes accompanies or follows diphtheria. No clear and certain allusion to it has been discovered in medical literature until near the close of the sixteenth century. According to Sanné, Nicholas Lepois referred to it in 1580, and Miguel Heredia in 1690. Ghisi, in a letter describing the epidemic which occurred in Cremona on the north bank of the river Po in 1747–48, writes of his own son, who had paralysis in a severe form following diphtheria, "I left to nature the cure of the strange consequences, . . .

---

[1] Jahrb. für Kinderheilk.

[2] Revue de Médecine, 1881.

[3] Jour. d'Anat. et de Phys., Nov. 1880.

which had been remarked in many who had already recovered, and which had continued for about a month after recovery from the sore throat and abscess. During this period, this child spoke through the nose, and food, particularly that which was least solid, returned through the nares, in place of passing down the gullet." In France, also, diphtheritic paralysis began to attract attention at or about the time when Ghisi, in Italy, wrote the above. Chomel, in 1748, described two cases, following what he designated gangrenous sore throat. The first patient, he says, had not quite commenced convalescence at the forty-fifth day of the disease, having still difficulty in articulating, speaking through the nose, and having the uvula pendulous. In the second case the patient became squint-eyed and deformed, but day by day as his strength returned he regained his natural appearance.

In America, in 1771, Dr. Samuel Bard, of New York, also related a case of this form of paralysis. A girl of two and a half years had recovered from a diphtheritic sore throat, and a diphtheritic pseudo-membrane upon the skin following the application of a blister had disappeared, when her convalescence was retarded by paralytic symptoms. "Whenever," says Bard, "she attempted to drink, she was seized with a fit of coughing; yet she was able to swallow solid food without any difficulty. She improved, but in the second month she could scarcely walk or raise her voice above a whisper."

From the time of Chomel, Ghisi, and Bard, more than half a century elapsed during which diphtheritic paralysis attracted little attention, though Jurine and Albers alluded to it in 1809. It cannot be doubted that cases occurred in this long period wherever diphtheria prevailed, but it might have been of such a type that the paralysis was infrequent, for Bretonneau, although he was familiar with Ghisi's and Bard's writings, did not recollect that he had seen a case of diphtheritic paralysis prior to 1843. Although a close observer of diphtheria, the paralysis had not been observed by him, or at least had not attracted his attention, until it occurred in the person of his townsman Dr. Turpin in 1843. Twelve years subsequently, in 1855, Bretonneau had made a sufficient number of observations to convince him that diphtheria frequently gave rise to a peculiar form of paralysis, and in his writings of this year he called the attention of physicians to this fact. But the opinions expressed by the eminent physician of Tours did not gain general acceptance until his friend and admirer Trousseau, at first distrustful of the existence of such a paralysis, had made a series of observations which fully established in his mind the theory of Bretonneau. His remarks on this subject, published in his "Treatise on Clinical Medicine," are interesting as showing how gradually important truths are revealed in medicine. He had seen as far back as 1833 a marked case in the service of Récamier in the Hôtel-Dieu, and another equally severe and typical case in 1846, but it was a long time before he recognized this ailment as one of the results of the diphtheritic poison. Says he, speaking of the cases seen in 1833 and 1846, "They were a dead letter to me, yet I was acquainted with the case described by Dr. Turpin of Tours. Bretonneau related it to me, and said that it was a case of diphtheritic paralysis. The statement seemed to me incredible. I refused to see anything more in the case than a coincidence. . . . It was not till about the year 1852 that, enlightened by new cases, better studied and better interpreted, I understood diphtheritic paralysis as Bretonneau understood it. From this time, whenever an opportunity occurred, I in my turn called the attention of my colleagues to this important subject." The clinical teachings and observations of Bretonneau and Trousseau were widely read, and the profession throughout the world soon recognized the fact that diphtheria often gives rise to a form of paralysis which, if not peculiar to it, is yet rare in other infectious diseases. Since these observations of Trousseau were published, many observations have been made and many monographs on diphtheritic paralysis have been written by such men as Roger, Germain Sée, Herman Weber, Charcot and Vulpian, Gubler, Landouzy, Suss, H. von Ziemssen, A. Jacobi, and W. H. Thomson. But the nature of this paralysis and the manner in which it occurs are still undetermined. The fact that there is such a paralysis was slow in gaining acceptance in the minds of physicians, and so the cause and pathology of the paralysis are still not fully ascertained.

Clinical History.—The statistics of different writers vary in regard to the frequency of diphtheritic paralysis. Probably it is different in different epidemics, and some observers may overlook the milder cases, which soon recover, and which are indicated by a slight impediment in swallowing and a slight nasal intonation of the voice. We may accept as approximating the truth as regards its frequency the following statistics of well-known and painstaking clinical instructors, who would be likely to detect the mildest forms of paralysis. In nine hundred and thirty-seven diphtheritic cases observed by Cadet de Gassicourt, paralysis occurred in one hundred and twenty-eight. Sixteen and six-tenths per cent. of Roger's cases of diphtheria had paralysis, and eleven per cent. of Sanné's cases.

But it must be borne in mind that, since paralysis is in most instances post-diphtheritic, those severe cases which are speedily fatal from blood-poisoning or croup do not live long enough to suffer from it, and such cases would be more likely to have the paralysis, if they lived, than the milder cases, which recover. Hence it has been estimated that, if all diphtheritic patients lived sufficiently long, one in every four, or even one in every three, would exhibit paralytic symptoms.

Time of Commencement.—In most instances the paralysis does not begin until the period of apparent convalescence from diphtheria, and the pseudo-membrane has nearly or quite disappeared. Sanné says it most frequently appears from eight to fifteen days after recovery, the limit perhaps extending to thirty days, but he adds that it may appear from the fifth to the eleventh and even as early as the second or third day of diphtheria. Cadet de Gassicourt states that in twenty of his cases the paralysis began before the disappearance of pseudo-membrane, most frequently about the seventh or eighth day of diphtheria. In two it commenced on the third day, and once in a prolonged diphtheria it began as late as the thirty-fifth day, the pseudo-membrane still being present. Usually, according to my observations, when paralysis follows diphtheria the nasal voice and some impediment in swallowing are observed early in the stage of convalescence, and at a later period muscles remote from the fauces may or may not be affected. Dr. L. E. Holt exhibited to the New York Clinical Society in December, 1887,[1] a child of two years who had diphtheria in August and a second attack in the middle of October. She convalesced slowly, and in her convalescence had no paralytic symptoms, except a nasal voice, until December 1, when multiple paralysis suddenly developed. A brother of this patient also had diphtheria in October, moderately severe, and early in convalescence paralysis of the muscles of the palate began, followed by that of other muscles; but it was not until the middle of December that the lower extremities were paralyzed. These cases are examples of the usual mode of commencement and extension of the paralysis.

Diphtheritic paralysis is, therefore, with few exceptions, a late symptom of diphtheria, or a sequel; but Dr. Boissarie[2] has related cases in which the paralysis was not preceded by the ordinary symptoms of diphtheria, and which, so far as I am aware, are unique. An officer in the police had been ailing two or three days: he had a nasal voice, and drinks returned through the nose. On inspection, the velum palati was found insensible and motionless, but the fauces were otherwise in their normal state. In the hospital alongside the barracks in which the above case occurred, a young man without fever, redness, or swelling of the fauces had also a nasal voice, and return of liquid food through the nose. The porter of the hospital was similarly affected, and the doctor stated that certain other patients in like manner presented symptoms of paralysis, without the history of an antecedent diphtheria. Dr. Reynaud, called in consultation, expressed the opinion that the paralysis had a diphtheritic origin; and this opinion was strengthened by the occurrence immediately afterwards of an epidemic of diphtheria in the place where these cases occurred. It appeared as if the diphtheritic poison had attacked the kidneys without manifesting its action in any other part of the system. Certainly such remarkable cases should have been more minutely examined. It is remarkable, inasmuch as diphtheria is so widely spread and so closely studied, that, if paralysis is sometimes the only manifestation of the operation of the diphtheritic poison, other similar cases have not been observed and reported. It is, in my opinion, more probable that in the above cases diphtheria had occurred of so mild a form that it escaped notice. I have related elsewhere a case in which diphtheritic albuminuria was preceded by diphtheria of so mild a form as regarded the usual manifestations that it nearly escaped detection, and yet the renal complication or sequel was so severe that death resulted. In another instance a little girl, not complaining of herself, left a call for a visit to her brother, whom I found with diphtheria of rather a severe type. At the time of my visit she was playing with other children in the street, and it occurred to me to call her in and examine her throat. To the surprise of the family, the characteristic diphtheritic patch was observed over one tonsil. Such mild walking cases are not infrequent in New York City, where diphtheria, established for many years, is constantly present, sometimes pernicious, and speedily fatal, but in other instances having a type at the extreme of mildness and with no evidence of blood-poisoning. All physicians who have had much experience with diphtheria, as in localities where it is naturalized or endemic, can recall cases in which a sequel of diphtheria, such as paralysis or albuminuria, has led to an accurate diagnosis of a pre-existing throat-affection which was so mild that its true nature was not suspected. In this respect diphtheria resembles scarlet fever, which also presents an equally variable type, from extreme mildness to a fatal severity. Hence it seems probable that in Boissarie's cases diphtheria of so mild a form that it escaped notice had preceded the paralytic manifestation.

---

[1] New York Medical Journal, Dec. 1887.

[2] Gazette Hebdomadaire, 1881.

The paralysis, as a rule, affects both motor and sensory nerves. Thus, in paralysis of the velum and pharynx, anæsthesia more or less marked occurs of the velum, the isthmus of the fauces, and the walls of the pharynx, in addition to the motor paralysis. In the more severe cases, anæsthesia with absence of reflex action occurs not only over the entire pharynx, but also over the epiglottis. The combination of motor and sensory paralysis should be borne in mind in studying the cause and nature of the ailment. The muscles affected by diphtheritic paralysis atrophy as in other forms of paralysis. Dr. H. von Ziemssen[1] says that such marked atrophy does not occur in any other disease, except in acute poliomyelitis and saturnine paralysis.

The symptoms and course of diphtheritic paralysis vary according to its location and the muscles affected. Therefore we will sketch the clinical history of its various forms separately, beginning with that which is first in time, most frequent, and least dangerous.

1. *Loss of the Tendon Reflexes.*—In 1882, Dr. Buzzard made the observation that the knee-jerk is absent in cases of diphtheritic paralysis. Bernhard[2] stated that loss of knee-jerk may precede other nervous symptoms, or may occur without other symptoms indicating impairment of the nervous system. He also stated a fact now generally admitted, that the loss of knee-jerk may have a diagnostic value in indicating the diphtheritic nature of a pre-existing obscure disease. But the profession in this country had little knowledge of the loss of the tendon reflexes in diphtheria until Prof. R. L. McDonnell, of the Montreal General Hospital, read a paper on this

---

[1] Klinische Vorträge, 1887, No. iv.

[2] Virchow's Archiv, Bd. xcix.

subject before the Canada Medical Association, August 31, 1887, and published it in the *Medical News* of Philadelphia in the following October. Dr. McDonnell's observations relate to eighteen cases of diphtheria admitted into the General Hospital. Of these eighteen patients, ten had loss of knee-jerk at the time of admission, while in the remaining eight it was present. The cases observed by the doctor were sufficient, he believed, to enable him to make the following statement: *Knee-jerk, in many cases of diphtheria, is absent from the very first day of the illness.* It is a noteworthy fact that in most of the cases detailed by McDonnell, in which there was loss of the tendon reflex, other forms of paralysis subsequently appeared.

Since the publication of Dr. McDonnell's paper, many observations have been made confirmatory of his statement. At a meeting of the New York Clinical Society, held December 23, 1887, Dr. L. E. Holt exhibited a brother and sister of five and two years, with multiple paralysis, who had lost the knee-jerk, and the examination of one of them showed complete loss of the plantar reflex. Since the attention of the profession has been directed to the loss of the tendon reflexes, all observers admit that it is not only the earliest but also the most frequent of the paralytic symptoms, probably occurring in one-third to one-half of all cases under treatment. Dr. Angel Money, in a discussion before the London Clinical Society, September, 1887, stated that he had observed an initial increase of the knee-jerk, preceding its abolition. Dr. H. von Ziemssen remarks that, while the tendon reflexes are so often lost, the cutaneous reflexes are frequently exaggerated.

The loss of the tendon reflexes, while, as we have stated, it is the first in time of the paralytic symptoms, appears also to have the longest duration. In cases of multiple paralysis it seems to be the last to disappear. Thus, Dr. McDonnell states that the loss of knee-jerk in a boy of fourteen years continued four months, and in his two sisters it was still present when all other symptoms of the disease had disappeared.

2. *Palatal Paralysis.*—With the exception of the loss of the tendon reflexes, the most common form of diphtheritic paralysis is that in which the velum palati and muscles of the pharynx are affected. This form of paralysis is revealed by a nasal intonation of the voice, slow speech, snoring during sleep, difficult deglutition, and return of liquids through the nares. As the paralysis increases in severity and extent and the palato-glossus and constrictor muscles of the pharynx become paralyzed, the difficulty in swallowing increases. The patient finds it necessary to throw his head backward in swallowing and to swallow slowly and in small amount. The food descends in the œsophagus by its weight, and with but little aid from the pharyngeal muscles. On examining the fauces, we discover the velum relaxed and motionless, and the uvula, deprived of its tonicity, drops on the base of the tongue. On touching the uvula with the point of a pen or pencil, it is found to be insensible, no reflex action occurring. Sensory paralysis occurs, as a rule, in typical cases, the patient experiencing no pain when

the parts are pricked with a pin or other instrument. The fauces should be inspected and tested from day to day, in order to determine the progress of the paralysis. In mild cases it may be limited to the velum and palate, but it frequently extends to the epiglottis and upper part of the larynx, so that in attempts to swallow, portions of the food enter the larynx, exciting a cough. The affected muscles may regain their use in less than a week, but frequently from one to two months elapse before their function is restored.

Palatal paralysis terminates favorably, with few exceptions, if the patients are otherwise in good condition; but if there be much prostration from the antecedent diphtheria and from the dysphagia, death may occur from inanition. Cadet de Gassicourt has cited two cases of death from this cause, although life was probably prolonged by feeding through an œsophageal tube introduced through the nostrils. Rarely, also, death has occurred from the descent of food into the air-passages and the plugging of a bronchus. Tardieu and Peter have each related a case of this mode of death. As a chief function of the velum palati is to close the posterior nasal fossæ during deglutition, food, especially if liquid, is liable to be returned through the nostrils until the function of the velum is restored.

3. *Multiple Paralysis.*—This form of paralysis is commonly preceded by loss of the tendon reflexes. In most instances it begins with loss of power in the muscles of the palate; but exceptions occur. Cases are reported in which the muscles of the eye, those of motion and of accommodation, are first paralyzed, the palatal muscles being unaffected or subsequently attacked. Trousseau has stated that in cutaneous diphtheria the first loss of muscular power is sometimes in the lower extremities instead of in the palate; and other observers have recorded cases in which multiple paralysis commenced in one or more of the extremities. Therefore the order of the paralytic seizures differs in different cases, and muscles are affected in one patient that escape in another. The degree of paralysis varies in different muscles. In some the loss of power is complete, while in others it is partial. When the lower extremities are entirely motionless, the patient frequently has considerable use of the upper extremities.

Even in the severest cases many groups of muscles entirely escape. Therefore I prefer the term multiple paralysis to the term general paralysis employed by some writers to designate this form of the disease.

Trousseau speaks of what he designates the mutability of diphtheritic paralysis. He says the paralysis which occupies one limb disappears in this limb, to manifest itself in another. "The numbness, for example, which the patient has been experiencing in one leg will suddenly cease, and become greater in the other leg. To-day the right hand will not give a dynamometric pressure of more than ten or twelve kilogrammes, and to-morrow its power will have augmented, while that of the left will have diminished; then the parts which were first affected are a second time attacked, and become more affected." Even the dysphagia may vary on different days, as Cadet de Gassicourt has stated. He relates the case of a child of three and a half years in whom the velum palati suddenly resumed its function: the head, which had dropped from paralysis of the muscles of the neck, became erect, the patient was able to sit, and the upper extremities recovered their power, but the improvement was of short duration, the paralysis returning as at first. These sudden and unexplained variations in the degree of paralysis resemble, says Trousseau, the mutability of paralysis in hysteria. Among the most noteworthy of the paralyses resulting from diphtheria are those pertaining to the eye. The media and retina are unaffected, but the levator palpebræ, the muscles of accommodation, and the motor muscles of the eye are paralyzed in certain patients, so as to cause dropping of the eyelids, strabismus, and indistinct vision. In addition to the muscles already mentioned, various muscles of the trunk, of the neck, the sphincter ani, and the sphincter vesicæ are sometimes paralyzed, producing deformity and incontinence of urine and fæces. The paralysis of the muscles of accommodation is usually such that patients become presbyopic, seeing distinctly distant but not near objects.

The muscles of the face are also occasionally paralyzed. Many observers have related cases of facial hemiplegia. When general paralysis of the facial muscles occurs,—fortunately, a rare event,—whatever the mental state, however great the excitement, the features are entirely devoid of expression; the aspect is dull and idiotic; the face is flabby and motionless; the lids and lips droop; saliva flows from the mouth, and speech is slow and difficult. At the same time the mental faculties, deprived of the usual mode of expression, are sound and active.

But the most accurate idea of the symptoms of multiple paralysis can be imparted by the narration of a case; and I select for this purpose the graphic description of this form of paralysis published by Dr. C. W. Fallis in the *Medical Summary* for January, 1888. He describes the ailment as it occurred in his own person, as follows. "About three weeks after the subsidence of the disease [diphtheria] the paralytic symptoms began to show themselves. Impaired vision was the first trouble noticed, inability to accommodate the eyes to near objects, and in taking up the paper to read, one morning, I found I could scarcely see a word, and soon after, although distant objects could be seen as well as ever, high-power glasses were required to read any kind of print. Double vision was noticed afterwards. At about the same time numbness of the tongue was felt, the muscles of deglutition became paralyzed, so that swallowing was attended with strangling and regurgitation of food through the nose. There was a rapid pulse, 120 to the minute, showing that the pneumogastric was involved. Weakness of the limbs, causing a staggering gait, appeared; fingers became weak and numb, so that small objects could not be picked up, the symptoms becoming worse and worse as the disease progressed. The muscles of the left side of the face became affected with all the symptoms of facial paralysis from organic disease. Motion became more and more impaired, till I could neither stand nor walk, and when at the worst I was perfectly helpless, could not feed myself, had to be lifted from chair to chair, turned in bed, and could not even lift my hand to my head, or throw one limb over the other. Sensation was so impaired that hands and feet felt like lifeless weights, and in the dark I could not tell whether my feet were on the floor or not. The muscles of respiration were at no time affected to such an extent as to render breathing difficult, and the power of perfect speech was retained. Paralysis of the bowels necessitated the use of warm-water injections to promote their action. Some of the symptoms abated, while others became more aggravated, those first to appear being generally the first to subside: however, the smaller-sized muscles recovered rapidly, while the large fleshy ones were more tardy in reaching their normal state, the facial paralysis lasting but a few days, while locomotion was either labored or impossible for many weeks. The course of the disease from the beginning to the worst stage was about nine weeks, when it remained stationary for two weeks. Improvement was at first very slow and tedious, but after I could walk a little it was much more rapid, and by the fifteenth week, with the exception of some weakness, I was well."

Multiple paralysis not infrequently continues from two to six months. As might be expected, the prognosis is less favorable when the paralysis is multiple than when it is restricted to the velum and pharynx. In thirteen cases observed by Cadet de Gassicourt, six died.

4. *Cardiac Paralysis* (the *cardio-pulmonary paralysis* of certain French writers).—In cases of the first, second, and third forms of paralysis which have been considered above, the vital organs are not directly involved. These paralyses, however inconvenient they may be, are not directly fatal. The paralysis which we are about to consider presents a very different clinical aspect, inasmuch as the organs affected are among the most important in the system, a serious impairment of their functions rendering death inevitable.

Physicians who have had experience in the treatment of diphtheria have met cases in which symptoms, usually of sudden development, indicated dangerous heart-failure. Perhaps the patient has been gradually improving, the pseudo-membrane has nearly or quite disappeared, the temperature is not far from normal, the swallowing is better and more nutriment is taken, the family are cheerful in the prospect of a speedy recovery, and the physician expects soon to discharge the patient cured. Suddenly the scene changes. The pulse becomes feeble and abnormally slow or rapid,—it is usually at first slow and subsequently rapid,—the respiration is superficial, and the surface becomes pallid, often slightly cyanotic. In the more favorable of these cases the patient may rally by active stimulation, and perhaps he eventually recovers, or after some hours or a day or two of comparative comfort he succumbs to a return of heart-failure. There is no other disease in which these sudden, unforeseen, and fatal attacks of heart-failure occur so frequently as in diphtheria. There is no other disease in which physicians are so frequently deceived in their prognosis, for various reasons, but largely on account of the occurrence of these unexpected attacks of heart-weakness.

But a clear and accurate idea of the clinical history of these cases of sudden heart-failure can be best imparted by the relation of typical cases. For this purpose I will briefly narrate cases occurring in the hospital service of one of the most trustworthy clinical teachers of the present time, M. Cadet de Gassicourt; though I believe that all physicians who have been several years in practice where diphtheria is prevailing can recall to mind cases equally striking and typical. I select his cases on account of the completeness of his records.

A child of two years entered Cadet de Gassicourt's service on January 3, with diphtheritic pharyngitis of ten days' continuance. The tonsils were large, still covered with pseudo-membrane, and the submaxillary glands were also enlarged. He had no laryngeal symptoms, and his urine was without albumen. On the following day the velum and pharyngeal muscles were slightly paralyzed, the speech nasal, and deglutition moderately embarrassed. He was quiet during the night of January 4 and in the morning of the 5th, but at ten A.M. he became chilly, his face and extremities feebly cyanotic, and slight dyspnœa and dilatation of the alæ nasi were observed. His pulse, at first abnormally slow, became rapid, he was agitated, uttered loud screams of distress, and fell back cyanotic and dead. The death-struggle did not occupy more than one minute. Another infant, also two years of age, entered the same service, having diphtheritic pharyngitis of two days' continuance. The fauces presented the usual red appearance, the tonsils were swollen and covered with a thick exudate, but there was no albuminuria nor croupiness. Two days later the pseudo-membrane had diminished, but the velum palati was paralyzed. On the following day the general appearance was satisfactory, and the pseudo-membrane had still further diminished. At eight P.M. the infant was suddenly seized with vomiting, accompanied with great dyspnœa, rapid pulse (160), and a cyanotic hue of the face and extremities. He was restless, and uttered cries of distress. Two hours later he screamed loudly, raised himself in bed, and fell back dead. A child of five years was admitted with diphtheritic pharyngitis of two days' continuance, having enlarged tonsils covered with pseudo-membrane, and enlarged cervical glands, but without cough or albuminuria. Seven days later, the ninth of the disease, the pseudo-membrane had disappeared, but the velum palati was paralyzed. On the following day there was little change, except occasional vomiting, but the general state was good, and sleep tranquil. At seven A.M. on the following day, the eleventh of the disease, after a calm night, the child uttered two or three cries, the pulse became rapid, the respiration embarrassed, the features, extremities, and finally the entire surface, cyanotic, and at eight A.M. death occurred quietly.

The similarity of these three cases is apparent. Paralysis of the velum and palate had continued in the first case eighteen hours, in the second case thirty-six hours, and in the third case forty-eight hours, when suddenly the heart and lungs were greatly embarrassed in their functions, and death occurred within one hour from the commencement of the severe symptoms. The agitation, repeated cries of distress, and the shrill cry that preceded death, indicated extreme suffering.

Severe pain, præcordial, epigastric, or abdominal, is present in some if not in most of these cases of sudden heart-failure, as we shall see from cases presently to be related. It was probably experienced by these three patients, who were too young to express clearly their subjective symptoms.

Gombault made a minute microscopic examination of the affected organs in these three cases, after the tissues had been properly hardened by chemical agents. In one of the cases he examined the pneumogastrics and myocardium, and both were found in their normal state. As regards the nervous centres, the anatomical changes were alike in all three. In the spinal cord lesions were found at the origin of the anterior roots of the spinal nerves, characterized by fragmentation of the medullary substance in the nerve-fibres, numerous granules and minute globules appearing in this substance and occupying its place.

In addition to this, undue swelling of the axis-cylinders was observed. In the three cases the gray substance in the anterior cornua had undergone a sort of rarefaction, the microscopic sections being more transparent and the elements in the section being wider apart than in the normal state. No meningitis or injury of the blood-vessels was observed in the spinal columns, but numerous nerve-cells were deprived of their prolongations. The medulla oblongata, the centre and source of the nervous supply to the heart, lungs, and stomach through the pneumogastrics, was also carefully examined in the three cases. Nothing abnormal was observed in this organ, except small masses of leucocytes in the vessels. The substance of the medulla oblongata and the nerve-fibres constituting the roots of the pneumogastrics seemed healthy. The small masses of leucocytes in the blood-vessels were not sufficient to obstruct the circulation, and the appearance of the blood-corpuscles was normal. Hence in the opinion of Gombault the small aggregations of leucocytes in the vessels had no effect on the innervation of the thoracic organs derived from the medulla. The points of special interest in the microscopic examination of the three cases were the apparently healthy and normal state of the pneumogastrics and myocardium in the one case in which they were examined, and of the medulla oblongata in the three cases, while the gray matter of the spinal cord, which has no immediate nerve-connection with the heart, showed marked degenerative changes.

The above are striking examples of sudden and fatal heart-failure occurring during apparent convalescence, when the symptoms of diphtheria appeared to be abating, with the exception of the paralysis of the velum and palate. The following cases presented a clinical history in some respects different. A child of eight years had been under treatment for diphtheria since February 9, 1883. On February 20 the membrane had disappeared, but slight paralysis of the velum and left upper extremity was observed, and the urine contained a little albumen. At three P.M. she was seized with severe abdominal pains, followed by vomiting, slow respiration, slow and feeble but regular heart-beat, imperceptible pulse, coolness of surface, and cyanosis. These symptoms increased, and at half-past six P.M. death occurred. The clinical history differed from that in the three cases related above in the fact that there was no agitation or moaning at the close of life, and that the heart-beat remained abnormally slow unless during the last moments. In another case paralysis of the velum and palate began on the third day of diphtheria, while the pharyngeal and nasal inflammations were in full activity. The urine was slightly albuminous. Three days subsequently, in the morning, the muscles of the nucha and right shoulder were paralyzed. At two P.M. the child complained of violent abdominal pains, followed by nausea and vomiting. The vomiting was partially relieved, but dyspnœa and a rapid heart-beat followed. The cyanosis increased until it extended over the entire surface, and death occurred three hours after the commencement of symptoms referable to heart-failure. A boy of five years had diphtheritic croup, for which tracheotomy was performed and the canula inserted. He subsequently did well for a time, but afterwards lost his appetite. On the eleventh day of the disease he had paralysis of the velum and palate. On the twelfth and thirteenth days the disease seemed to be stationary, and the child was quiet. Suddenly at seven P.M. on the thirteenth day multiple paralysis occurred. Liquid food taken by the mouth was returned in part through the nostrils, and a part entered the larynx and escaped from the tracheal opening. An hour later the muscles of the nucha, the arms, and both sides of the trunk were paralyzed, and the head dropped. At seven A.M. on the following day, vomiting, dyspnœa, cyanosis of the face and extremities, and a very rapid pulse occurred. The asphyxia increased, the pulse grew more feeble, the surface cool, and death took place three hours later.

Cases like the above are not infrequent in severe epidemics of diphtheria, but in some instances the loss of power in the heart occurs more gradually. A boy of twelve years had diphtheritic pharyngitis from which he was apparently convalescing. Some days after the disappearance of the inflammation, the velum palati and muscles of the pharynx were paralyzed. Then succeeded paralysis of the muscles of the nucha, of the muscles of accommodation, and of those of the upper and lower extremities. The march of the paralysis was for a time progressive. Then it seemed to recede; but the improvement did not continue. One month from the commencement of diphtheria, the child uttered plaintive cries, became motionless as if from general paralysis, and a state of asphyxia slowly occurred, accompanied by cyanosis. During the following night the patient lay in a stupor, and on the ensuing morning the features presented a cadaverous and slightly cyanotic hue, the extremities were cool and blue, the tongue pallid, moist, and of a normal warmth, the respiration hurried and without auscultatory signs of disease, the pulse feeble and rapid (148). Finally the sphincters were paralyzed, the urine and fæces escaping involuntarily. Within ten minutes after the above notes were written, the patient died of heart-failure. The feature of special interest in this case was the long continuance of multiple paralysis when the cardiac and pulmonary symptoms occurred.

Sudden heart-failure in diphtheria is usually fatal; but recovery is possible. Cadet de Gassicourt in his large clinical experience met one recovery to fourteen deaths. This case is interesting since the heart-failure preceded the palatal and other forms of paralysis, instead of being preceded by them, as is ordinarily the case. Twenty days after the commencement of diphtheria, and when in apparent convalescence, the patient was seized with extreme pain in the præcordial region, attended by a fall of pulse to 42. He had cold sweats, rigors, and vomiting. In one and a half hours these symptoms abated. Three days subsequently another similar attack occurred, and subsequently two others, but less severe than the first. On the twenty-eighth day from the beginning of diphtheria, and eight days after the syncopal attacks, paralysis of the velum and pharynx began, soon followed by paralysis of the vocal cords, of the muscles of accommodation, and of those of the extremities, which continued three months, when recovery was complete. Cases of recovery from sudden and alarming symptoms of heart-failure have also been related by Sanné, Billard, and others.

What is the cause of this sudden loss of power in the heart in diphtheria, occurring usually during apparent convalescence? Does it result from disease in the muscular structure of the heart, from thrombosis or ante-mortem clots in the cavities of the heart, or does it result from disease of the central organ of innervation, the medulla oblongata, or from disease and deficient conducting power in the important nerve which controls the heart's action, the pneumogastric, or in the branches which this nerve supplies to the heart as well as the lungs and the stomach?—for these three organs appear in most instances to be affected simultaneously.

Bouchut and Lagrave attribute sudden heart-failure in diphtheria to endocarditis; and yet it is very seldom that a *bruit* or heart-signs indicative of endocarditis have been observed during life. The belief in the occurrence of this inflammation is based on the appearance of the free edge of the mitral valve, and sometimes of the aortic valves in addition. They have appeared roughened as if from the presence of minute vegetations. At the same time the surface of the valves and the endocardial surface have undergone no appreciable change such as an endocarditis would be likely to cause. Since the announcement of the theory of Bouchut and Lagrave, and attention has been drawn to the subject, the roughened edge of the mitral and aortic valves, upon which their theory of an endocarditis as the causative agent of sudden heart-failure is based, has been found with equal frequency in children who have perished with other diseases. The late Prof. Parrot says Cadet de Gassicourt expressed the decided conviction that the roughening of the tips of these valves does not have an inflammatory origin, but is an anatomical peculiarity which originates in the fœtal development. Sanné says in reference to Bouchut and Lagrave's theory, "My personal investigations are absolutely negative. Observations of diphtheria, to the number of one hundred and forty-nine, taken in these later years, . . . have not furnished a single case of endocarditis. I should fear to express myself in such a positive manner if I should trust to the single testimony of my senses; but a large number of these patients were auscultated by Barthez and by D'Espine and Gombault. . . . The conclusion . . . therefore is that diphtheritic endocarditis is extremely rare, as pathological anatomy and clinical observation alike demonstrate." Therefore the theory which attributed sudden heart-failure to endocarditis has not been sustained by recent observations, and does not appear to be tenable.

Weakening of the heart's action in diphtheria, with sudden death as a consequence, has with more probability been attributed to granulo-fatty degeneration in the muscular fibres of the heart consequent upon a prolonged and severe diphtheritic attack. Oertel says, "When the general disease lasts long and is very intense, and especially in cases in which death is caused suddenly by paralysis of the heart, the muscle appears pale, soft, friable, broken by extravasations of blood, and on microscopical examination most of its fibres are seen to be already in an advanced stage of fatty degeneration."[1] Such degenerative changes if occurring in a considerable proportion of the muscular fibres of the heart would inevitably render the contractile power of this organ feeble, and perhaps inadequate. Still, if we regard it as a cause of sudden heart-failure, it can be regarded as such in only a relatively small number of instances, for in most cases the weakening of the power of the heart is sudden and during convalescence, —at a period, therefore, when degenerative changes are not likely to occur. In most of the recorded cases the contractile power of the heart does not appear to have been notably weakened previous to the attack of heart-failure, as it would probably have been were degenerative changes in the myocardium the sole or chief cause. The clinical history is as if the heart were suddenly overpowered by an agent of rapid—never slow—development. Moreover, in typical cases of sudden heart-failure the microscope sometimes reveals a healthy myocardium, as in one of the cases related above. We must look, therefore, for some other cause, although admitting that degenerative changes in the muscular fibres of the heart, when present, contribute to a weakened action of this organ.

Sudden heart-failure in diphtheria has also been attributed to cardiac thrombosis; but, as several writers have pointed out, the heart-clots are identical in appearance and kind with those found in the heart after death from other diseases than diphtheria. There is every reason for the belief that they occur during the death-struggle, and therefore are not the primary cause of the heart-failure, but are secondary or consecutive.

---

[1] Ziemssen's Cyclopædia, vol. i.

Among the most strenuous advocates of the theory that cardiac thrombosis is the common cause of sudden heart-failure and sudden death in diphtheria is Dr. Beverley Robinson, now a distinguished physician of New York, whose able thesis on this subject, published in 1871, when he was a resident of Paris, attracted much attention and is alluded to by nearly all recent French writers on this subject. But the opinion of most pathologists in reference to this theory is, I think, expressed by Cadet de Gassicourt in the following passages published in his clinical treatise: "I have often shown you these clots, and I have enabled you to see that they occur equally in children who have died of diphtheria, as well as in those who have succumbed to other maladies, in subjects struck with sudden death and in those who have not been attacked by any sudden casualty. This objection is in itself conclusive. You have been able to see also that the constitution of these clots does not have any of the characters which authors the most competent have assigned to clots formed during life: they are the clots of the agony." Sanné also writes in almost identical language.

In searching for the cause of sudden heart-failure in diphtheria, we must note the fact that, as a rule, in typical cases it is preceded by palatal and often multiple paralysis. The paralysis has continued for a time, extending perhaps from one group of muscles to another, when suddenly the heart passes under some powerful influence which restricts and overpowers its action. The theory of deficient innervation or a true cardiac paralysis appears most tenable under the circumstances. It affords the most satisfactory explanation of those unfortunately not infrequent cases in which death suddenly occurs during apparent convalescence from diphtheria, when the symptoms are fast disappearing, with the exception of the palatal or other paralysis. It affords best of all the theories an explanation of the occurrence of sudden death from heart-weakness in those obscure cases which have puzzled physicians, cases in which the post-mortem examination has revealed an apparently healthy state of the heart. The theory of an arrested or deficient innervation of the heart also furnishes an explanation of the occurrence of concomitant symptoms in these cases of sudden heart-failure, —such symptoms as vomiting, epigastric pain, and dyspnœa, or irregular respiration; for the heart derives its innervation from the same source as the lungs and the stomach,—that is, through the pneumogastric. For the reasons now given, we feel justified, in our classification of the forms of diphtheritic paralysis, to make a distinct class having the designation cardiac paralysis, or, to adopt in our language the French expression, cardio-pulmonary paralysis.

Etiology.—The four forms of diphtheritic paralysis—first, the abolition of the tendon reflexes, the most common, the earliest, and the least dangerous of all; secondly, palatal paralysis, which may occur as early as the third day of diphtheria, but is most common during its later stages, or in the period of convalescence; thirdly, multiple paralysis, in which various muscles throughout the system are paralyzed; and, fourthly, cardiac paralysis, the most dangerous of all—probably are produced by the same cause and have the same pathology in most instances. We may, therefore, in the following pages, in studying the cause and nature of diphtheritic paralysis regard the various forms which it exhibits as manifestations of one disease What is true of cardiac paralysis as regards its cause and nature we may assume to be true in reference to palatal and multiple paralysis and even the abolition of the tendon reflexes. The most dangerous and fatal paralysis, the cardiac, is, as we have stated above, in nearly all patients associated with the milder forms, showing that the same cause or causes are operative at the same time in the individual.

Gubler, in his memoir published in 1860–61, attributed paralysis of the velum and palate to disease of the terminal nerves produced by contiguity or propagation from the inflamed fauces, and he held that the same injury of the nerves and paralysis might result from any anginose inflammation, if severe enough. But this theory was short-lived, for physicians soon perceived that it was inadequate to explain the occurrence of paralysis at a distance from the inflamed surfaces, and palatal paralysis sometimes occurs after cutaneous and other forms of diphtheritic inflammation in which both the fauces and the nares entirely escape and remain healthy.

Trousseau, impressed with the inadequacy of Gubler's theory, directed his attention to the nervous centres. He was led to believe, from the fact that the paralysis usually terminates favorably, and because in certain fatal cases he was unable to discover any lesion sufficient to produce the paralysis in the brain, spinal cord, or meninges, that it did not occur from any structural change in the nervous system. Trousseau, an unsurpassed clinical observer, was not a microscopist, and, being unable to discover any anatomical cause of the paralysis, he relates the case of the crew of a vessel who were paralyzed by eating an eel which contained some poisonous ingredient, and, after alluding to instances of paralysis resulting from small-pox typhoid and typhus fevers, and cholera, continues, "Well, then, diphtheritic paralysis belongs to the same category: its real cause is the poisoning of the system by the morbific principle which generates the malady, on which the paralysis depends, and in regard to the mode of action of which in producing the paralysis we shall always perhaps remain in ignorance."

Since the time of Trousseau, many eminent pathologists have endeavored to discover the anatomical characters and elucidate the nature of diphtheritic paralysis by patient and thorough microscopic examinations. We have already detailed the microscopic appearances in Cadet de Gassicourt's three memorable cases. In 1862, Charcot and Vulpian stated that they had examined the nervous filaments in the velum palati paralyzed by diphtheria and found certain of them entirely free from medullary matter, granular bodies occupying its place; but partial degeneration was more common. In some of the fibres the medullary matter was intact. Lionville in 1872 stated that he had found degenerative changes in the phrenic nerve of a patient who had died of asphyxia following an attack of diphtheria. The contents of certain of the fibres constituting this nerve were amorphous, filled with granular bodies instead of the normal nerve-substance. Leyden in 1872 discovered lesions in the peripheral nerves and in the central organ, upon which he bases his theory of an ascending neuritis. Roger and Damaschino in 1875 examined the nervous system in four children who had died of diphtheritic paralysis, and found atrophy of the nerve-fibres in the peripheral nerves. The medullary matter appeared granular in certain points, and in others it had entirely disappeared, while the axis-cylinder was not notably altered.

Such observations, to which others might be added, have fully established the fact of peripheral nerve lesions, such as would be likely to result from a neuritis, in the paralysis of diphtheria; but it must be borne in mind that the various observers, while they report degenerative changes in certain of the nerve fibres or tubes in the peripheral nerves of the paralyzed part, also state that others in the same nerves were to appearance normal and capable of performing their function. Such are the facts upon which the theory that diphtheritic paralysis is caused by peripheral nerve lesions, a peripheral neuritis, is based.

In the endeavor to elucidate the cause of diphtheritic paralysis, attention has also, as might be expected, been directed to the state of the brain and spinal cord, and anatomical changes have been discovered in them quite as marked as in the peripheral nerves. Buhl, Roger and Damaschino, Pierret, Vulpian, Déjerine, and Oertel discovered in different cases, in the brain and spinal cord, in those who died of paralysis, various anatomical changes, among which we may mention small extravasations of blood and slight softening in the cerebral substance, extravasations of blood and thickening of the neurilemma in the roots of paralyzed nerves (Buhl), endo- and peri-neuritis at the point of origin of the affected nerves, thickening of the walls of the vessels and accumulation in them of white corpuscles (Pierret), rarefaction of the connective tissue and degenerative change in the nerve-cells in the anterior cornua of the cervical and upper dorsal region of the spinal cord (Vulpian), atrophy and granular degeneration and fragmentation of the myeline in the nerve-tubes in the anterior roots of the spinal nerves, increase of nuclei in the white substance of Schwann, disappearance of the axis-cylinder and slight fatty degeneration of the walls of the capillaries (Déjerine.)

Déjerine in the microscopic examinations of five cases of paralysis discovered anatomical alterations in the gray substance of the spinal cord, the white substance being intact. He observed in the gray substance cells atrophied or in process of atrophy, with the disappearance of their prolongations, so that healthy cells were comparatively infrequent. The cells seemed to have undergone the change which occurs in acute or subacute myelitis. The vessels in the gray substance were dilated and flexuose. They were in a state of hyperæmia or congestion, and at points small intestinal hemorrhages had occurred. Around the central canal and in the commissures the nuclei were increased. The white substance of the spinal cord presented the normal appearance. These anatomical changes in the cord apparently resulted from a myelitis. The spinal nerves whose roots originated in the diseased gray matter of the cord were found to have undergone a similar change in their peripheral distribution. Therefore in the five cases in which such minute examinations of the nervous system were made, the lesions in the cord and the nerves were similar.

In 1883, Dr. E. Hyla Greves, of Liverpool, Pathologist to the Royal Infirmary, obtained permission to examine the spinal cord in a child of three years who had died of sudden heart-failure after having suffered from an aggravated form of multiple diphtheritic paralysis. She had had anæsthesia of the fauces and all her extremities, liquid food regurgitated through her nostrils and entered her larynx, she passed urine and fæces in bed, she could not stand or sit without support, her head dropped helpless, her speech was indistinct, her tongue could not be protruded, her respiration was slow and shallow, her pulse 50 per minute and feeble, and she was nourished by enemata of pancreatized milk. The paralysis increased so that the diaphragm alone acted in respiration, the pulse became slower, irregular, and more feeble, and death occurred suddenly. At the autopsy, which was limited to the spinal cord, the veins of the lower part of the cord were much congested; the white substance of the cord presented the normal appearance to the naked eye, but the gray matter of the lumbar and lower dorsal regions was extensively softened, and in the left half of the cord diffluent, so as to flow from the section, leaving a cavity. Higher up in the cord the gray substance was hyperæmic, but not diffluent. The diffluent gray matter was unsuitable for microscopic examination, but other portions of the cord were examined, with the following result: many ganglion-cells of the anterior cornua were destroyed or in the state of "cloudy swelling;" others had lost their processes and were reduced in size; increase in the number of nuclei in the neuroglia throughout the cord; gray substance in the right half of the cord in an early stage of softening; in the dorsal and cervical regions everywhere the ganglion-cells were in a state of "cloudy swelling." No appreciable change in the white matter of the cord. It is evident that this was an extreme and rare case of degenerative change in the cord, and one in which the paraplegia, had the patient lived, would have been permanent, for the diffluent gray matter in the cord could not have been restored to its normal integrity. It was not, therefore, an ordinary case, inasmuch as the paralyzed muscles, as a rule, recover their function in those who survive.

Such is a summary of the lesions, peripheral and central, in the nervous system, which have been discovered in fatal cases of diphtheritic paralysis. We have presented the facts upon which the theory of the cause and nature of this disease must be based. Are we able to present a theory which will hold good in regard to cardiac paralysis characterized by sudden heart-failure, to pulmonary paralysis characterized by superficial or embarrassed respiration, to palatal and multiple paralyses, with their many inconveniences, and to the loss of the tendon reflexes?

Must we, with Trousseau, rest satisfied with the belief that the manner in which diphtheria produces paralysis is beyond our comprehension and will probably never be known? Dr. Abram Jacobi, seeing the inadequacy of the various theories to explain all cases or forms of diphtheritic paralysis, wrote in 1880 as follows, in his classical treatise on diphtheria: "It may be positively asserted that diphtheritic paralysis does not in every case depend on one and the same cause."

The theory which is most strongly advocated at the present time, and which appears to be accepted by a large proportion of the specialists in nervous diseases under the lead of Charcot, is, as we have stated above, that diphtheritic paralysis results from a peripheral neuritis. Others, observing central lesions in the nervous system, have naturally inferred that they have an important share in the production of the paralysis. It is very important that the practitioner when confronted by this grave malady should have a clear conception of its cause and nature, that he may be better able to apply the appropriate remedies. We will, therefore, examine with the light obtained from clinical experience the prevailing theory that diphtheritic paralysis results from anatomical changes, peripheral or central, or both, in the nervous system. Is this theory adequate to explain the paralysis as it commonly occurs? We will give a brief summary of the objections to it, at the risk of repeating what we have already stated.

1. Cases occur in which carefully-conducted microscopic examinations reveal an apparently normal state of the nerve supplying the paralyzed part, and also of the nervous centre from which this nerve originates.

Thus, in the three cases of typical cardiac paralysis described above occurring in the practice of Cadet de Gassicourt, the pneumogastric and its branches examined in one case appeared normal, and no lesion sufficient to cause paralysis was found in a careful examination of the medulla oblongata, the central organ of innervation of the heart.

2. Palatal paralysis sometimes occurs as early as the second or third day of diphtheria, and loss of the tendon reflexes as early as the first day. Can we believe that a peripheral neuritis or anatomical changes in the cerebro-spinal axis have occurred at so early a date, so as to cause the paralysis?

3. In its commencement diphtheritic paralysis often exhibits what Trousseau designates mutability. It suddenly shifts from one group of muscles to another. Muscles paralyzed on one day have their normal action on the following day, while other muscles are attacked, and on the third day the group of muscles first attacked are perhaps again paralyzed. This mutability of the paralysis, this sudden shifting from one group of muscles to another, militates strongly against the theory that the cause of the paralysis is a structural change in the nervous system, whether cerebral or peripheral.

It would seem impossible that there should be a sudden recovery from the paralysis and then on the following day a recurrence of it, if it resulted from degenerative changes, either central or peripheral, occurring in the nervous system. These lesions do not undergo such sudden fluctuations, such mutability, as we observe in the paralysis. A persistent cause should produce a persistent and continuous effect.

4. Several, if not all, of the microscopists who discover degenerative changes in the peripheral nerves which supply paralyzed muscles, state that some nerve-fibres have undergone complete or nearly complete degeneration, others partial degeneration, and others still seem to be intact. Would complete paralysis result from such a state of the peripheral nerves? Would, for instance, the velum palati, as we observe it, be motionless like a curtain, not exhibiting the least sensitiveness when pricked by the point of a pin or other instrument, if the sole cause of the paralysis were degenerative changes in the nerves? Would not the nerve-fibres which are still intact be sufficient to produce some motion? May we not in at least some instances regard the paralysis as the cause of the degeneration in the nerves?—for it is a well-known pathological fact that if a muscle be paralyzed, as, for instance, from a central cause, the nerves supplying it usually undergo more or less degenerative change.

5. A clinical fact antagonistic to the theory that lesions in the cerebro-spinal axis cause the paralysis has been alluded to both by Dr. A. Suss and Dr. W. H. Thomson in their interesting and instructive papers. It is that diphtheritic paralysis, motor and sensory, is sometimes limited to the muscles supplied by a single branch of a nerve, while the other branches have their normal function. This fact is, of course, not antagonistic to the theory that peripheral nerve lesions cause the paralysis, but it affords a strong, if not conclusive, argument against the theory that central lesions are the cause.

Such are the clinical facts which militate against the theory that inflammatory or degenerative changes in the nervous system are the primary and sole cause of diphtheritic paralysis. We have stated above that the theory relating to the causation of diphtheria, which is now gaining acceptance in both continents with pathologists and specialists in diseases of children, is that the specific microbe of diphtheria acts locally upon the surface, and systemic infection occurs from ptomaines produced by microbic action, which, entering the lymphatics and blood-vessels, are carried to the interior of the body and exert their action upon the blood and the tissues. If this theory be true, the symptoms which indicate systemic infection are referable to the ptomaines. Dr. Thomson in his paper already alluded to writes as follows: "It is quite conceivable that a ptomaine may follow upon the changes which the diphtheritic process sets up in the organism, and thus produce all its characteristic symptoms. The special tendency of diphtheritic inflammation to cause necrotic and gangrenous lesions lends further support to this surmise."

The ptomaines spring into existence suddenly and unexpectedly under favoring conditions, as we see in the case of the cheese or the milk ptomaine, the tyrotoxicon; and it is not improbable that chemistry brought to the aid of microscopy will yet reveal the fact that the common cause of diphtheritic paralysis is a ptomaine or chemical agent produced by microbic action. If the cause be a ptomaine, it probably acts in a measure like the poison of the eel in the cases alluded to by Trousseau, or like curare. Clinical facts appear to harmonize best with the theory that this is the common cause of the paralysis, especially in those cases in which it occurs early and the use of the paralyzed muscles is soon regained. But it would be idle to argue that the marked degenerative central and peripheral lesions which are frequently present in the nervous system, in those who have died of diphtheritic paralysis, do not prolong and intensify the paralysis, and perhaps are sometimes the primary cause of it.

Prognosis.—The prognosis of diphtheria, like that of scarlet fever, varies greatly in different cases according to its type. In some epidemics a large proportion of the cases are mild and recovery occurs with simple treatment. Between the mild and the most severe cases, attended by profound blood-poisoning, there is every grade of severity. Cases that are apparently mild in the beginning, and seem likely to recover with simple measures, sometimes become severe, dangerous, and even fatal. On the other hand, cases that set in with severity may become modified and end favorably with simple treatment. So variable is the type of diphtheria that in certain epidemics or localities a large proportion recover, as many even as ninety or ninety-five per cent., while in other epidemics or localities the proportion that perish is much larger.

The prognosis is usually favorable when the inflamed surface and pseudo-membrane are of little extent, the fever and swelling moderate, and the neighboring lymphatic glands and underlying connective tissue but little involved. In many such cases, as we have seen from the description given above, the patient remains in good general health, or feels but slightly indisposed. On the other hand, if the inflamed surface be extensive, the pseudo-membrane deep-seated and exhaling an offensive odor, while the adjacent lymphatic glands are markedly swollen, the patient will probably perish. Nasal diphtheria, which is commonly present in severe cases, and which produces an offensive, irritating, and highly infectious discharge, always involves great danger. It is likely to give rise to systemic infection, since the submucous connective tissue of the nostrils contains numerous lymphatics, which take up the poisonous products and convey them to every part of the system. If while the local disease is severe and extensive, the breath and exhalations become offensive, and the countenance and surface generally begin to have a dusky, pallid hue, profound blood-poisoning has occurred, and the patient will probably die.

Physicians of experience are guarded in the expression of a favorable prognosis in diphtheria, since there is no other disease in which the prognostic signs on which a favorable prediction is based are so likely to be fallacious. We hear much in medical circles of the deceptive character of diphtheria. Error in expressing a favorable prognosis, of which even physicians of ample experience complain, is largely due to the fact that diphtheria terminates fatally in several different ways. Death may occur from—

1. Diphtheritic blood-poisoning,—systemic infection by the specific principle, whether acting directly or through the agency of ptomaines which it produces.

2. Septic blood-poisoning, produced by absorption from the under surface of the decomposing pseudo-membrane, or from gangrenous tissues. But our knowledge is not sufficiently advanced to enable us to discriminate between the constitutional effects of ordinary sepsis and those produced by the agency of the diphtheritic poison. Septic infection is obviously most likely to occur in those cases in which the pseudo-membrane is extensive, deeply embedded, and its decomposition attended by an offensive effluvium. Cervical cellulitis and adenitis, which cause considerable swelling of the neck, often occur from septic absorption from the faucial surface, the septic matter being conveyed by the lymphatic vessels to the adjacent glands and causing inflammation of the glands and surrounding connective tissue. Considerable tumefaction of the neck, therefore, seldom occurs in diphtheria without manifest symptoms of toxæmia, and it is to be regarded as a sign of its presence.

3. Diphtheritic croup, or pseudo-membranous laryngo-tracheitis, a most important disease, and fully treated of in the proper place.

4. Uræmia, or diphtheritic nephritis, also one of the most important of the local maladies pertaining to diphtheria, and produced by the action of the diphtheritic poison.

5. Sudden heart-failure. The action of the heart may be feeble from granulo-fatty degeneration of its muscular fibres, or from anæmia or general weakness; but sudden and unexpected death from heart-failure is commonly, as we have seen, due to paralysis of this organ.

6. Suddenly-developed passive congestion and œdema of the lungs, probably due to feebleness of the heart's action, or to paralysis of the respiratory muscles. Death sometimes occurs apparently from this cause during the period of supposed convalescence, and when the visits of the physician have been discontinued. Thus, in a case in my practice, symptoms of œdema pulmonum (abundant moist râles in both sides of the chest, and embarrassed respiration) suddenly occurred nearly one month after the disappearance of the faucial pseudo-membrane and inflammation. The urine, which had contained considerable albumen during the active period of the malady, had for some time shown no trace, or but slight trace, of this principle, by the proper tests. By active stimulation these symptoms entirely disappeared in a few hours, and the heart's action seemed normal, except that it was a little weakened. On the following day the symptoms reappeared, and death occurred before I was able to reach the house.

That physician is obviously least likely to err in prognosis who recognizes the fact that patients are liable to perish in any of these different ways, and carefully examines in reference to all the conditions which involve danger. Many physicians, as I have had the opportunity to observe, are remiss in not examining more frequently the urine of diphtheritic patients; for there is often a large amount of albumen with granular casts in the urine in diphtheria, indicating a poisonous quantity of urea in the blood, and yet the appearance of the urine to the naked eye is probably normal.

Among the symptoms which render the prognosis unfavorable are repugnance to food, vomiting, pallor of countenance, and general anæmia, with progressive weakness and emaciation, indicating blood-poisoning; a large amount of albumen, with casts, in the urine, showing uræmia, to which the irritability of the stomach is often due; an abundant irritating discharge of muco-pus from the nostrils, or occlusion of them by membranous exudation or inflammatory thickening, showing that the Schneiderian membrane is seriously involved; hemorrhage from the nostrils, buccal cavity, or fauces, showing an altered state of the blood, or of the walls of the capillaries, or plugging of the capillaries by masses of microbes or leucocytes. Diphtheritic laryngo-tracheitis, or pseudo-membranous croup, largely increases the aggregate of deaths from diphtheria, whether it be treated by improved inhalations, intubation, or tracheotomy. Some of the above symptoms have been present in most of the fatal cases which I have observed. On the other hand, the prospect of recovery improves in proportion to their absence.

Preventive Treatment.—Diphtheria is so highly contagious, and when epidemic it is so likely to spread from one household to another, and its severe forms are fatal in so large a proportion of cases, that preventive measures are of the greatest importance. The area of contagiousness of diphtheria is small. Dr. Lancry cites cases to show that it is limited to a few feet. Dumez also relates an instance showing that the contagious area is of small extent. In a school the boys and girls in the same hall were separated by an open space a few yards wide. Diphtheria prevailed among the girls, but did not affect the boys. In this respect, as in so many others, diphtheria resembles scarlet fever, and is unlike pertussis and measles.

The most efficient method of preventing diphtheria is the isolation and disinfection of patients, the prompt and thorough disinfection of the apartments in which patients have been treated, and of the bedding and furniture in these apartments, and the exclusion or prevention of all noxious gases, especially those ascending from the sewers and from filthy accumulations of all kinds.

Dr. H. B. Baker, of Lansing, has published statistics showing that in 102 outbreaks of diphtheria the average number of cases where disinfection and isolation, one or both, were neglected was 16, and the average deaths 3.26, while in 116 outbreaks in which isolation and disinfection were enforced, the average number of cases per outbreak was 2.86, and the average deaths .66. Therefore these precautionary measures prevented 13 cases and 2.57 deaths for each outbreak; in the total, 1545 cases and 298 deaths. These statistics related to only one year.[1]

It is obvious that, in order to prevent the spread of diphtheria, wherever a case has occurred prompt and efficient personal and domiciliary disinfection should be practised so far as the condition of the patient will allow. But there is reason to think that disinfection as commonly practised is inadequate. In the winter of 1887–88 and the following spring, an epidemic of diphtheria occurred in the New York Infant Asylum, and it extended to the maternity ward. In this ward five of the new-born infants contracted diphtheria, and two of these five had at the same time umbilical phlegmons in addition to the usual diphtheritic exudate upon the fauces. It was evident from the occurrence of these cases that the maternity ward was infected to such a degree that subsequent patients could not be safely admitted without its thorough disinfection. The ward was therefore vacated, the windows, doors, and crevices closed, and forty pounds of sulphur, or two pounds to the hundred cubic feet of air, were burnt until it was consumed. After some hours the windows and doors were opened, and Drs. Prudden and Cheeseman immediately raised a dust from the floor and bedding and allowed it to settle in culture-media. All other sources of infection were excluded from the media. The cultures produced so large a number of microbes that they overlay each other; but the observers were able to distinguish the streptococcus pyogenes in the media, identical in form and appearance with the streptococcus which they had previously discovered in the umbilical phlegmon. Although more sulphur was employed than is recommended by the New York Health Board, and employed in the manner recommended by this Board, it was inadequate to destroy the microbes. It was evident that some more efficient mode of domiciliary disinfection was required.

Since the ordinary mode of disinfection was apparently futile in the maternity ward, it seemed to me advisable to obtain the views of so eminent an authority as Dr. E. R. Squibb, of Brooklyn; and he has kindly favored me with the following note:

"Within the past ten years the efficacy of sulphur-fumigation against infectious material has been repeatedly denied and reaffirmed upon very good authority, and observations, apparently made with accuracy and care, have been reported from time to time to prove both sides of the question: so that all that can now be said is that burning sulphur is of doubtful efficacy, with the weight of the highest authorities in bacteriology against it. But to this it must be added that it is still largely used by very intelligent bodies in large institutions, boards of health, etc., where it would not be likely long to maintain an unearned confidence.

"How often the fumes are applied dry, and how often moist, no one can tell from the current record; and how many of the failures of the dry gas would be successes in the presence of moisture, there is no means of knowing.

---

[1] American Lancet. (See Ann. Univ. Med. Sci., 1888.)

"Formerly, when sulphur was burned in closed chambers as a disinfectant, the surfaces were all wetted, and the pot of burning sulphur was set in water or wet sand, that the heat might evaporate off a constant supply of watery vapor.

"These conditions are now frequently, if not generally, neglected; and where this is the case, failure, on principle, should be the rule.

"Nearly all, if not all, chemical disinfectants are in a state of tension, ready to change on coming in contact with the matter to which they are applicable; and these changes are either by oxidation or deoxidation, and the moment of greatest power or activity is the moment of change, when they by reacting on infectious matter pass from a state of tension to a state of rest under new relations. The agency through which these changes almost universally become operative is the vapor of water.

"When sulphur is burned in a close chamber, the dioxide is formed by condensing two molecules of oxygen from the air upon each molecule of the sulphur, and a heavy gas is the result, which tends to settle at the bottom of the chamber and to run out through the lower cracks. Any moisture present is at once seized by this rather inactive anhydride, first forming sulphurous acid, and then, by oxidation from the air, sulphuric acid. The dry gas, or anhydride, not only seizes with avidity all watery vapor in the air, but also the water held in the surfaces of all bodies with which it comes in contact, and in the presence of this moisture only is it ready for further oxidation. Then it is by this oxidation that it deoxidizes the matters with which it is in moist contact, filling the surfaces of these matters first with sulphurous acid, then, by the change, with sulphuric acid; and it is during these changes that its power is exerted.

"If there be no moisture supplied to the burning sulphur, that which was present in the air and the surfaces of the chamber is soon used up, and the dry gas remains indefinitely in a comparatively inactive, ineffective condition. The dry passive anhydride would necessarily destroy all organisms which breathed in any degree, because breathing-surfaces are moist. But in embryonic life protected by shell, as in seed, if the shell be dry the gas would be impotent. Many bacteriologists have admitted that burning sulphur would kill bacteria, but not germs."

It seems probable that the apparently negative effect of burning sulphur for the purpose of destroying the microbes in the maternity ward, as stated above, was due to the absence of moisture, for it was burnt dry. The above note from Dr. Squibb conveys very important information. If the facts as stated by him were more generally known and acted on by Health Boards and by physicians in family practice, the results of domiciliary disinfection would probably be better. It is so important that the specific principle of diphtheria should be destroyed wherever this disease appears, in order to prevent its propagation, that any safe measures which will aid in producing this result should be employed in addition to sulphur-fumigation. To accomplish this purpose, Dr. Llewellyn Eliot recommends during the continuance of a case the constant evaporation of turpentine over a water-bath, so that the vapor fills the room. In every instance in which he has employed this treatment, no second case of the disease has occurred.[1] I have employed the following prescription for the purpose of disinfection during my attendance on cases, with apparently so good a result that I am encouraged to continue its use:

℞ Acidi carbolici, ℥i;
Ol. eucalypti, ℥i;
Spts. terebinth., ℥viii.
Misce.

Add two tablespoonfuls to one quart of water in a pan with broad surface, and maintain in a constant state of ebullition or simmering, in the room occupied by the patient. This disinfecting vapor was employed in the quarantine wards of the Infant Asylum, in which diphtheritic patients were treated, and to a certain extent in the other wards, and no subsequent cases have occurred.

In Bellevue Hospital, where pyæmia had been prevalent, Prof. R. Ogden Doremus employed chlorine gas mingled with steam to secure disinfection, in the following manner. Strips of paper having been pasted over the crevices around the doors and windows, equal parts of common salt and black oxide of manganese (about two hundred pounds) were placed in troughs formed of sheet lead, the edges being turned up to make receptacles. A carboy of sulphuric acid was emptied into small basins and other vessels and placed beside the troughs. The floors were moistened with water, and abundant steam was allowed to escape from the heaters into the ward. With the aid of assistants, the sulphuric acid was quickly poured upon the mixture in the troughs, and the room hastily vacated, the door being nailed up to prevent accidental entrance, for the large quantities of chlorine evolved would have been fatal. The following day the windows were opened from without, and, after ventilation, the contents of the troughs were stirred and sulphuric acid added as before. In the ward most infected, this process was repeated once, fresh salt and manganese being used. No further cases of pyæmia occurred in these wards.

In order to prevent as far as possible the spread of diphtheria, stringent measures should be taken to prevent propagation of the disease by walking cases, by children mildly affected who are allowed to attend school and ride in public conveyances. I have in a number of instances seen children with diphtheria sitting with other children in the clinics at Bellevue. Recently I saw in consultation a child with fatal diphtheria, which apparently was contracted in the street by embracing a playmate who had been allowed to go out for the first time after an attack of the disease. In another instance a child went with its parent to a Sunday mission-school in one of the tenement-house sections of New York. Four or five days subsequently it had diphtheria, which was communicated to other children of the family, and one of them died. The philanthropic endeavor to benefit the poor children of New York by conveying them to rural localities in midsummer has, it is said, resulted in the occurrence of diphtheria in farming sections where it was previously unknown. I have now under treatment a family with diphtheria, and the child first attacked states that a school-mate sitting near her in the school complained of sore throat a few days previously. Certainly the safety of the public requires that all children with sore throats should be excluded from the schools whenever diphtheria is prevalent, and it should be the duty of teachers, acting under the direction of Health Boards, to see that this is done.

---

[1] Medical Bulletin.

In a paper relating to the therapeutics of diphtheria, read before the Philadelphia County Medical Society, May 23, 1888, and printed in its Transactions, Dr. A. Jacobi remarks that the well children in a family where diphtheria is occurring should not go to church or school, and that schools should be closed during an epidemic of diphtheria, or, if not closed, that teachers should every morning inspect the throats of the pupils and send home those with sore throats. He recommends also the disinfection of coaches and railroad-cars at regular intervals during an epidemic. He also states that a patient recovering from diphtheria may contract it anew from the curtains, carpets, and furniture which he has infected during his sickness, so as to have a renewal of the disease. He has seen patients die from these renewals, and has seen other patients improve immediately when removed to other apartments. He also states that an irritated surface is more likely to contract diphtheria than one that is healthy, and therefore buccal, faucial, and nasal catarrhs should be promptly treated, the cure of these diminishing the liability to diphtheria. Chronic nasal catarrh, he says, should be treated with two or three daily injections of a solution of salt (1 to 130), to which a one-per-cent. solution of alum may be profitably added, and the same may be gargled in the treatment of faucial catarrh. A nasal spray of nitrate of silver (1 to 500 or 1 to 1000) also hastens the cure. The inflamed buccal surface should be treated by the potassium chlorate or sodium chlorate. Enlarged tonsils, which may harbor the diphtheritic virus, should be reduced by the galvano-cautery, and enlarged cervical glands should also be treated as a preventive measure. Very similar views were expressed in a paper read before the New York Academy of Medicine in January, 1888, by Dr. A. Caillé, who believes that he has prevented the recurrence of diphtheria in those who have suffered repeated attacks of it by prolonged daily antiseptic treatment of their exposed surfaces, which harbored the poison or constituted a nidus favorable for its lodgement and propagation. These somewhat novel views of Drs. Jacobi and Caillé certainly require consideration and experimental testing.

Treatment.—Although diphtheria has been one of the most common of the severe infectious maladies in Europe and America during the last thirty years, physicians are far from agreeing in reference to the proper mode of treatment. The diversity of opinions in regard to the use of therapeutic agents is due in part to a variation in the type of the malady in different epidemics and localities, in part probably to the fact that other forms of inflammation of a severe type have been mistaken for the diphtheritic, but more to the fact that different theories have been held respecting the cause and nature of diphtheria. Hence one physician recommends with confidence a medicine or mode of treatment as eminently successful in his hands, of which another speaks disparagingly.

The germ theory, as described in the foregoing pages, according to which diphtheria is produced by micro-organisms, has had a marked influence upon the mode of treatment. The question has been much discussed, whether diphtheria is primarily a constitutional or a local malady. Acceptance of the germ theory does not require us to believe that diphtheria is primarily local, for the specific microbe might enter and infect the blood through the lungs before any symptom occurred, and, as we have stated elsewhere, the long incubative period of six or seven days in certain cases harmonizes with the theory of a primary blood-disease, rather than with the theory that diphtheria is in the beginning strictly local, its seat being upon one of the exposed surfaces, where the microbe has effected a lodgement. But the latter theory is, as we have seen, more generally accepted, and certain facts lend strong support to it. But if diphtheria be primarily local there can be no doubt that, as in the vaccine disease, the system becomes quickly infected in cases of ordinary severity, so that successful treatment requires the use of both constitutional and local remedies. Acceptance of the germ theory evidently leads to the employment of germicide remedies, the so-called antiseptics or anti-ferments, externally and internally, in order to destroy the specific principle of the disease. Hence, in proportion as this doctrine was accepted, carbolic acid, the chlorine preparations, bromine, the sulphites, salicylic acid, and the most prompt and efficient agent of this class, corrosive sublimate, came into use.

Hygienic Treatment.—The patient should be placed in an airy room, and the evacuations should be promptly disinfected by chlorine, carbolic acid, or other disinfectant, and removed from the room. Purity of the air in the apartment is required; but in the ventilation, draughts of air through the room should be avoided, on account of the liability to diphtheritic croup, which produces about one-third of the deaths from diphtheria. M. Jules Simon recommends that the windows of the apartment be constantly closed, and that ventilation be obtained through the open window of the adjoining apartment. In bathing the patient, care must be taken that he be not chilled. Bathing should be performed expeditiously in a warm room, with perhaps some increase of the stimulants administered. The patient should be constantly in bed, and the temperature of the apartment should be from

70° to 75° F. A uniform temperature of the apartment at about 73° F. is safest.

All physicians of experience recognize the importance of the use of the most nutritious and easily-digested food, and the preservation of the appetite, for diphtheria produces rapid destruction of the red corpuscles and loss of flesh and strength, and it may soon produce a state of dangerous weakness. Beef tea or the expressed juice of meat, milk with farinaceous food, etc., should be administered every two or three hours, or to the full extent without overtaxing digestion. I have sometimes employed the pepsin preparations before each feeding, with apparently good results, as in the following formula:

℞ Pepsini puri, in lamellis, ʒi;
Acidi muriat. dilut., ʒii;
Glycerinæ, ℥i;
Aquæ puræ, ℥iv.
Misce.
Dose, one teaspoonful before each feeding.

In cases of feeble digestion the predigested foods are often very useful, as the beef peptonoids of Reed and Carnrick, the sarco-peptones of the Rudisch Company, and peptonized milk. Failure of the appetite, and refusal to take food, are justly regarded as very unfavorable signs. Trousseau says, "Alimentation occupies the first place in the general treatment; and I have observed that the severer the attack the more imperative is the necessity to sustain the patients with nourishing food. Loss of appetite—that is, disgust for every kind of food—is one of the most alarming prognostic signs. We must try to overcome this loathing of food by every possible means; and to get nourishment taken I sometimes do not hesitate, in the case of children, to threaten punishment. When the patient retains his appetite for food, there is good hope of recovery."[1] Occasionally, when great dysphagia is present, whether from the severity of the pharyngitis or from palatal paralysis, it is necessary to resort to rectal alimentation. The rectum absorbs but does not digest, and it is capable of absorbing peptonized food to such an extent that life may be sustained for an indefinite time without stomach-digestion and solely by rectal alimentation. For the purpose of rectal alimentation I have usually employed peptonized milk containing in solution peptonized beef, as the sarco-peptones of the Rudisch Company. If this is administered through a No. 12 to No. 14 elastic catheter introduced far enough to reach the sigmoid flexure, and retained for half an hour by a compress pressed closely against the anus by the fingers, the result is, I think, better than when we depend, as Trousseau did entirely, on stomach-digestion. One objection to the use of the brush, instead of spraying the fauces with the atomizer, is that it is more likely to cause vomiting, by which nutriment that is so much required is lost. In malignant cases of diphtheria, as in scarlet fever of a similar type, patients are

---

[1] American Lancet.

sometimes allowed to slumber too long without nutriment. It is
slumber of toxæmia, and should be interrupted at stated times in orde
give food and stimulants.

Stimulants.—M. Sanné, in his treatise on diphtheria, says, "De tous
antiseptiques donnés à l'intérieur, l'alcool est de beaucoup le plus sûr. P
l'infection est prononcée, plus il faut insister sur les composés alcooliqu
He states that Bricheteau reports the history of a patient who took d
during diphtheria a bottle and a half of the wine of Bordeaux, without
least symptom of intoxication or headache. A similar case was relate
me, in which nearly one and a half pints of brandy were given in twe
four hours, without any ill effect, and with an apparent good result on
general course of the disease. The same rule holds true in diphtheria a
other acute infectious maladies, that while mild cases do well without a
holic stimulants, they are required in cases of a severe type, and should
administered in large and frequent doses whenever pallor and loss of ap
tite or of strength and flesh indicate danger from the diphtheritic or se
infection. It matters little how the stimulant is administered, whet
milk punch or wine whey, provided that the proper quantity is emplo
If given early and frequently in grave cases, as, for example, one
spoonful every half-hour of brandy or Bourbon whiskey, it does seem
have a tendency to render the disease more tractable; but to be inst
mental in saving life in malignant cases it must be given boldly from
start. If there be marked diphtheritic toxæmia when its use is commen
it will not save life, but it may prolong it. Although the liberal empl
ment of alcohol is apparently useful, it cannot be regarded as a spec
In the quarantine wards of the New York Foundling Asylum in M
1878, were four children, between the ages of three and five years, who
been sick a few days with severe diphtheria, and it was evident at a gla
that they must soon perish with the ordinary mild sustaining remed
Quinine, iron, the most nutritious food, and a moderate amount of alcoh
stimulants were being given, and we determined to increase the Bour
whiskey to a teaspoonful every twenty to thirty minutes day and ni
Nevertheless, whatever the result might have been with the earlier c
mencement of this treatment, the blood-poisoning was now too profou
and one after the other died. That intoxication is almost never produ
in this disease by large and frequent doses of the alcoholic stimulant
probably in part due to its quick elimination from the system, but more
the nature of diphtheria.

In fulfilling the indication of sustaining treatment, the vegetable to
have been long used, especially cinchona and its alkaloid principle quin
The compound tincture of cinchona, and the fluid extract, have been u
and recommended by physicians of experience, but of vegetable age
quinine has been and is still more frequently prescribed than any ot
But the doses employed vary greatly in size and frequency in the prac
of different physicians. It is administered for its antipyretic effect in la

doses, so that twenty or thirty grains are given daily, and in small doses, as one or two grains every fourth hour, for its tonic effect. That there is nothing antagonistic in the action of quinine to the diphtheritic virus, and that it is beneficial in the same way as in the other acute infectious diseases, and no further, is, I think, generally admitted by physicians. Large and frequent doses do not, apparently, produce any controlling action on the course of the disease or diminish the blood-poisoning. Cases might be cited in illustration. In the case of a child of four years with malignant diphtheria, forty-eight grains administered daily had no appreciable effect in staying the fatal progress of the disease.

Quinine in doses of three to five grains has been employed as an antipyretic in diphtheria, as also in the other infectious diseases; but as an antipyretic it is not very efficient, and the temperature after the first two or three days in diphtheria is not often so elevated that an antipyretic is required. As a tonic in doses of one to two grains it is probably to a certain extent beneficial, and it has been highly recommended by good observers for its local action upon the fauces when used by insufflation. The late Prof. Rochester, of Buffalo, recommended and practised in the treatment of diphtheria the insufflation of sulphate of quinine, in powders of two grains, upon the faucial surface, every two hours.[1] It is not improbable that benefit may result from its local action, for used in this manner it is antiseptic. But the employment of this agent by insufflation is very unpleasant to the child, and is likely to be resisted. Given in solution in doses of two grains, as in the following formula, it produces some local action on the fauces, if drinks be withheld subsequently for a few minutes, and at the same time some tonic effect probably results from its use in this manner:

℞ Quiniæ sulphat., ʒss;
Syr. pruni Virginiani
seu
Elix. tarax. comp., ℥ii.
Misce.

Give one teaspoonful every two to four hours to a child of five years. I have often prescribed quinine in this manner, with apparent benefit, in the treatment of diphtheria.

Tinctura Ferri Chloridi.—All physicians who are familiar with diphtheria have noticed the pallor, and loss of appetite, flesh, and strength, which commence before the close of the first week in severe cases, and which are always unfavorable symptoms, indicating, as they do, rapid and progressive deterioration of the blood. The use of iron is at once suggested as the proper medicinal agent to arrest this blood-change, from its known effect in increasing the number of red blood-corpuscles and the amount of coloring matter in these corpuscles. By its effect on the red corpuscles, which are the carriers of oxygen, it increases the functional activity of organs and improves the general nutrition. The ferruginous preparations, therefore, hold an important place in the therapeutics of diphtheria. The one which has stood the test of experience and is now commonly employed is the tincture of the chloride of iron. It should be given in large and frequent doses, as five drops hourly to a child of three years.

---

[1] New York Medical Journal.

Ferguson[1] regards the tincture of the chloride of iron as the most valuable of all the remedies for diphtheria. He examined the blood daily or every second day in twenty cases of diphtheria, and was astonished to observe how rapidly the red blood-corpuscles were reduced in number, those remaining presenting an unhealthy appearance. He believes that the iron partially arrests the blood-change. He administers as much as can be tolerated. To a child of ten years he gives hourly one teaspoonful of the following mixture in water:

℞ Tinct. ferri chloridi, ℥i;
Syr. simplic., ℥iii.
Misce.

If the stomach do not tolerate this dose, half a teaspoonful is administered every half-hour. An infant of seven months, greatly prostrated, took every hour one teaspoonful of the following:

℞ Tinct. ferri chloridi, ʒii;
Syr. simplic., ℥iv.
Misce.

A lady of twenty-two years, greatly prostrated, having an excessive formation of pseudo-membrane and a very fetid breath, took daily one and a half fluidounces of the iron for ten days.

M. Jules Simon says,[2] "For internal treatment, from three to six drops of the tincture of the chloride of iron should be given in a little water every two or three hours; but it should not be given with milk or gum-water, or from a metallic spoon, on account of the decomposition which occurs, which may produce digestive troubles." Dr. Whittier believes that this medicine, given so as to saturate the system, is the best that can be employed. In thirty-six consecutive cases in which the fauces were covered with the exudate, all recovered under the use of the iron as the principal medicine.[3] Dr. S. Baruch, of New York, prescribes hourly doses of this remedy in quantities varying from eight to twenty-five drops mixed with glycerin and water. Food and stimulants are administered before the iron, but not immediately afterwards, so that the iron may have a local action upon the faucial surface.[4] Dr. Billington recommends hourly teaspoonful doses of the following mixture:

℞ Tinct. ferri chloridi, fʒi;
Glycerinæ,
Aquæ, āā ℥i.
Misce.

---

[1] Canadian Practitioner.

[2] Le Progrès Médical.

[3] Boston Medical and Surgical Journal.

[4] New York Medical Record.

Prof. Joseph E. Winters says that he has given two drachms of the tincture of the chloride of iron every half-hour for forty-eight hours, with manifest benefit, to a child of eight years.[1] But it is only in the most severe or malignant form of the disease, the form described by Sanné as septic phlegmonous, that such large doses are proper or are required. In mild cases from three to five drops given hourly or oftener suffice. This is the dose recommended by Jules Simon, of Paris.

Several recent writers make the plausible statement that the indication of treatment by the iron is to saturate the system as soon as possible, employing for this purpose as large and frequent doses as can be tolerated by the stomach. The tolerance of a drug depends largely on the manner in which it is employed. The best vehicle for the tincture of the chloride of iron is glycerin and water. It may be conveniently prescribed with two or three times its quantity of glycerin, and a certain number of drops administered in water. The advice of Simon should be borne in mind, not to give it with gum-water, nor with milk, nor from a metallic spoon.

That now after nearly half a century of the constant use of iron in diphtheria in both hemispheres there is an almost unanimous verdict in its favor, renders it probable that the few who have not observed its good effects have treated unusually bad cases or have given the medicine in small and inadequate doses. We shall see that the opinions of physicians have not remained equally favorable in regard to the use of the agent with which the iron has been commonly combined, the potassium chlorate.

Potassium Chlorate.—This agent produces a curative effect on buccal inflammations, and its beneficial action when employed for the various forms of stomatitis has led to its extensive use in pharyngitis. When taken internally, it is eliminated in part by the salivary glands, so that it continues to exert in part a local action on the surface of the mouth and fauces until it is entirely eliminated. This medicine, the potassium chlorate, has of late years become also a domestic remedy; but the laity should be cautioned in reference to its use. It is an irritant to the kidneys in large doses, producing intense inflammatory congestion of these organs and arresting their function. The melancholy fate of Dr. Fountaine, of Davenport, Iowa, in 1861, whose life was sacrificed by an experimental dose of potassium chlorate, is remembered by the older physicians. Fountaine took half an ounce in a gobletful of warm water at eight A.M. Free diuresis occurred, which ceased at four P.M. Though fatigued and pallid, he ate a hearty supper. During the following night he was in collapse, with vomiting and purging, and severe abdominal pain. Early in the following morning he voided two ounces of dark urine, after which no urinary secretion occurred. The choleraic symptoms returned, with collapse, but he again rallied. He had vomiting and intense and constant abdominal pain during the subsequent six days, when death occurred. The total cessation of fæcal and urinary evacuations for six days was a notable fact. At the autopsy the lesions of an intense and general gastro-intestinal inflammation were present, the mucous membrane hanging in shreds and patches; the bladder was empty, and its mucous membrane presented a similar appearance to that of the stomach and intestines. The condition of the kidneys is not stated, except that there was liquid resembling urine under the capsule of one kidney, and crystals of the chlorate were in the pelves of the kidneys. A few years since, in my practice, a child of three years with active diphtheritic pharyngitis was allowed to quench its thirst by drinking water from a small pitcher in which three drachms of potassium chlorate had been dissolved, and which had been ordered as a gargle. In the morning I was summoned in haste, and found the surface of the patient cold and blue, and pulse feeble. The urine was totally suppressed, and instead of it a few drops of blood passed from the urethra. Death occurred before night.

---

[1] Diphtheria and its Management, 1885. (See Ann. Univ. Med. Sci., 1888.)

Jules Simon[1] says that potassium chlorate, acting wonderfully well in diseases of the mouth, produces no beneficial effect in diseases of the fauces, and it weakens the little patient when given in large doses. Dr. J. P. Esch says that he has observed that the potassium chlorate used internally in diphtheria almost invariably produces symptoms of nephritis. Ferguson[2] totally condemns its use in any dose or mode of administration in diphtheria. In every case in which he employed it, if albuminuria were present it increased the amount of albumen. Von Focke[3] believes that any benefit which may be derived from the potassium chlorate in diphtheria results from the oxygen in it. To render the oxygen more efficient, he adds hydrochloric acid. He prepares a two-per-cent. solution of the chlorate, with a one-and-a-half-per-cent. solution of the acid, and administers a half-teaspoonful to two teaspoonfuls, according to the age, every one to two hours. All the benefit obtained from this mixture may be derived from a prescription long used and favorably known in New York, and probably more frequently written than any other prescription for diphtheria. The tincture of iron in the mixture contains one minim of free muriatic acid in each drachm, but a small amount of this acid is added to the mixture in addition. The prescription, with some variations in its proportions in the practice of different physicians, is as follows:

℞ Tinct. ferri chloridi, ʒ ii–iii;
Potas. chlorat., ʒ i;
Acidi muriat. dilut., gtt. x;
Syr. simplic., ℥iv.
Misce.
Dose, one teaspoonful hourly or each second hour.

After such an extensive use of potassium chlorate during nearly half a century, its therapeutic uses should be clearly defined, and any ill effects which may result fully determined. From what is now known of its action, it would probably be better to abandon its use in diphtheria, since it is a remedy of doubtful efficacy for throat-affections. If it be employed, it should certainly be administered in small doses sufficiently diluted. If it be prescribed, it should not, I think, be in larger quantity than half a drachm in twenty-four hours for a child of five years.

---

[1] Le Progrès Médical.

[2] Canadian Practitioner.

[3] Wien. Med. Wochenschr.

The remedies mentioned above are those which have been most largely employed for internal medication by physicians of the present and the preceding generation; but the belief that diphtheria has a microbic origin, that the action of the microbes gives rise to poisonous ptomaines, and that the virulence of the disease is due to these organisms and chemical products, has during the last few years brought into prominence the germicide and antiseptic treatment. The attempt is now made, and apparently with considerable success, to cure the patient by antagonizing and destroying the cause of diphtheria. We look with interest and for enlightenment to the results of treatment by the antiseptics, and compare them with the results obtained by the use of tonics, stimulants, and alimentation, which have been heretofore employed.

Among the most useful of the statistics bearing upon the action of germicide and antiseptic remedies in the treatment of diphtheria are the following, made by N. Lunin in the hospital of Oldenburg in 1882.[1] In this hospital two hundred and ninety-six children had diphtheria, and one hundred and sixty-four, or 55 per cent., died. The treatment by corrosive sublimate consisted in brushing the pharynx every two hours with a solution of 1 part to 1000, or in spraying by the irrigator of Rauchfuss with a solution of 1 part to 5000. The patients subjected to this treatment numbered fifty-seven. Forty-three of them had the fibrinous form of the disease, and fourteen the septic phlegmonous form. Thirteen of each class died, or 45 per cent. of the whole number. The tincture of the chloride of iron Lunin employed in small doses, only one drop every quarter-hour, or two drops every half-hour, in ninety-four cases, forty-three having the fibrinous form and fifty-one the septic phlegmonous form. The total mortality was 56.3 per cent. Irrigation of the fauces was also employed in these cases with a three-per-cent. solution of boric acid. Lunin made use of chinoline in twenty-eight cases,—nineteen of the fibrinous form and nine of the septic phlegmonous form. Fifteen died, or 53 per cent. This agent was prescribed in a five-per-cent. solution, the medium being half water and half alcohol. Twenty-nine children were treated by resorcin, a solution of ten per cent. being applied by the brush twice hourly, and irrigation with a one-per-cent. solution once hourly. Sixty-five per cent. died. A solution of bromine and bromide of potassium was applied from one to three times hourly to the fauces in thirty-three patients, but 69.7 per cent. died.

Finally, twenty-three infants were treated by turpentine, a tablespoonful twice daily, and in some of the cases an additional hourly dose during two or three days. The mortality was 43.4 per cent. In the fibrinous form the percentage of deaths from the different modes of treatment was as follows:

---

[1] Archiv für Kinderheilk., 1886.

	PERCENTAGE.		PERCENTAGE.
By turpentine	8.30	By chinoline	31.60
" resorcin	20.00	" tinct. ferri chloridi	32.60
" corrosive sublimate	30.20	" bromine	46.70

In the septic form the deaths were as follows:

	PERCENTAGE.		PERCENTAGE.
By tinct. ferri chloridi	76.5	By resorcin	89.5
" turpentine	81.0	" corrosive sublimate	92.9
" bromine	88.9	" chinoline	100.0

Therefore, according to Lunin's statistics, turpentine was the most useful agent in the fibrinous form of diphtheria, and the tincture of the chloride of iron in the septic phlegmonous form.

Hydrargyri Chloridum Corrosivum (*Hydrargyri Perchloridum*, Br. Phar.).—The use of this agent in the treatment of diphtheria is based on the theory of the microbic origin of this disease. Corrosive sublimate is the most active and certain of the germicide agents employed in medicine, whether used locally or internally. It quickly destroys all micro-organisms with which it comes in contact, and in safe medicinal doses it is believed to penetrate all parts of the system. The employment of corrosive sublimate in the treatment of diphtheria is not new, since it appears that the late Dr. Tappan, of Steubenville, Ohio, prescribed it with apparent benefit in 1860–61; but it was seldom prescribed as a remedy in this disease until within the last four or five years. The establishment of the theory of the microbic origin of diphtheria, and a knowledge of the fact that the sublimate is the most efficient germicide, have made it the favorite remedy with many physicians. Of course its employment demands caution, and is justified only by the fact that the disease for which it is prescribed has hitherto been very fatal with other modes of treatment. Though this agent is now widely used for diphtheria, medical journals thus far contain very few reports of its supposed toxic or injurious action, while many physicians believe that it diminishes the virulence of diphtheria and increases the percentage of recoveries.

In ordinary cases the following may perhaps be regarded as about the proper quantities, which should be administered in divided doses, in twenty-four hours. For a child of two years, gr. $\frac{1}{6}$ (gr. $\frac{1}{72}$ every two hours); for a child of four years, gr. $\frac{1}{4}$ (gr. $\frac{1}{48}$ every two hours); for a child of six years, gr. $\frac{1}{3}$ (gr. $\frac{1}{36}$ every two hours); and for a child of ten years, gr. $\frac{1}{2}$ (gr. $\frac{1}{24}$ every two hours). Thus, if we employ the vehicle which Dr. Tappan used one-quarter of a century ago, the following prescription might be written for a child of six years:

℞ Hydrarg. chlor. corros., gr. i;
Alcoholi, ʒii;
Elix. bismuthi et pepsinii, q. s. ad ℥iv.
Misce.
Dose, one teaspoonful every two hours.

According to the statement of physicians, considerably larger doses have been administered with safety and apparent benefit, and in severe cases, attended by profound blood-poisoning, such as Lunin designates septic phlegmonous, certainly the maximum medicinal dose is required, if we depend on the sublimate as the main remedy. Dr. Grant (Bey) administered to a child of four years one-half grain every half-hour till six doses were taken, and then hourly during the first day, every second hour on the second day, and on subsequent days at longer intervals. Dr. A. Jacobi states that an infant a year old may take one-half grain every day for many days in succession, with very little if any intestinal disorder, and with no stomatitis. Although certain children may tolerate doses so large as those recommended by Dr. Grant (Bey), safer doses are those which we have recommended above, and they seem to be sufficient for protracted use. Dr. P. Werner[1] recommends in the treatment of diphtheria the sublimate dissolved in distilled water, in half-hourly doses or at a little longer interval, so that the following quantities are taken in twenty-four hours. For an infant of one and one-half years, 0.015 (grain 0.231) of the sublimate in 120.0 (4 fl. oz.) of water; for a child at the age of six to seven years, 0.3 in 180 (grain 0.45 to 6 oz.) of water. This quantity is to be given in divided doses in the twenty-four hours. At night, if the child sleep, the doses should be less frequent and proportionately larger than in the daytime. Dr. I. N. Love, of St. Louis, states that he has employed the sublimate in doses of one one-hundredth to one-fiftieth grain every hour or second hour, according to the age, preceded by large draughts of water. Its action as thus used seemed to be both local and constitutional.

Those who denounce the use of mercurials in diphtheria, like Jules Simon and one at least of our distinguished American writers, grouping together calomel, the oleate, the unguentum, the cyanide, the biniodide, and corrosive sublimate, condemning them in a body, on the ground that they enfeeble the system, do injustice to the therapeutic virtues of the sublimate. Medicines having the same base often differ widely in their action upon the system; and it is the common and probably correct belief that the sublimate in safe medicinal doses does not enfeeble the system, but in some instances acts rather as a tonic.

In my practice excellent results have apparently occurred from the local use of corrosive sublimate,—its use by the atomizer. If the sublimate be administered internally at the same time, care must be taken not to employ too much. The solution which I have employed with the atomizer consists of two grains of the sublimate to one pint of water, and in spraying the fauces the bulb of the atomizer is compressed from three to five times. In ordinary cases the spray is used every second hour. Oatman, of Nyack, New York, has lost but one patient in twenty-three by the following local treatment. Cotton is firmly wound around the end of a stick about the size of a lead-pencil, being drawn out as it is wound, and made to project beyond the end. This is dipped into a solution of the bichloride of mercury, two grains to the pint (1 to 3840), and passed into the throat until it touches the posterior wall of the pharynx. It is then instantly withdrawn and burnt. This treatment is repeated hourly with a new swab each time, until the inflammation begins to subside, which is usually in forty-eight hours. Jules Stümf[1] treated thirty-one cases, with two deaths, by inhalation of the sublimate, using the apparatus of Richardson. For infants under the age of two years he employs 1 part to 4000; from five to six years, 1 part to 2000; for those over six years, 1 part to 1000. Dr. Thomas Welcher recommends in the treatment of diphtheria,[2] used as a gargle or employed as a spray, a solution of corrosive sublimate of 1 to 1000. In most instances, when this local treatment had been employed a few times at intervals of one to two hours, the pharyngeal disease began to abate, and the general condition improved. Dr. Welcher also employs small doses of the sublimate internally. It is evident from the experience of other physicians that when this agent is used as a spray in so strong a solution as 1 to 1000, it should be used with caution. Two or three compressions of the bulb will be sufficient. Prof. A. Jacobi recommends for washing the nares a solution of corrosive sublimate 1 part to 2000 to 1 part to 10,000, with or without 10 to 50 parts of table-salt or 60 to 300 parts of boracic acid.

---

[1] St. Petersburg. Med. Wochenschr., 1886.

The medical journals during the last three or four years contain abundant testimony to the beneficial results of both the internal and the local use of corrosive sublimate in diphtheria. An important question evidently arises, to wit, how to use this active agent internally and locally at the same time without administering too large a quantity. Some physicians administer the amount that can safely be employed in twenty-four hours, dissolved in water and in frequent doses (every hour or second hour), and if no drinks be given subsequently for a few minutes the local effect upon the fauces is to a considerable extent obtained. Perhaps this is the safest and best mode of employing this very efficient and useful antiseptic agent in the treatment of diphtheria.

Calomel.—Dr. Simon Baruch begins the treatment of all cases of diphtheria not attended by diarrhœa by a dose of four to eight grains of calomel, followed, if necessary, by a laxative.[3] He cites the experience of Dr. Coester, who administered, in the preliminary treatment of diphtheria, calomel in sixty-nine cases and lost only one. Prof. Simon, of Paris, in the treatment of diphtheria discards (1) blisters, which are always followed by the reproduction of pseudo-membrane, (2) bleeding and mercurials, which enfeeble the patient, (3) preparations of opium, which produce rapid depression of the vital powers, and (4) potassium chlorate in large doses. The reference of Simon to mercurials is probably more particularly to calomel.[1]

---

[1] Münch. Med. Wochenschr.

[2] Deutsche Med. Zeit.

[3] New York Medical Record.

On the other hand, Dr. Geo. B. Fowler considers calomel the best remedy with which to combat diphtheria. When croupy symptoms supervene, he increases the dose from gr. $\frac{1}{6}$ to gr. $\frac{1}{3}$ or even one grain every hour.[2] Dr. I. N. Love remarks that the most marked recent recommendation of the use of calomel in diphtheria is from the pen of Dr. Wm. H. Daly, chairman of the Laryngological Section of the Ninth International Medical Congress.[3] Dr. Daly's method is to administer the calomel two to five grains every one, two, or three hours until free catharsis follows, and then at longer intervals, but so that three or four daily evacuations are produced. The editor of the *Therapeutic Gazette* writes, "We have so frequently seen an apparently severe attack of diphtheria abruptly aborted in its inception under the influence of large doses of calomel, that we can scarcely believe that the drug has no pronounced effect. A grain of it should be put dry in the mouth of the child every hour or two, until frequent, very loose, liquid evacuations are produced."

In addition to those already mentioned, other physicians of ample experience have recommended calomel in the treatment of diphtheria, some in laxative doses and only at the beginning of the attack, and others in doses of the fractional part of a grain every two to four hours during the sickness. The majority of physicians, very properly in my opinion, discourage the employment of calomel in laxative doses during the sickness, believing that it tends to weaken the patient and increase the anæmia, which in all cases of severe diphtheria soon becomes very manifest, whatever the treatment; but a single laxative dose is perhaps sometimes useful. It may do good, as in other infectious diseases, to unload the *primæ viæ* in the commencement of the attack, so that the remedies to be employed are more readily absorbed and without alteration by admixture with chemical products in the intestinal tract. What change calomel undergoes so that it can be absorbed has not been clearly ascertained.

Turpentine.—This has been highly recommended recently by physicians of experience, when used locally as well as internally, for its prompt action in arresting the formation and extension of the pseudo-membrane, and as an antidote to the diphtheritic virus. Dr. Rewentauer states that an infant of two years treated by other remedies began to have symptoms indicating invasion of the larynx on the fourth day. Tracheotomy was resolved upon, but previous trial was made of pure turpentine in a teaspoonful dose. The croupiness ceased, other symptoms improved, and the patient recovered without tracheotomy.[1]

---

[1] Jour. de Méd. de Paris.

[2] New York Medical Record.

[3] Weekly Medical Review.

Delthil, and following him, Schenker employed a mixture of coal-tar and turpentine, which was burnt in the room occupied by the patient either constantly or several times through the day. Schenker's observations led him to believe that the benefit from this treatment occurred chiefly from the turpentine, and largely from its general effect on the system. He therefore decided to employ turpentine internally, in doses of ten minims to one teaspoonful, one to three times daily, in milk, sugar-water, or gruel. At the same time he employed it as a spray. Alcoholic stimulation, cleanliness, and a diet of beef tea, milk, and egg were enjoined. Of thirty-six cases which Dr. Schenker treated by turpentine, thirty-one recovered.

Röse, of Hamburg, employed turpentine in teaspoonful doses mixed with spirit of ether (ether one part, alcohol three parts) three times daily. A teaspoonful of a two-per-cent. solution of salicylate of sodium was also given every two hours. Under this treatment the temperature and pulse diminished, other symptoms improved, and in fifty-eight cases thus treated, 95 per cent. recovered.[2] Sigel also employed turpentine in teaspoonful doses in forty-seven cases, in fourteen of which the question of tracheotomy arose. A manifest reduction of temperature followed the use of the turpentine. The percentage of deaths in all thus treated was 14.9, while of those treated by corrosive sublimate, salicylic acid, potassium chlorate, etc., 32.5 per cent. died. Dr. Llewellyn Eliot also reports good results from the vaporization of turpentine.

The recent recommendation of turpentine in the treatment of diphtheria by many physicians of large experience and sound judgment, among whom we may mention Drs. S. Baruch and A. Jacobi, has extended and established the use of this agent. Its supposed efficacy depends on the fact that it is antiseptic and germicidal, and that when vaporized and inhaled, or taken by the stomach, it penetrates all parts of the system. The descriptions long given in the text-books of the physiological action of turpentine have had the tendency to induce physicians to employ it in small doses. But I am not aware that any writer has recorded ill effects from the use of turpentine in diphtheria, although it has been employed by a considerable number of physicians in the last year or two, and in quantities which exceed the medicinal doses mentioned in text-books.

It is well known that the constitutional effects of the oleum terebinthinæ, even to impaired vision, strangury, and bloody urine, may be obtained by the prolonged inhalation of its vapor;[3] and I have employed the vapor of the oil of turpentine during the last two or three years with such apparent good results that I confidently recommend this mode of using the turpentine.

---

[1] Centralbl. f. Klin. Med.

[2] Therap. Monatschr.

[1] Stillé and Maisch.

The accompanying prescription is mixed with water in the proportion of two tablespoonfuls to one quart of water. ℞ Acidi carbolici, ol. eucalypti, āā ℥i; spts. terebinth., ℥viii. This is placed in a shallow vessel or vessels with a broad surface, and maintained in a constant ebullition or simmering, upon a gas or other stove. The vapor, which is not unpleasant, soon fills the room, and even adjoining rooms. As regards the effect on the patient, the turpentine vapor passing over the inflamed surfaces, which are the seat of the exudate, with every inspiration probably produces more or less local disinfection, apart from the systemic disinfection which it may cause by entering the blood and the tissues generally. Thus employed, the turpentine is also apparently a useful domiciliary disinfectant, affording protection in a measure to other members of the family, and to the physician and nurses, as we have stated elsewhere. The oil of turpentine will probably in the future be a very important remedy in the treatment of diphtheria, whether taken by the stomach or received as a spray.

Pilocarpine.—Certain physicians have recommended pilocarpine in the treatment of diphtheria, because it is supposed that the salivary and mucous secretions which it produces aid in throwing off the pseudo-membrane. Dr. Lax states that ten patients treated by him, some of them severely sick, all recovered.[1] He employed the following prescription:

℞ Pilocarpini hydrochlorat., gr. $\frac{1}{2}$ to $\frac{3}{5}$;
Acidi hydrochlorici, gtt. ii–iii;
Pepsini, gr. x–xii;
Aquæ destillat., ʒ xviiss.
Misce.
Dose, a teaspoonful or tablespoonful in wine.

Guttmann treated in a year and a half eighty-one cases by this remedy without a death. Gelsner and Delewsky also report good results. On the other hand, I have seen the most disastrous effects from the use of pilocarpine in diphtheria, the secretions filling the bronchial tubes and being expectorated insufficiently and with great difficulty. Death resulted. The symptoms which occurred were like those in extreme œdema of the lungs. I cannot, therefore, recommend its use. Its employment appears too hazardous, especially in young and feeble children.

Sodium Benzoate.—Dr. I. N. Love recommends the sodium benzoate in five, ten, or fifteen grain doses.[2] He remarks that Salkowski in 1879 noticed that this drug largely increased the secretion by the kidneys of nitrogenous and sulphurous compounds, and concluded that it would aid in depurating the blood of noxious matters. Salkowski, Fleck, and Buckholtz ascertained that the benzoate arrested the growth of micro-organisms in putrid liquid, and Graham Brown that diphtheritic liquids became non-contagious by the addition of the benzoate. Helferich, Graham Brown, and Sanné, from experiments made on animals, consider the benzoate of sodium a specific against the virus of diphtheria.[1] On the other hand, M. Dumas, surgeon to the Hôpital de Cette, has not derived any marked benefit from its use, and Prof. A. Jacobi says that it does not deserve the eulogies bestowed upon it from theoretical reasonings.[2]

---

[1] Medical News.

[2] Weekly Medical Review.

Such are the more important remedies, used internally, which have been up to the present time employed in the treatment of diphtheria. The number, it is seen, is large, and most of them are no doubt useful in certain cases. Diphtheria, being a disease of variable type, must be treated according to the indications in each case. The internal remedies which in my opinion have been most useful and which should be most frequently employed are the tincture of the chloride of iron, quinine, corrosive sublimate, turpentine, and the alcoholic preparations.

Among the other remedies which have been recommended by good observers, we may mention the following. Copaiba and cubebs are employed and recommended by distinguished French physicians. Jules Simon prescribes copaiba and cubebs for patients over the age of five or six years.[3] Dr. I. H. Fruitnight has employed the sodium hyposulphite in eight cases, giving hourly drachm doses of the following: ℞ Sodii hyposulph., ʒi; aquæ, f℥ii. The result was favorable. Illingworth[4] recommends the biniodide of mercury. Dr. C. B. Galentine recommends the internal use of hydrate of chloral, given with the potassium chlorate to a child of six years in about two-and-a-half-grain doses. Herbert L. Snow recommends sulphurous acid, Dr. Hofmokl the hydrogen dioxide, and E. S. Smith the oil of eucalyptus and Warburg's tincture. In diphtheria, therefore, as in other diseases which in a large proportion of cases end favorably, whatever the treatment, the number of recommended remedies is large.

Local Treatment.—*Solvents.*—The belief is becoming prevalent in the profession that the early destruction and removal of the exudate from the faucial or nasal surface is not an imperative duty, as was formerly practised under the teachings of Bretonneau and Trousseau, provided that thorough disinfection of the pseudo-membrane and the surrounding and underlying tissues be effected. Patients are injured by irritating lotions or instrumental treatment designed to remove the pseudo-membrane, which immediately reappears in greater extent and thickness than at first, on account of the increase in the inflammation in consequence of the severe measures employed. The employment at short intervals of mild but efficient antiseptic applications in place of the stronger and irritating lotions formerly used has been a great improvement in the treatment of diphtheria. But antiseptic lotions, vapors, or sprays are inadequate to produce complete disinfection, if the pseudo-membrane have great thickness. Its under surface, which is in immediate relation with the lymphatics and blood-vessels, and from which systemic poisoning occurs, from absorption of the diphtheritic germ, septic matter, or ptomaines, is probably not reached by the antiseptic sprays or lotions as commonly employed. Any painless and unirritating application which diminishes the thickness of the pseudo-membrane by its solvent action, or, better, entirely dissolves and removes it, is therefore useful. Of the unirritating solvents, alkalies, pepsin, trypsin, and papayotin have been chiefly used, and have in the highest degree the confidence of the profession. The efficiency of solvent treatment depends largely on the manner in which it is employed, the kind of instrument used, and the frequency of the application. The solvent agent heretofore most largely used has been lime-water or the spray of slacking lime. Its solvent action is probably due chiefly to its alkalinity, but its alkalinity and its solvent action can be greatly increased by adding to it the sodium bicarbonate. From observing its effects in a considerable number of cases, the writer recommends with confidence the following formula:

---

[1] La France Médicale.

[2] New York Medical Record.

[3] Le Progrès Médical.

[4] British Medical Journal.

℞ Ol. eucalypti, ʒ ii;
Sodii benzoat., ʒ i;
Sodii bicarbonat., ʒ ii;
Glycerinæ, ℥ ii;
Aquæ calcis, Oi.
Misce.

To be used with the hand-atomizer from three to five minutes every half-hour, or with the steam atomizer almost constantly. This alkaline spray not only exerts a solvent action on the pseudo-membrane, but also renders the muco-pus thinner, less viscid, and therefore so changes its character by diminishing its viscidity that it is more easily expectorated.

The use of *pepsin* as a solvent is suggested, from its well-known action in digesting nitrogenous substance. It has been employed with varying results. It is well known that some of the preparations in the shops are much more active than others, and hence perhaps a chief reason for the difference in the results claimed. It is well to remind the reader that it should be employed alone or with an acid, for it is comparatively inert if used with an alkali. It may not be improper to state that, in comparative tests of the pepsins in the shops made by Dr. George B. Fowler and related before the Pediatric Section of the Academy of Medicine, the solvent action of the pepsin prepared by Parke, Davis & Co., of Detroit, was especially noticeable.

Rossbach states[1] that he has used a solution of *papayotin*, or vegetable pepsin, frequently applied to the fauces. In young children a few minims may be placed on the tongue every five minutes. If the drug be good, he states that the membrane is dissolved in two or three hours. Prof. Jacobi says that this agent is readily dissolved in twenty parts of water.[2] It may, he says, be brushed over the surface or used as a spray. Mixed with water and glycerin in greater concentration (1 to 4–8), it has been used by him with fair results. Dr. J. K. Bauduy, Jr., also writes favorably of the solvent action of papayotin on the pseudo-membrane.[1]

---

[1] St. Petersburg. Med. Wochenschr., 1886.

[2] New York Medical Record.

Trypsin, unlike pepsin, is an active solvent in an alkaline medium, and it may be added to the alkaline mixture described above. Dr. F. C. Fernald relates the case of a boy of six and a half years, who had perforation of each membrana tympani and began to complain of sore throat. A pseudo-membrane appeared upon the tonsillar portion of the fauces, and the right auditory canal was covered with a diphtheritic exudate, entirely occluding it, so that liquids did not flow from the external ear to the fauces as formerly. The ear was filled every half-hour with the following mixture: ℞ Tripsin., gr. xxx, sodii bicarb., gr. x, aquæ destillat., ℥i. The pseudo-membrane gradually dissolved and disappeared, the passage through the ear and Eustachian tube became open, and the patient recovered.[2] Dr. E. N. Liell also relates a case in which trypsin apparently produced a solvent action on the pseudo-membrane. Probably, therefore, in the present state of our knowledge we can apply no better solvent mixture upon the diphtheritic pseudo-membrane than trypsin added to the alkaline solution described above.

Albuminuria.—This being due to septic nephritis, patients have seemed to me to be more benefited by the tincture of the chloride of iron, in frequent and rather large doses, than by any other remedy. If while this is being used a marked diminution in the quantity of urine occurs, it may be necessary to employ diuretics and laxatives, as in scarlatinous nephritis. The potassium bitartrate or acetate, and perhaps the more laxative salines, may be needed under such circumstances. But marked diminution of urine, and especially anuria, in diphtheria, end fatally, with few exceptions, according to my observations, whatever the treatment.

Paralysis.—The loss of the tendon reflexes, and palatal and multiple paralyses, require the same stimulating and sustaining remedies which are appropriate for the primary disease diphtheria. Iron and other tonics, nutritious and easily-digested diet, massage, and, in some instances, electricity, suffice to restore the use of the affected muscles; but sometimes weeks and even months elapse before their use is fully restored. So long as the paralysis does not affect any vital organ, a favorable prognosis may be expressed, although recovery may be slow.

On the other hand, it is evident from its nature, and from the cases which have been related, that cardiac paralysis is exceedingly dangerous, and must be treated promptly and by the most active remedies. As we have seen, the attack of cardiac paralysis is usually sudden, with little forewarning, and is often fatal before the physician, promptly summoned, is able to arrive. The patient should be as quiet as possible in bed, with the head low, and alcoholic stimulants should be administered at once. In the sudden seizures such as have been related above, hypodermic injections of brandy act most promptly in sustaining the heart-action. Ammonia, camphor, musk, and the electrical current may be useful auxiliaries. The predigested beef preparations, peptonized milk, and other concentrated foods designed for those with feeble digestion, are useful. If the urgent symptoms are relieved by these measures, such remedies should be employed as are useful in other forms of diphtheritic paralysis. The patient is ordinarily feeble, anæmic, and with poor digestion. The beef extracts and concentrated foods should be continued. Iron, quinine in moderate doses, and alcoholic stimulants are indicated. The use of the electric current is suggested by the nature of the attack. Many physicians believe that they have obtained benefit from its use in the treatment of the more common forms of diphtheritic paralysis, while others speak doubtfully of its efficacy. If there be reason from the symptoms to suspect the presence of central lesions in the nervous system, the galvanic current in short sittings has been recommended, and not the faradic. In ordinary cases either the direct or the induced current may be employed.

---

[1] Medical Weekly Review.

[2] Medical News.

Strychnine is, however, regarded by good observers as the most efficacious nerve-stimulant in the various forms of diphtheritic paralysis. Oertel's objection expressed twenty years ago to the use of strychnine in this disease, that, acting as an excitant of the spinal cord, it is likely to aggravate central lesions, was founded on a wrong understanding of the pathology of the paralysis. Prof. Henoch cured diphtheritic paralysis in three weeks by hypodermic injections of strychnine. W. Reinard[1] states that a boy three and a half years of age fifteen days after the appearance of the diphtheritic patches on the tonsils had paralysis of the inferior extremities and the velum palati, a tottering gait, nasal voice, and difficult deglutition. At the end of twelve days death seemed imminent, the paresis of the lower extremities had become a complete paraplegia, and the paralysis of the upper extremities, and of the muscles of the nucha, larynx, and thorax, was complete. He was unable to sustain himself in the sitting posture, his head falling heavily on his chest. He had also dyspnœa, hoarse cough, tracheal râles, and aphonia, probably from cardio-pulmonary paralysis. Reinard made a hypodermic injection each day of one milligramme (about one-sixty-fifth of a grain) of sulphate of strychnine, in the nucha. Improvement occurred in twenty-four hours in the tonicity of the muscles. On the third day the cardiac and pulmonary paralysis had so improved that the tracheal râle had ceased. The respiration was more normal, and deglutition possible. On the fifteenth day of this treatment, and after fifteen injections, the patient was considered cured. Dr. Gerasimow[2] relates the case of a child six years of age who had paralysis of the velum, pharynx, larynx, and lower extremities. Six weeks after the commencement of paralytic symptoms, subcutaneous injections of strychnine, two centigrammes (or about one-thirty-first of a grain), were given daily. With this treatment the patient improved, and after seven injections of this strength, followed by twelve of one-twenty-second of a grain, the cure was complete.

---

[1] Deutsche Med. Wochenschr., 1885, No. 19.

[2] Med. Obosr., No. 20.

With such strong testimony in favor of the use of strychnine, it is perhaps remarkable that physicians of experience state that they have not observed any marked benefit from its use in the treatment of diphtheritic paralysis. At a meeting of the New York Clinical Society, held December 23, 1887,[1] Dr. Holt stated that he was yet to be convinced that strychnine possessed any specific value in this disease, though it was of much value as a general tonic. At the same meeting Dr. A. A. Smith stated his belief that tonics and time did more for diphtheritic paralysis than anything else. He had used electricity and strychnine, and had never been able to satisfy himself that electricity did any good, and the effects of strychnine seemed to be not specific, but those of a general tonic. On the other hand, Dr. Thatcher, of New York, has reported a case in which galvanism was employed on the two paralyzed upper extremities alternately, on each for a week at a time. It was invariably found that the arm receiving the electricity gained more rapidly than the one untreated, the strength being tested by the dynamometer. This test seems to have been conclusive as showing the efficacy of galvanization.

NOTE.—In the preparation of this article I have availed myself of extracts from former publications of my own, whenever my present views coincided with those already presented.

---

[1] New York Medical Journal, Jan. 14, 1888.

MEASLES.

By F. E. WAXHAM, M.D.

Synonymes.—Rubeola, Morbilli.

Definition.—Measles is an acute epidemic, contagious disease, characterized by a peculiar papular eruption, occurring usually on the fourth day of the attack, preceded by catarrhal symptoms and followed by slight desquamation.

History.—This disease was described with small-pox by Rhazes, A.D. 900, who undoubtedly recognized the difference between them. Before that date there is no authentic account of the disease.

It continued to be confused with scarlatina and small-pox until 1670–74, when Sydenham and Morton declared the former to be a distinct disease.

While the origin of measles is buried in obscurity, at present it is disseminated nearly all over the world, seemingly following the footsteps of civilization. Only in those distant countries where civilization has not penetrated is the disease unknown.

Etiology.—Measles is due to a specific poison that has not yet been isolated. It is both epidemic and contagious. All authorities agree that it cannot originate *de novo.*

That it is epidemic is manifest from the fact that the disease is far more common during certain seasons or years than others. A community may be comparatively free from the disease for a time, when at length it will sweep over it like a cyclone and but few will escape. A period of immunity will then prevail, lasting for a longer or shorter time, when it will again make its appearance.

That it is highly contagious needs no argument to prove. It indeed ranks with small-pox in this regard. The contagiousness begins with the catarrhal symptoms and continues until after desquamation. The contagious principle exists in the breath, the exhalations from the skin, the blood, the tears, the nasal and bronchial secretions, and undoubtedly in the urine and faecal discharges. The poison of the disease gains access to the system in the great majority of cases through the mucous membrane of the respiratory tract, the inspired air carrying the active contagious principle. The disease is equally prevalent in both sexes. Although a disease of childhood, the adult is noway exempt from it. The great majority of children, having suffered from the disease in early life, escape it later, but those who do not receive such immunity are susceptible.

The infant under six months is generally exempt from the disease, although there are many notable exceptions.

In speaking of the etiology of measles, it must be mentioned that micrococci have been discovered in the breath and in the blood of patients suffering from the disease; but it is not yet clear whether these micrococci are *post hoc* or *propter hoc*.

Pathological Anatomy.—The morbid anatomy varies considerably, and is influenced in a great measure by the complications which are so frequently the cause of death. The blood is sometimes bluish or brownish red, sometimes thin and deficient in coagulability, and sometimes thick. The mucous membranes usually present evidences of catarrhal inflammation, and should death result from some complication, which is usually the case when the termination is fatal, we will observe the lesions characteristic of such complications.

Symptomatology.—The disease may be divided into four stages: first, the stage of incubation; second, the stage of invasion; third, the stage of eruption; and fourth, the stage of decline.

While some claim that after infection the poison of the disease lies dormant in the system for a certain length of time before manifesting its presence, the majority of authorities believe that the disease commences, that is to say, the active contagious principle exerts its influence, the moment it enters the system, and when it has attained sufficient force to upset the equilibrium of the system we get the characteristic symptoms.

The period between exposure and the commencement of the manifest symptoms constitutes the first stage, or that of incubation. This period varies from seven to twenty-one days, with an average of about twelve.

Stage of Invasion.—This stage may be ushered in abruptly by vomiting, chills, fever, and pain in the head, back, and limbs, accompanied by symptoms of a catarrhal nature. Usually, however, the onset is gradual, and loss of appetite, malaise, and mild catarrhal symptoms, with slight fever, are first observed.

The catarrhal symptoms rapidly develop, and soon become the most prominent feature of this stage. The mucous membranes of the eyes, nose, throat, larynx, trachea, and bronchial tubes become implicated. The conjunctivæ become reddened and congested, and increased lachrymation is observed, the eyes being suffused with tears. The mucous membrane of the nasal passages becomes reddened and swollen with at first a thin watery discharge, which soon becomes abundant and muco-purulent in character. As a result of the irritation in the nasal cavities, frequent sneezing occurs and is a common symptom.

The inflammation of the mucous membrane of the larynx and bronchial tubes gives rise to a frequent troublesome cough. Sometimes the cough becomes decidedly croupy and the respiration embarrassed from the swelling of the mucous membrane of the larynx. Occasionally alarming symptoms result from œdema of the glottis. At times the inflammation of the pharynx assumes a diphtheritic type, which may extend into the larynx. This dangerous complication, however, is not frequently observed. Nausea and vomiting are often present, and indicate disturbance of the digestive system. Diarrhœa occurs in a small proportion of cases, and would seem to indicate irritation of the mucous membrane of the intestinal tract. Convulsions rarely occur, but when they are present they are of very serious import.

Epistaxis occasionally results, but seldom becomes alarming.

The fever that may have preceded the catarrhal symptoms increases in intensity with the development of these symptoms, and the temperature usually ranges from 102° to 104° F.

The symptoms that have been enumerated as characteristic of the stage of invasion vary greatly in intensity. In some cases they are so mild that the child is supposed to be suffering only from a slight cold, while in other cases they are so severe as to make the patient quite ill, and occasionally they assume a dangerous character.

Stage of Eruption.—About the fourth day the catarrhal symptoms have reached their height, and upon this day in the great majority of cases the eruption first begins to make its appearance. The eruption first appears upon the forehead, temples, and cheeks, soon extending to the face, breast, extremities, and trunk. At first the eruption appears in the form of minute red spots; these rapidly increase in number and size, and become distinctly papular and perceptible to the touch. These papules, of a dark-red color, are in many cases surrounded by areas of skin of normal color, but on certain portions of the body, especially the face, neck, and forearms, they become confluent, and these portions present a peculiar blotched and swollen appearance. By the end of the sixth day of the disease, or the second from the first appearance of the eruption, the disease is at its height. The eruption is now fully developed, and has extended to all parts of the body, although more marked in some portions than in others. The catarrhal symptoms and the fever which were present during the stage of invasion continue unabated, and diarrhœa, if not present before, frequently occurs at this stage. The tongue continues moist throughout the attack, and covered with a light coating. Enlargements of the submaxillary and anterior cervical glands are common.

The disease, having reached its height by the end of the sixth day, remains stationary, all the symptoms persisting, but not increasing in severity, for two days, when the eruption rapidly fades, the fever diminishes, and the catarrhal symptoms abate. We now reach the last stage of the disease.

Stage of Decline.—This stage is characterized by the diminution of all the symptoms. Although the eruption rapidly fades and the fever subsides, yet a bronchitis remains for some days and is the last symptom to disappear. As the active symptoms disappear, the appetite and natural disposition of the child return, and the patient is soon in ordinary health.

The stage of decline terminates with a fine, furfuraceous desquamation, which begins about the tenth or eleventh day. It is most marked when the eruption has been the most intense.

Atypical Course.—The regular course of measles may be interrupted, or the disease may present variations from the typical course just described. We may meet occasionally with those cases styled *morbilli sine catarrho*, in which the catarrhal symptoms are wanting or very slight, and where the eruption occurs with scarcely any premonitory symptoms. Again, cases are occasionally met with styled *morbilli sine exanthemate*, where the catarrhal symptoms are well pronounced, but where the eruption is very scant or entirely absent. Other cases, again, are met with where the eruption is long-continued, and still others where the eruption is of very light color, in strong contrast to others, where the eruption is of a deep dark red and confluent, at times livid, with evidences of extravasation.

The disease, again, is sometimes modified or suddenly interrupted by intercurrent affections.

The so-called **black** or **malignant hemorrhagic measles** presents a series of symptoms somewhat dissimilar from those enumerated. Evidences of great depression occur early in the attack. The pulse becomes extremely rapid and feeble, the temperature high, the extremities cold, the patient anxious and restless or somnolent, with a tendency to convulsions or coma. The eruption may scarcely make its appearance, and death may occur before it is developed, or it may occur, usually in the confluent form, and the papules assume a dark livid or even black color, as they fade leaving dark-yellowish stains. This type of measles is extremely fatal, and occurs most frequently in broken-down subjects, those suffering from some constitutional dyscrasia, or those whose hygienic surroundings are unfavorable.

Diagnosis.—During the stage of invasion it is difficult to distinguish measles from a severe attack of coryza or bronchial catarrh.

Known exposure to the disease would be the strongest evidence of its real character. After the appearance of the eruption we may confuse the disease with rubella, or German measles, scarlet fever, variola, varicella, or typhus fever.

In rubella we have catarrhal symptoms, but they are slight. In rubella the eruption appears within twelve or twenty-four hours after invasion, and very frequently the premonitory symptoms are so slight that the patient is not considered sick until the eruption is discovered. The eruption, instead of being a deep dark red as in measles, is of a lighter color, and the papules are much finer, and we do not see the swollen and blotched surface that is so characteristic of measles. The temperature does not run so high, the pulse is less rapid, and the disease runs a much shorter and milder course.

The differential diagnosis between measles and scarlet fever is based upon the shorter period of invasion in scarlet fever, the presence of sore throat, the absence of catarrhal symptoms, and the difference in the appearance of the eruption. In measles the period of invasion is four days, in scarlet fever two. In scarlet fever the initial symptoms are sore throat and nausea or vomiting. In measles these symptoms are absent, and in their place we have the catarrhal symptoms. In scarlet fever the eruption occurs in the form of minute points of a brick-red color, which coalesce, forming a more or less uniform erythematous redness quite different from the papular eruption of measles.

Measles undoubtedly has been more frequently confused with variola than with any other disease. We often have catarrhal symptoms in variola, but they are not usually so marked as in measles. During the first twenty-four hours of variola the eruption often resembles very closely that of measles; but if there is any uncertainty a delay of a few hours will usually make the diagnosis clear. A very important consideration in the differential diagnosis is the fact that in variola with the appearance of the eruption all the active symptoms abate; the pain in the back, the headache, the high fever, all disappear; but not so in measles. In variola the eruption soon becomes more markedly papular, presenting the *shotty* feeling when the hand is passed over the surface. In the course of the disease these papules become vesicles, and then pustules. In measles, however, the eruption remains papular throughout its whole course, and these papules are but slightly elevated above the surface.

There should be little or no difficulty in distinguishing measles from varicella. In the latter disease the absence of catarrhal symptoms and the rapid development of the eruption into vesicles should be sufficient to render the diagnosis easy.

In typhus fever we meet with a petechial eruption somewhat resembling that of measles, but it does not become confluent, as is frequently the case in the latter disease. The eruption does not appear until the seventh day, while in measles it occurs on the fourth, and there is almost an entire absence of the catarrhal symptoms that are so characteristic of measles.

Complications.—The most common complications of measles are inflammations of the mucous membranes. These inflammations exist during the natural course of the disease to a greater or less extent, and are not properly complications unless so intensified as to give rise to grave or dangerous symptoms. Thus, conjunctivitis is present and constitutes a prominent symptom, but when so intensified as to become purulent, or when involving the cornea in purulent inflammation, it may be properly considered a complication.

Stomatitis is a common complication, varying greatly in severity. It may range from a simple inflammation to ulceration, or even to cancrum oris or noma. Gangrenous inflammation of the mouth, however, more frequently follows as a sequel than as a complication.

Diphtheritic inflammation of the fauces not infrequently occurs, and becomes a source of great danger, especially when it invades the larynx. There is a difference of opinion as to whether this complication is diphtheria engrafted upon the pharyngitis of measles, or whether the membrane which is observed is due directly to the intensity of the inflammation. Inflammation or œdema of the larynx independent of membrane-formation occasionally constitutes a grave complication, that calls for surgical treatment.

Bronchitis, when so severe as greatly to increase the rapidity of respiration, elevating the temperature, increasing the rapidity of the pulse, and giving rise to fine bronchial râles, adds greatly to the danger, and constitutes a true complication.

Pneumonia is one of the most common as well as one of the most fatal complications of measles. Catarrhal pneumonia will be indicated by the expiratory moan, rapid respiration, short painful cough, elevation of temperature, increased rapidity of pulse, crepitant râles, and *slight* dulness on percussion. Lobar pneumonia will present much the same series of symptoms, with the exception that the dulness on percussion is much more marked. The dyspnœa, however, is not so great as in the catarrhal form.

Enteritis and colitis not infrequently occur as complications, while deafness, otalgia, or suppurative inflammation of the middle ear often results from the extension of the inflammation from the pharynx through the Eustachian tube to the drum of the ear.

Prognosis.—The prognosis will depend very greatly upon the previous state of health of the patient, the surroundings, and the care and attention the patient will receive. In private practice, where patients will receive good and careful attention and where the surroundings are healthful, but little danger may be anticipated.

It must be remembered, however, that there are exceptions to this rule. The writer well remembers a notable example. A young man attending college, and in the very prime of early manhood, was taken with malignant measles, and, notwithstanding that the surroundings were perfect and that the attention and care were all that wealth could secure, a fatal termination followed in a few days.

Measles occurring in crowded tenement-houses, where the most careless nursing is usually given, in camps, where patients are exposed to inclement weather, or in crowded hospitals, is a disease to be justly dreaded. Under such circumstances the prognosis must be very guarded, for it is there that fatal complications are most liable to occur.

In some epidemics the death-rate is much higher than in others, and the tendency to fatal complications much greater. The prognosis is favorable in those cases that pursue an even and regular course, but in all cases of great severity, bordering upon malignancy, or that pursue an irregular course, or that develop complications, it should be most guarded. The development of diphtheritic pharyngitis adds greatly to the danger, and the prognosis is generally unfavorable. The development of membranous laryngitis involves still greater danger, although a few will survive after surgical measures. The occurrence of capillary bronchitis, or of catarrhal or lobar pneumonia, also involves the case in great danger. Entero-colitis and dysentery add greatly to the distress and danger, but these complications can generally be overcome. The occurrence of convulsions during the premonitory stage or at the onset of the eruptive stage, while increasing the danger, does not necessarily indicate a fatal termination; occurring, however, later in the disease, almost invariably a fatal termination follows.

The continuance of high fever after the disappearance of the eruption is generally an unfavorable indication, denoting, as it does, the presence of some complication.

Treatment.—The treatment of measles should be preventive, hygienic, and therapeutic.

Preventive treatment consists in the prompt isolation of the patient on the first occurrence of the catarrhal symptoms, thorough disinfection of the apartment and of all clothing, and the use of antiseptics applied to the body of the patient in the form of ointments.

As the disease is not generally recognized during the catarrhal stage, isolation is not usually effective, as measles is highly contagious during this early period. In case, however, of known exposure, the patient should be isolated on the appearance of the earliest symptoms, either by sending the well children from home or by removing the patient to a distant room in the house. The attendant should not mingle with the other members of the household without first changing the dress and washing the face, hands, and head with some antiseptic solution. All discharges should be at once disinfected, and soiled clothing placed in some antiseptic solution and then thoroughly boiled. During the illness antiseptic solutions may be applied directly to the body of the patient two or three times daily, thus preventing the diffusion of the poison. For this purpose carbolized oil, and cold cream or vaseline with carbolic acid, may be employed. After recovery the patient should receive a warm bath before mingling with the other members of the family.

The room should be thoroughly fumigated by burning sulphur moistened with alcohol, and all playthings used by the patient should be burned.

Inoculation as a preventive measure has been employed, but the disease is not sufficiently modified by this means to justify the operation.

The *hygienic* treatment is of great importance in measles. As the disease cannot be aborted or abridged by any known treatment, we must endeavor by careful nursing and by hygienic measures to prevent complications and conduct the case to a successful issue. The patient should be placed in a large, well-ventilated apartment, which should be shaded from bright light, but *not completely darkened*, and the temperature should be uniform. The covering should be light and comfortable, and an abundance of water given when the patient is thirsty. It is a mistake, too frequently made, to bundle a child in heavy blankets and give nothing but hot drinks.

When the eruption is tardy in making its appearance, a warm bath and an occasional drink of hot lemonade may be useful; but to cover a patient almost to suffocation and to give nothing but hot drinks adds greatly to the discomfort of the child and accomplishes little good. Little food is required, especially during the first few days, and that which is given should be simple and easily digested. The patient should be placed in bed at the onset of the catarrhal symptoms, and should remain there until the entire disappearance of the eruption. If this rule were always enforced, we should far less frequently meet with dangerous complications.

Therapeutic Treatment.—Little medication is required in an ordinary attack of measles. There is no specific or known remedy that will cut short the disease. We must be content with warding off complications or meeting them promptly when they occur. In case the cough is troublesome, an occasional Dover's powder may be given, or a soothing expectorant mixture. In case of great restlessness, an occasional dose of bromide of potassium is indicated. In the early eruptive stage, if nausea and vomiting occur, a sinapism should be placed over the pit of the stomach, and equal parts of lime-water and milk, with the white of one egg to each cupful, should be given in small quantities and at frequent intervals. In case of constipation, an injection is preferable to a cathartic; for we must remember the tendency to intestinal irritation.

Personally, in treating measles the writer employs a soothing expectorant mixture, alternating with moderate doses of quinine. The former quiets the frequent harassing cough and gives comfort to the patient, while the latter acts as a tonic, supporting the system against the disease and assisting in controlling the febrile action. As indications arise for special treatment, they are properly met. In case the temperature runs above 103° F. it is controlled by antipyrin, ten grains in a teaspoonful of warm water, injected per rectum and repeated every hour until it is reduced.

In case there is a tendency to malignancy, characterized by the peculiar appearance of the eruption and by rapid and feeble pulse, whiskey is given in large and frequently-repeated doses. Keating has very clearly pointed out the fact that in malignant measles micrococci are found in the blood in great abundance. In culture-solutions it is well known that alcohol is one of the most active destroyers of these micrococci; and the benefit derived from the administration of this agent in malignant measles would indicate that it has the same effect upon these micrococci in the blood. When the patient remains pale and anæmic after the attack, arsenic and iron are especially indicated. When complications arise, they should be promptly met and treated the same as if occurring independently of the disease under consideration.

Owing to the bronchitis, and the involvement of the gastro-intestinal mucous membrane, which accompany this disease, the greatest care should be taken during convalescence that the patient be not exposed to sudden changes of temperature or to draughts. The patient should be sponged off daily with cool water and thoroughly dried, so that the functional activity of the skin shall be maintained. It should be insisted upon that flannel be worn, however light it may be in weight. If it irritates the skin, a fine linen garment may be worn beneath. Especially should the chest, abdomen, and feet be protected against cold. Just as in scarlet fever the greatest precaution must be taken during convalescence to prevent renal congestion, so in measles a pulmonary disease may subsequently terminate fatally a case neglected during convalescence in one who is constitutionally weak.

RUBELLA.

(RÖTHELN.)

By WILLIAM A. EDWARDS, M.D.

Definition.—Rubella is a specific, epidemic, and contagious eruptive fever, occurring independently of the existence of either measles or scarlatina, and possessing characteristic symptoms in its incubation, invasion, eruption, and period of duration. Furthermore, it will reproduce itself only in those exposed to its contagion. One attack usually protects from subsequent invasion, but will not afford immunity from either measles or scarlatina. Children are most susceptible. An almost constant manifestation of the disease—indeed, it may be considered a prodromal symptom—is enlargement and induration of the cervical, submaxillary, auricular, and sub-occipital glands. At times other glands are affected; but suppuration never occurs.

Synonymes.—The disease has been most unfortunate in the number of synonymes and various titles that have been applied to it,—a fact that has much retarded its proper study and classification and has also caused a vast amount of confusion in its recognition. The Germans use the terms "rubeola" and "rötheln" interchangeably, and the French "rubéole," whereas many English and American writers have adopted the term "rubella" advanced by Veale,[1] accepted by Squire, and further receiving the sanction of Dunglison's Dictionary. A recent writer (Griffith) objects to the term rötheln, because it is usually mispronounced and for its proper pronunciation we have no equivalent vowel-sound in English; advocating the title rubella as being a diminutive of rubeola and expressing at once the usually slight import of the disease and its relationship to measles, akin to that existing between varicella and variola.

As a further illustration of the multiplicity of terms, we cite: German measles; French measles; false, bastard, or hybrid measles; rubeola sine catarrho; rubeola epidemica, morbillosa, scarlatinosa, notha; roseola; roseola epidemica; rosalia; rosalia idiopathica; hybrid scarlatina; and many more, which space forbids us to mention.

History.—Not until about the middle of the eighteenth century[2] did rubella receive separate recognition and description as a distinct eruptive fever, and from that time almost until the present day medical opinion has been somewhat divided upon the effect of its distinctive character or of its being a combination of measles and scarlatina. Indeed, as late as 1865 Kostlein[1] held rubella to be a variety of measles. Within the last two decades, however, a consensus of opinion has been secured, and at the present time it is a rarity to observe a writer advocating the hybrid nature of the disease. Strümpel, indeed, goes so far as to say that the existence of the disease can be denied only by those who have never seen it. In the latter part of the eighteenth century papers were published by German writers (Selle, Ziegler, Stark, and others) giving accounts of epidemics more or less severe in character and maintaining the specific nature of the disease: indeed, Formey[2] states that between the years 1784 and 1796 eleven hundred and eighty persons died of it in Berlin, while during the same interval there were but two hundred and three deaths from scarlatina and one hundred and three from measles. Thomas,[3] however, gives these figures very differently, stating that according to Formey there died between 1784 and 1794 in Berlin four hundred and fifty-seven from rubeola, one hundred and seventy-two from scarlet fever, and fifty-three from measles. Opportunity has not been afforded me of consulting the original, and I am unable to decide which is correct. In 1840, Patterson[4] wrote advocating the distinct character of the disease; and his views for a time received support, particularly from Balfour[5] and Tripe.[6] Many, however, did not believe at all in its separate existence. Göden[2] and Jahn[2] were of this class, the former confounding it with scarlatina, and the latter denying its existence; Heim[2] regarded it as an anomalous scarlatina, more dangerous than that disease itself, and he was supported in this view by Reil, Hufeland, and Frank. Other writers declared that it was measles, and attributed its peculiarity to a "certain individuality:" these are the writers who describe a "rubeola morbillosa et scarlatinosa," to give this view a terse expression. Hildebrand and Schönlein discussed an hermaphrodite form of measles and scarlatina, maintaining that in rubella we had a disease which was a hybrid of the other two fevers. This "hybrid" disease for a time took fast hold upon professional opinion, and was advocated by Geertsema, Busche, Paasch, Gelmo, and many others, as stated by Griffith.[7] Copland and Aitken both agreed that it was a hybrid, Gintrac, Hebra, and others also sharing in this view. Indeed, as late as the present decade a writer has denied the specificity of the disease. But, on the other hand, the overwhelming mass of testimony is in favor of its distinct character, as we before stated: indeed, not to recognize it as a separate and distinct disease is almost a confession of not having witnessed a series of cases.[1]

---

[1] Edin. Med. Jour., Nov. 1866.

[2] Hoffmann, De Bergen, and Orlow, who published their papers between 1740 and 1758.

---

[1] Wiener Med. Presse, 1868, xiii. (Atkinson).

[2] Quoted by Griffith, Med. Rec., July, 1887.

[3] Ziemssen's Cyc., vol. ii., Amer. edit., p. 131.

[4] W. A. Edwards, Amer. Jour. Med. Sci., Oct. 1884.

[5] Edin. Med. Jour., 1857, p. 718.

[6] Medical Times, 1852, v. 457.

[7] Ibid.

Let us review for a moment the grounds upon which we, in common with most writers for the last twenty years,[2] decide that rubella is an independent disease. First, rubella is a disease *sui generis,* and is in no way related to either measles or scarlatina; it is not a combination of these, nor is it a hybrid; it has never given rise to any disease but itself in those exposed to its contagion.

Epidemics of rubella prevail without any regard to the existence of cases or epidemics of either measles or scarlatina. An attack of rubella will not protect from either of these diseases; the converse of this proposition is also well established by clinical experience. One attack probably protects from a second. In my own experience of over two hundred cases I have never observed a second attack in the same individual, excluding, of course, relapses, which we will consider later.

To conclude, its symptomatology, invasion, eruptive course, and duration differ much from those of either of the other forms which it is said to resemble. There is no dearth of clinical material to show that rubella does attack those who have had either measles or scarlatina or both: many of my cases had had these diseases; some, in fact, were just recovering from them when prostrated by rubella. The literature presents many similar instances, to which I have before referred. Since then recent writers have further strengthened the statements by the citation of additional examples: for instance, Griffith states that quite a number of the cases reported in his article had previously had measles or developed it afterwards; of Hatfield's one hundred and ten cases many had had measles and scarlatina; Atkinson observes the same condition; most of the one hundred cases reported by Park had had measles; Riggs's cases had previously suffered from measles or scarlatina; out of sixty-three cases reported by Clement Dukes, thirty-nine had had measles, one had rubella, and measles three weeks afterwards, another measles twenty-two days later; and of Shuttleworth's twenty-seven cases, fifteen had had measles and scarlatina, and five had both diseases in later years.

A most interesting case, as bearing on the point at issue, to which I have already referred, is that reported by Tompkins, in which a girl was attacked by rubella, lasting five days; three days after recovery she was attacked by scarlatina, as she had been sent to the hospital as a scarlatinous case and there exposed to it.

---

[1] The independent nature of this disease has been and is now accepted by the following writers: Alibert, Arnold, Atkinson, Barthez and Rilliet, Balfour, Behrend, Bourneville and Bricon, Brown, Cheadle, Collin, Cotting, Cuomo, Damaschino, Davis, Delastre, De Man, Dukes, Earle, Emminghaus, Edwards, Faber, Fleischmann, Gerhardt, Green, Griffith, Grove, Hatfield, Hardaway, Hennig, Homans, Kingsley, Küster, Liveing, Lindwurm, Longuet, Lubanski, Maton, Mettenheimer, Murchison, McLeod, Nymann, Oesterreich, Park, Patterson, Roger, Roth, Salzmann, Schwarz, Smith, Strümpel, Steiner, Shuttleworth, Squire, Thomas, Thierfelder, Tonge-Smith, Trastour, Tripe, Trousseau, Veale, Vogel, Wagner, Wilson.

[2] The following recent writers deny its specificity: Fagge, Goodhart, Henoch, Stewart, Oxley. Descroizilles does not acknowledge its existence at all.

Etiology.—There is but little doubt that rubella is directly *contagious;* it is also more prone to be epidemic than its congeners, and its contagiousness seems to depend greatly upon the exposure and amount of the contagium absorbed, be it what it may, which appears to come off in the cutaneous exhalations and the breath and to be conveyed by fomites, clothing, etc. Many of my earlier cases—indeed, quite seventy-five per cent.—could be distinctly traced to infected ships, particularly the "bunks" of steerage-passengers, an environment which would also present heat and moisture, potent factors in its production. For example, in the island of Malta, after the rainy season the disease prevails to its greatest extent. My own experience leads me to conclude that rubella is one of the most contagious of all the eruptive fevers, more particularly when occurring in large institutions, as in my first one hundred cases in the Philadelphia Hospital. Still, it must be remembered that these cases were particularly virulent, showing a mortality of four and a quarter per cent., and occurred among children who had just passed through the experience of an immigrant aboard the large ocean-steamers, an experience which is certainly not conducive to a very high state of health in young children who left the vessels to become inmates of a large city hospital. Some writers, however, doubt the fact of its contagiousness. Atkinson believes that it is not violently contagious,—far less so than measles. Steiner goes further, and denies its contagiousness altogether. Nymann and Klaatsch consider it very feebly contagious, and Picot, Mettenheimer, Arnold, De Man, and Lindwurm think that it is probably contagious. Thomas, Liveing, Tonge-Smith, Bourneville, and others consider it less contagious than measles, whereas Jacobi, Dukes, and Squire consider it very contagious, the latter stating that this contagiousness is marked even before the appearance of the rash, and persists for two or three weeks after its disappearance. Griffith concludes from his own experience that the contagious nature of rubella is very decided: for example, out of about one hundred children in a "home" which he attended, thirty-seven took the disease in spite of the most prompt and careful isolation; out of approximately the same number in another institution, twenty-six were attacked. In Hatfield's experience, one hundred and ten of the one hundred and ninety-six inmates of the asylum suffered from the disease.

Let us for a moment consider the question of its *infectiousness.* Many of my first cases could be directly traced to infection from the bunks in ships. Emminghaus considers this proved; Thomas, Veale, and others are of a similar opinion.

The disease is more prone to be *epidemic* than either measles or scarlatina. Liveing and Thomas are of a similar opinion; and J. F. Meigs, in a personal communication to Griffith, considers it more apt to be epidemic than contagious. My own experience leads me to agree entirely with Squire in that the disease is contagious throughout its entire course, from the period of incubation, during and well into the stage of convalescence.

As stated in an earlier paper (1884), the specific cause of the disease had not been isolated, nor has any advance in this particular been made since then. An examination of the blood in my Philadelphia Hospital cases (one hundred in number) proved the presence of micrococci in the blood, liquor sanguinis, and white corpuscles, although, as then stated, I was unable to trace any direct relation between these bodies and the disease under consideration.[1] Opportunity has not offered itself for me to pursue the subject further, and, so far as I know, other observers have not taken up the investigation, either to verify or to disprove, with the exception of Hatfield, who considers the virulence of the epidemic to be due to the ravages of the rubella-microbe. Steeves makes the somewhat remarkable statement that under certain constitutional conditions the contagion of scarlet fever may be the exciting factor in producing such a poison in the system as shall determine the specific disease in question, thus showing that even in the present decade a writer will occasionally assert the now wholly-disproved hybrid nature of the disease.

Age.—Rubella is pre-eminently a disease of childhood, most cases occurring at or before the fifth year. Hatfield considers that the liability increases in inverse proportion to the age; Sholl has seen it transmitted from the pregnant mother (seven months) to the unborn child and developed a few days after the birth of the child; many of Smith's and Hardaway's cases were infants. Roth and Steiner record a case in a babe of six months. On the other hand, Griffith considers early infancy as almost exempt. The majority of cases occur between five and fifteen years of age, but adults are often attacked. I have witnessed several severe and prostrating attacks in adults from twenty to thirty years of age. That more adults are not attacked is caused by the fact that they are not so much exposed, as the described outbreaks are usually in children's asylums, and not in general hospitals. I do not recognize the fact that adults possess any special immunity from the disease, but rather the fact that they are not often exposed, or that they are protected by a previous attack. This, however, is not Griffith's experience, as there were many adults connected with the institutions for children in which one hundred of his cases occurred, yet but one of the number was attacked. Kassowitz noted but five adults among sixty-four cases in private practice. As illustrating the extremes of age attacked, Seitz reports a case in a woman aged seventy-three.

Sex.—Sex is not an etiological factor worthy of consideration. Of three hundred and thirty-one cases, one hundred and fifty-one were males and one hundred and eighty females (Hatfield). In my own experience more adult females were attacked than males; but a moment's reflection will explain this, since they were more exposed to the contagion than the male members of the family.

---

[1] A similar though more marked appearance was seen in the blood in cases of malignant measles, in which we were able to show a definite relation between the cocci and the disease. —Keating, Trans. Coll. Phys. Phila., June 7, 1882.

Stage of Incubation.—This is, of course, most difficult to decide positively, as symptoms are almost entirely absent during this time. Squire, however, states that epistaxis and enlargement of the post-cervical glands may be noted, also that the throat may be a little sore,—symptoms that we should be now inclined to class among those of invasion rather than of incubation.

As nearly as I have been able to ascertain, the duration of this stage is about ten days,—certainly between ten and twelve: the shortest period recorded was six days, and the longest twenty-one. The duration of this period, however, must be very variable, as almost every observer has allotted a different time: for example, Griffith, five to eleven days; Musser, six days; Atkinson, fourteen to twenty-one days; Hatfield, ten days; James Robinson, six to seven days; Glaister, four to five days or longer; Duckworth, sixteen days; James Pollock, six to sixteen days; L. A. Clausen, seventeen to twenty days; J. L. Smith, seven to twenty-one days; Earle, seventeen to twenty-one days; Bourneville and Bricon, eight to ten days; Jacobi, fourteen to twenty-one days; Squire, eight to twenty-one days,—generally two weeks; Sholl, five to twenty-one days; Cheadle, eleven to twelve days; Dukes, twelve to twenty-two days,—average, fifteen to sixteen days; Steiner, ten to fourteen days; Cuomo, seventeen days,—never less. Many observers place it at "two weeks or longer;" several at "two to three weeks;" some at two and one-half to three weeks; and one observer (Cotting) at three weeks. Hardaway remarks that, "taken as a whole, it is probably longer than is observed in measles."

Griffith considers this varying period of incubation to be of diagnostic value, thus differentiating from the fixed period of measles.

Stage of Invasion.—In most cases this stage is apt to be, at least in its incipiency, without any very characteristic symptoms. Indeed, excluding hospital cases, and referring to those in private practice, I may say that it is almost without any symptoms until a few hours before the eruption appears. More extended experience has convinced me that epidemics of rubella occurring in asylums, nurseries, hospitals, and the like differ in many essential characters from those that we see in private practice and among the better-housed and better-fed class of children. For example, my Philadelphia Hospital cases were of the most severe type, presenting many symptoms during the period of invasion, whereas those outside of the hospital were comparatively exempt from symptoms at this time.

My notes record the following symptoms: chilliness, languor, faintness, headache more or less severe, pain in the back and limbs, coryza, red and watery eyes, sore throat, cough, and occasionally a hoarse, husky voice. As illustrating the more severe character of some of the first one hundred cases, we note a rise of temperature during this period. Many of the patients did not show a higher registration than 100° F., others varied from this point to 103° F.; nausea and vomiting, delirium and convulsions, and epistaxis in three cases. Other observers have noted marked prodromal symptoms during the invasion: two cases of hemorrhage from the eyes and ears have been recorded by Prioleau; convulsions by Smith and others; delirium by Hardaway and Cuomo; urticaria by Cullingworth in four cases; rigors by Nymann and dizziness by Squire; Mettenheimer notes fainting, and Balfour a croupy condition; Earle, Kingsley, and Thierfelder report a prodromal rash, and Cuomo and myself an erythema preceding the specific rash.

In a former paper I have considered the average duration of the invasion stage as three days. Again in this particular do we find a great difference of opinion, some, as Griffith, McLeod, Murchison, and Berens, placing the duration at two days (twenty-four to forty-eight hours); other periods recorded are as follows: one to three days;[1] two to three days;[2] three days;[3] three to four days;[4] four days;[5] a few hours to five days;[6] two to six days;[7] six to seven days;[8] twelve hours to three days;[9] one day;[10] two hours to a half-day;[11] about a week.[12]

Most cases, except those in a severe epidemic, will have the shorter period of invasion and present but few symptoms.

Stage of Eruption.—After the existence of prodromal symptoms which have lasted a variable period, from a few hours to a week, the characteristic eruption appears. In some cases the prodromal symptoms have been so slight that apparently the eruption appears without them, and, as Thomas tells us, the child is found covered with it after a quiet night's sleep. Rapid extension of the eruption progressively downwards, about in the following order, was most frequently noted in my cases: face, neck, chest, arms, back, groin, and lower extremities. Many writers have observed the same course in the eruption. Some, however, have noted other points for its first appearance. For example, Earle observed it first on the neck and chest, but adds that the exanthem may be preceded some hours by a redness of the forehead and cheeks, really an erythema, and then the spots make their appearance. Willcocks and Carpenter state that the eruption first appears on the face at the margin of the hair. Morris and Liveing regard its first appearance to be on the back and chest; whereas Murchison and Balfour state that it is first seen on the breast and arms. Patterson and Copland are of the opinion that it comes out all over the body at once.

In considering this subject we must bear in mind that the eruption of rubella is by no means uniform, and that it differs in various epidemics and in isolated cases.

In some of Griffith's cases the eruption appeared on the brow, the body of the lower jaw, and the neck, but not on any other part of the body.

---

[1] Mettenheimer, Henderson, Emminghaus, Roth, Thierfelder, Kingsley.

[2] Cuomo, Cheadle, Copland.

[3] Cullingworth and myself.

[4] Aitken, Patterson.

[5] Lindwurm.

[6] Hemming.

[7] Balfour.

[8] Clausen.

[9] Roth.

[10] Veale, Day.

[11] Thomas.

[12] Willcocks and Carpenter.

Park has observed that the eruption is often seen first on the roof of the mouth, then appears on the neck. Heim is further of the opinion that there may be in this disease a local eruption; and the case recorded by Reed, in which the eruption appeared only upon the tonsils and velum palati, no rash whatever manifesting itself on any external part of the body, gives strength to the statement.

My own experience is that no part of the body is exempt, not even the soles and palms, as stated by Emminghaus and Smith. On the other hand, I have noted a local eruption confined to a small part of the brow, face, or neck, in which the diagnosis, unassisted by the known presence of an epidemic of the disease, would have been very difficult indeed.

Tonge-Smith has noted the eruption within the oral circle.

In my own cases the rash was multiform in character, more or less confluent, occasionally ill defined, in color rosy or pale red. A few cases of the brightest scarlet and some purplish tints were observed. The rash was punctated; small macules were noted; over the more vascular parts the rash was sometimes elevated, producing a rough skin easily detected by the touch. The patches were very irregular in outline, shape, and size, the last factor being the most irregular. The centre of each patch was much higher in color than any other part.

Much hyperæmia of the intervening skin was present in many cases; itching was then a more marked symptom. In rare instances the eruption goes on to the formation, upon hyperæmic spots, of a varying number of vesicles resembling miliaria.

In my experience the eruption was generally discrete, and had but little tendency to become confluent: when confluence occurred it was most marked on the face or on the extremities, particularly the joint-surfaces and those parts warmed by contact, as the groin or nates. On the posterior surface of the body the rash is paler and of a different color,—more brownish.

Griffith states that pressure has much to do with the character of the eruption, and cites in illustration a patient in whom the eruption was developed in two circular bands, one around each leg, above the knee, where the garter had been worn. Klaatsch has noted the same thing.

According to Thomas, the eruption is due to a capillary hyperæmia of the papillary body of the uppermost layer of the corion. Heim and Patterson compare the appearance of the eruptive patch in color to that produced by a writing-quill dipped in red ink and having its point placed on moist white paper. Thomas, however, considers the eruption in color to be a pale rose-red, not so red as scarlatina, nor so bluish as measles. I have never seen the eruption of such an intense red as indicated by the comparison of Heim and Patterson.

Elevation of the rash above the skin has also been noted by several observers: indeed, Aitken states that it is more elevated even than in measles. Gerhardt and Thomas, with others, have observed that the eruption has smooth or irregular indented edges, and that when it occurs in the latter form the lesions are sometimes connected with one another by little prolongations. I have never observed this character of rash, but Emminghaus speaks of small blood-vessels which are seen in the skin, and Cuomo has observed a similar appearance. These observers also refer to a marbled condition of the skin. Dunlop says that he has seen petechiæ; Cheadle has noted the same thing, as has also Erskine, in the uvula and soft palate; and Glaister has observed a purpuric rash. Griffith and Clausen have noted a shot-like sensation beneath the skin. The former observer states that the eruption in this particular case appeared on the first day, and was composed principally of annular spots, from the size of a pea to that of a silver three-cent piece, of a pale rose-red color, with a distinct yellowish tinge in the centre.

The eruption may seemingly appear and disappear within twenty-four hours; but this is probably because it attains its maximum intensity in the various parts of the body at different times, and not because the eruption has totally disappeared at any one time. Griffith, however, states that he has seen one case in which it was invisible during one day and returned, and mentions a case of Musser's in which the rash was kept visible only by the repeated use of hot baths. This observer concludes from his own experience that there are two easily-recognizable types of anomalies in the typical eruption that we have been considering, and styles them (1) *rubella morbilliforme*, and (2) *rubella scarlatiniforme*. The former is composed of spots nearly or fully the size of a split pea, more or less grouped, and in this respect resembling measles very closely. I have noted many cases of this character, in which, but for the known presence of an epidemic of rubella, if considering the skin-appearances alone, I should have been compelled to hesitate in my diagnosis.

The latter form closely resembles the skin-appearances of scarlatina. Here again the resemblance is close indeed, and we can readily appreciate the confusion of the earlier writers which has handed the disease down to us as a "hybrid." In these cases the color is more vivid and the redness more uniform, the patches increasing in area and coalescing until the resemblance to scarlatina is almost absolute. Heim states that we may isolate the original lesions by pressing the surface firmly with the finger, when they will become less anæmic than the surrounding areas. In these cases of scarlatiniform eruption macules and papules are also present, but not so well marked as in the other form. The following observers have noted the close resemblance which the eruption may sometimes have to measles or scarlatina. Harrison, Klaatsch, Copland, and Goodhart consider that it may resemble either measles or scarlatina, and Byers, Picot, and Henderson have observed many cases in which it was morbilliform in one part and scarlatiniform in another portion of the same patient, Ladell adding three cases of this nature to those already recorded. Dukes and Kassowitz state that it may resemble measles, and Murchison, Liveing, and Tonge-Smith that it closely simulates scarlet fever. This list could be almost indefinitely prolonged, but to no purpose: sufficient has been cited to show that the eruption of rubella is indeed multiform in character. To my mind, a too minute and restricted consideration of the conditions of the skin is responsible for a vast amount of the confusion which has arisen in the recognition and classification of this disease, and for the various opinions that prevail in regard to the nature of the exanthem. If we pay more attention to the general constitutional condition of the patient, less confusion will arise, and the literature will not present the distinction made by a recent Russian writer of a rubeola scarlatinosa and a rubeola morbillosa. After all, we may agree with Maton, an early English writer, that the true distinctions of this disease are to be founded on the more general and constitutional conditions. In conclusion I can but refer to the three forms of eruption described by Thomas, depending on the size of the spots,—a classification which to my mind is decidedly strained,—and that of Emminghaus, who describes a confluent and a discrete type, Nymann and Klaatsch adding their quota by recording a punctate type and another whose characteristics are large spots. The eruption does not reach its height in all parts of the body at the same time: indeed, it is fading in one part and appearing in another, so that the part first affected has usually returned to the normal by the time the patches that last appeared have reached their maximum. These local areas of eruption have a developmental duration of a few hours to a day. My own experience has been that the face and upper chest are the regions of most persistent eruption, and that, while it appears in these regions usually first, it also remains there the longest. Many writers consider that the fact of the eruption reaching its height in different parts of the body at different times is a diagnostic sign of the greatest importance; I, however, have been unable to attach the same value to this matter as do Emminghaus, Mettenheimer, Hardaway, and Roth.

The total duration of the rash is much influenced by the character and type of the epidemic, and has been variously reported by different observers. The average duration in over two hundred cases of my own is five days. In this series the shortest was scarcely two days, and the longest of all the cases was fifteen. Writers have noticed the greatest variability in this period: for example, Clausen and J. L. Smith, three days; Emminghaus and Kingsley, two to four days; Maton and Picot, three to four days; Copland and Aitken, four to five days; Hatfield, four days; Willcocks and Carpenter, one to four days; Liveing, five to seven days; Klaatsch, one to five days; Trousseau, one to two days. Alexander has observed the duration to be almost as long (fourteen days) as that recorded in one of our cases. On the other hand, Gerhardt has seen it last but from one-half to one day, and Griffith, one to five days. We may conclude, from this mass of testimony, first, that the duration of the eruption of rubella is very variable, from one to fifteen days, and, secondly, that its average duration may be placed at from three to four days.

Desquamation.—The eruption in all my cases was followed by desquamation of furfuraceous scales. In quite a number of the cases the desquamation was well marked, in others only on particular parts of the body, in these instances especially about the nose. The buccal cavity also partook of the general desquamation; it was here best marked in the throat proper. The larger scales were those from the hands and feet. Usually the peeling was by furfuraceous scales, and it always commenced in the centre of an eruptive patch, thence extending to the circumference. There is not entire unanimity among observers as to the presence of this desquamation, but Griffith, Aitken, Patterson, Squire, Sholl, and Hemming have all observed it. On the other hand, it has never been observed by several writers of acknowledged worth, as Steiner, Thomas, Goodhart, Bourneville, and others. A delicate brownish-yellow pigmentation was not infrequently observed after the eruption had subsided: this coloring did not appear to bear any relation to the color of the eruption or its severity. This condition has been noted by many observers,—Griffith, Thomas, Rilliet and Barthez, Emminghaus, Cheadle, and Stone, who speak of a staining of the face lasting two or three days, while Kassowitz notes a mottling of the skin, lasting about eight days after recovery. In my own cases the duration of the desquamation was very indefinite: in no instance, however, have I seen it last over twenty days, and rarely so long. Squire has noticed its persistence until the third week, and Sholl until the fortieth day. A fair average duration would perhaps be about three days.

Symptoms of the Stage of Eruption.—Superadded to the previously-existing symptoms which we have already detailed under the head of incubation, the eruptive stage presented a rise in temperature of from 1° to 3° F.; 103° and 104° F. are recorded in my notes, the temperature, as a rule, being in proportion to the extent and severity of the eruption. Davis[1] records a temperature of 106° F. in a young boy, with livid eruption and convulsions and a rapid running pulse; Haig-Brown[2] one of 105° F. Once more do we meet a conflict of opinion regarding the presence of fever. Eustace Smith recognizes a slight febrile condition during the invasion stage, but has not observed that it is increased by the appearance of the eruption. Indeed, other writers say that it diminishes at this time. On the other hand, Griffith's experience agrees with my own, noting a temperature of 103° F., and in a considerable number of cases 100° F. is recorded, the temperature remaining at this point in many cases after the rash had entirely disappeared; in other cases the temperature fell abruptly to normal while the rash was still apparent. High temperatures are recorded by Fox, McLeod, Cheadle, and Patterson, ranging from 100° to 105° F.

The temperature-curve of rubella may be very variable, sudden rises and equally sudden falls, or it may rise but a few fifths above the normal throughout the entire case. Indeed, some, as Wunderlich, Earle, Picot, and Vogel, assert that there may be no fever at all, and others that it lasts but a few hours, at most a day; while others, again, do not recognize any association between the onset of the fever and the appearance or intensity of the eruption. Griffith has noted a wide-spread eruption with complete apyrexia.

---

[1] Brit. Med. Jour., Oct. 8, 1887, p. 767.

[2] Ibid., 1887.

Sore throat was always present in my cases, and enlargement of the tonsils to a great extent. Many of the cases also presented marked pharyngitis and dysphagia. This is the experience of almost every writer on the subject. Park is the only exception: he limits sore throat to twenty per cent. The condition of the throat in this disease is of marked diagnostic importance. Murchison, Liveing, Aitken, Copland, Hemming, Burnie, and many others have noted its occurrence. Thomas endeavors to show that the anterior and posterior parts of the throat are equally affected in rubella, while in scarlatina only the posterior parts are affected. I have also found in some cases an eruption scattered over the throat: this has been noted by several other observers. Thomas, Emminghaus, Griffith, Aitken, Cheadle, Patterson, myself, and others have noted hoarseness more or less severe.

Many of my little patients complained of a sense of constriction of the chest, and a cough was generally present, increasing in frequency and severity and sometimes becoming somewhat laryngeal. In quite a large proportion of the cases bronchitic râles more or less diffused were noted. These catarrhal and anginose symptoms lasted about as long as the eruption, so that, as a rule, they had entirely disappeared about the fourth or fifth day; although a slight cough and some hyperæmia of the throat may remain for some days longer. Shoemaker has noted a case in which sore throat lasted eighteen days from the first appearance of the eruption.

Enlargement and induration of the cervical, post-cervical, and post-auricular glands were present during the eruptive stage: occasionally only one or two glands were affected, in other cases the entire chain. This we may consider one of the most diagnostic signs of the disease. This statement is supported by the almost universal testimony of writers for the past seventy-five years: indeed, since 1815 it has not been disputed. Some writers, as we have already stated, say that it may be detected even before the eruption becomes visible, and place it under the category of prodromal symptoms. Eustace Smith and Kassowitz are the only writers that we have been able to find who do not recognize the constant presence of this symptom, the former noting it in only some epidemics, and the latter in but thirty-three per cent. of his cases.

The lymphatic glands in other parts of the body may also enlarge and become indurated, although I have never noted this condition; but Musser has observed a tumefaction of the axillary and inguinal glands, especially the latter. Thomas, Klaatsch, Emminghaus, Hardaway, and others have noted enlargement of the glands in various parts of the body.

In a fair proportion of our earlier cases, vomiting occurred as the eruption was approaching its maximum. In five of these cases it was almost uncontrollable. My experience in this respect is somewhat unique,—probably on account of the fact that these earlier cases occurred during a severe hospital epidemic, as I have never observed the same condition obtaining in private practice. This is the experience of most writers, their reports mentioning nausea and vomiting in the severest cases only.

Pulse.—The pulse-respiration ratio was in all the cases maintained, it falling with the temperature, and that with the disappearance of the rash. Pulses of 120, 130, 140, and 150 were recorded. Several of the cases presented well-marked symptoms of heart-failure, which, however, was successfully combated.

Tongue.—The tongue in these cases was coated as it is in those affected with scarlet fever, but exfoliation did not occur, as it does in that disease. The strawberry-tongue was never met with. The dry brown tongue appears in the notes of the more severe cases. Balfour, Hemming, and Tripe have noted strawberry-tongue. Murchison and Burnie give testimony to the same effect: the latter considers this condition of the tongue to be part and parcel of the symptoms of the disease. It is, indeed, hard to reconcile these statements with our own experience and that of the great host of writers on the subject. We can but conclude that the cases seen by these gentlemen must have been modified by some peculiar local condition. In my own experience, cleaning in patches has been the most usual method of return to the normal appearance.

Urine.—This secretion was such as is found in all similar states,—"febrile urine." My first one hundred and sixty-six cases presented slight albuminuria in about thirty per cent., but the next hundred cases showed only three per cent. with albuminous urine. In the first series nine cases presented well-marked albuminous urine (one-fifteenth bulk), with dropsy. In none of the cases could tube-casts be detected.

Let us for a moment consider the experience of other observers in regard to the presence of albumen. Hatfield has observed it twice, Cuomo three times. A case is reported by each of the following,—Cheadle, Duckworth, Reed, and Kingsley. Emminghaus records the condition as a possibility, and Roberts states that transient albuminuria is not uncommon, but in very rare instances does acute renal disease with dropsy arise. Curtman[1] states that he sometimes met nephritic trouble. Hardaway, Squire, Tonge-Smith, and Mettenheimer have never observed it: the former goes so far as to consider it an anomalous symptom, giving rise to a doubt in the diagnosis.

Œdema of the face occurring coincidently with the eruption is recorded by Douglas, and has been observed many times by Griffith. Thierfelder notes a febrile œdema of the face. It has never been my good fortune to see such a case, neither have I noticed the odor said by Heim to attend the eruption.

I have met cases in which the eruption caused severe itching. Thi symptom has been recorded by nine other observers. Elsewhere I have called attention to a roughness of the skin where the eruption appeared on the more vascular parts. This condition has also been observed by Shoemaker, Musser, Griffith, and Golson. In Griffith's cases the roughness not only accompanied the rash, but persisted, in severe cases, for days after all redness had disappeared.

---

[1] St. Louis Cour. of Med., 1880, iii. 531.

Complications.—By far the most frequent seat of complications is the respiratory apparatus. Pneumonia occurred three times in my series of cases, and twice in Griffith's one hundred and fifty cases. A number of cases presented more or less severe bronchitis. One case of pleurisy is recorded. Ryle has also seen one case. Cheadle, Smith, Earle, Emminghaus, and Park have met severe bronchitis and pneumonia as complications.

Ten cases of enteritis and two of entero-colitis occurred among my hospital cases. About forty per cent. of these cases presented gastro-intestinal irritation. This percentage is somewhat out of the common, and is due, no doubt, to the severity of the epidemic. Cuomo has noted diarrhœa under similar circumstances. Earle has met four cases of intestinal irritation. Balfour regards colonic catarrh as a usual symptom of the disease. As a rule, the bowels are but little affected, and are generally in the condition which accompanies a slight febrile state. Musser observed one case of icterus. Stomatitis arose in four of my cases, and aphthæ in thirty. Instances of the former are also reported by Hatfield, and of the latter by Earle. Rheumatism occurred in two instances. Slagle has noted its development in one case, and Earle has had a similar experience in a few cases. I have observed in one instance tubercular meningitis develop as a complication. My experience in this respect seems to be unique. Curtman was obliged to combat abscesses in various parts of the body; he also noted renal disturbances. De Schweinitz has observed several cicatrices in the popliteal space break down and ulcerate. Golson records abscesses in the submaxillary lymphatic glands; this observer also notes a numbness following desquamation, attended by loss of motion in the arms and legs, lasting for several days.

Alexander has met five cases of facial erysipelas as a complication occurring within a week after the disappearance of the rash.

Fifty per cent. of Park's cases presented marked adenopathy in the cervical region and under the tongue. Miliaria, urticaria, and pemphigus are occasionally met with. Thierfelder observes febrile œdema of the face, and Emminghaus a similar disturbance in the legs; Mettenheimer, a naso-pharyngeal catarrh, permanent swelling of the tonsils, and inflammation of the gums; Smith is of the opinion that diphtheria is liable to follow rubella. Painful enlargement of the thyroid gland has been observed by Slagle in half a dozen cases. Roth has remarked that mumps is apt to follow rubella. Ciliary blepharitis and otorrhœa have occurred in Hardaway's experience. Cheadle also remarks that earache may develop as the rash subsides. De Schweinitz has met two cases of phlyctenular keratitis.

Relapse.—I have noted relapse to occur once on the fourth day and once on the twentieth day. Griffith has noted it in three instances, in one case after an interval of eleven days and in the other cases three weeks after the onset of the disease. Cuomo seems to have had a peculiar experience, as it occurred in all of his ninety cases; this series, however, was somewhat anomalous in many of its manifestations. Köstlin, Earle, Lindwurm, and Golson have noted relapses in a limited number of cases. Kingsley is of the opinion that it occurs frequently.

During the relapse the disease may manifest itself with all its primary vigor, or it may be attended by a lesser degree of intensity of all the symptoms, particularly the prodromal.

Prognosis and Mortality.—This depends much upon the type of the disease and the character of the epidemic, and also upon whether our deductions are made from hospital or private practice. My own experience in about the first one hundred and fifty cases, which occurred in hospitals and among the destitute class, shows a mortality of about four and one-quarter per cent. (five deaths in one hundred and sixty-six cases), whereas in private practice, and of course outside of hospital or asylum, I have yet to encounter the first death from this disease. Hatfield records a mortality of nine per cent.; Hemming, Alexander, Cuomo, Slagle, Roberts, McFarlan, and Davis report deaths; Aitken, Patterson, and Copland say that the prognosis should be guarded. On the other hand, Atkinson says that the disease almost invariably results favorably. Tonge-Smith observed no deaths in one hundred and forty-five cases, and Park none in one hundred cases. Nymann, Oesterreich, Hardaway, Thomas, Robinson, Emminghaus, and Steiner consider the prognosis altogether favorable.

Complications sometimes prove fatal. Kronenberg, quoted by Klaatsch, reports four deaths from bronchitis, pneumonia, and cerebral congestion after rubella. Of my own cases two died of pneumonia, one of pneumonia and enteritis, two of entero-colitis, and, as before remarked, one of tubercular meningitis. It is to be noted that the cause of death in all my cases was verified by post-mortem examination.

Davis's case proved fatal during the primary affection by bronchopneumonia.

Diagnosis.—Should it be necessary to diagnose a single, individual, and isolated case of rubella, we must indeed admit that some difficulty would be encountered, and that we have no diagnostic guide that can be considered positive, pathognomonic, or characteristic, but that we are constrained to rely upon the *tout ensemble*. Should the disease pursue a typical course, but little difficulty will be met in the diagnosis.

For the purpose of comparison it is well to tabulate the differential diagnostic signs. In the following table I have endeavored to present a consensus of opinion of the authorities quoted:

DIFFERENTIAL DIAGNOSIS.

Rubella (Rötheln).	Rubeola (Measles).	Scarlatina (Scarlet Fever).
	Contagiousness.	
Contagious and infectious.	Very contagious.	Very contagious.
	Incubation.	
Very variable, from seven to twenty-one days.	From nine to fourteen days, rarely less or more.	One to seven days, rarely less or more.
	Prodromal Stage.	
Often absent. Rarely longer than from twelve to seventy-two hours; the latter is unusual, and is the extreme.	About three days.	One day, or less.
Catarrhal symptoms slight; nasal, faucial, or bronchial irritation not often present. May be slight conjunctival hyperæmia.	Catarrhal symptoms severe: nose and eyes affected. Bronchial cough. Even in mild cases of measles these are more marked than in severe cases of rötheln.	
Sore throat always present. May note an eruption in throat preceding cutaneous eruption.	Sore throat occasionally. Prodromal rash in throat.	Marked sore throat.
Vomiting unusual.	Vomiting occasionally.	Vomiting usual.
Fever, slight.	Fever, diagnostic temperature-chart.	Hyperpyrexia, rapid, running pulse. Nerve-symptoms early and marked.
Enlargement of cervical, occipital, auricular, and submaxillary glands. Occasionally other glands may be painful. Frequent during prodromal stage, and may last through eruption period.	Enlarged glands uncommon; if enlarged, are not painful.	Below angle of jaw glands usually enlarged.
	Eruption.	
First on face or over whole body at once; spreads rapidly; may fade in one part and appear in another. Duration, three to four days, never more, usually less.	First on face; spreads gradually; body covered by third day. Duration, four days.	First on lower neck and upper chest; spreads more slowly. Duration, usually longer; may be six days.
Rose-red in color, rarely a dusky red.	Deep red or purplish.	Dusky or livid; intense red.
Discrete; sometimes diffuse; rarely grouped. Elevated, but smaller spots than measles.	Papules arranged in concentric groups.	Confluent, minute red points.

DIFFERENTIAL DIAGNOSIS.—*Continued.*

RUBELLA (RÖTHELN).	RUBEOLA (MEASLES).	SCARLATINA (SCARLET FEVER).
	Eruption.—(Continued.)	
During eruption slight catarrhal symptoms are aggravated, or appear for first time.	All symptoms increase with appearance of eruption.	All symptoms increase during eruptive stage.
Temperature very variable. No constant relation to other symptoms.	Temperature reaches its maximum with eruption.	Rapid rise of temperature, early.
Pulse in direct relation to fever.	Pulse depends much on presence of respiratory complications.	Pulse rapid from onset.
Tongue slightly coated; never presents the "strawberry tongue."	Tongue much coated, sometimes dry, brown, and cracked, with sordes.	"Strawberry tongue."
Albuminuria rare, but occurs during severe epidemics.	Albuminuria somewhat uncommon.	Albuminuria almost always.
	Desquamation.	
Slightly branny; almost always present.	Branny.	Flakes.

Treatment.—That many authorities should dismiss the treatment of this disease in a few words seems to us to be due to an incorrect appreciation of the gravity of the disorder. Certainly a disease which presents a recorded mortality in one epidemic of nine per cent. and in another of four and a quarter per cent.—a disease whose victims in some cases succumb as early as the fourth day of the malady—cannot but be of sufficient importance to claim our best efforts in its treatment, the more so as in many cases we have not alone the disease itself to combat, but also the serious and alarming complications which may arise during its course, the nature of which we have already fully considered.

In hospital practice, at all events, one cannot help being convinced that rubella is a distinctly epidemic and contagious disease: so that our first thought should be isolation, preferably in a large, airy room with a temperature of about 65°–70° F. Particular care must be taken that the patients are not exposed to draughts or sudden chilling of the cutaneous circulation: this must be our endeavor until all danger of complication has passed away.

In some of my own cases, those with a tendency to marked catarrh of the respiratory apparatus, with deficient secretions and harassing cough, steam was admitted to the room, as is the practice in the treatment of tracheotomy cases.

As in all the other eruptive fevers, the treatment at the onset should be expectant. Very little, if any, medicinal treatment is required. The child should be put to bed, the room somewhat darkened, and all noise and unnecessary visiting prohibited. The little patient should be allowed to drink freely, if there be much thirst, milk well diluted with lime-water, barley-water, or lithia water, whey, or weak lemon- or orange-water flavored with glycerin. An occasional cup of tea, made by adding a small quantity of tea, for flavoring only, to a cup of hot milk-and-water, will frequently be of great advantage in bringing out the eruption. If there be headache, the head can be kept cool by cloths wrung out in camphor-water or sprinkled with spirit of camphor or mint-water, and a hot foot-bath administered. Should the child be restless, sweet spirit of nitre forms undoubtedly the best sedative, and may be given with sweetened water or added to the lemonade. Should the skin be dry, and the child restless and delirious, a hot bath is indicated. A fever-mixture may be given at intervals, such as the following:

℞ Tinct. aconit. rad., ♏i;
Spts. ætheris nitrosi, ℥ss;
Liq. ammon. acetatis, q. s. ad ℥ii.
M.
Dessertspoonful every two hours p. r. n.

As has been already noted, there are cases with a tendency to intestinal catarrh more or less severe. These should be carefully watched, and, when treatment is indicated, small and repeated doses of Dover's powder and calomel, or calomel, bismuth, and pepsin, administered:

℞ Hydrarg. chlor. mitis, gr. ss;
Pulv. Doveri, gr. vi;
Pulv. aromat., gr. vi.
M. Ft. chart. vi.
One every hour, if indicated, for a child a year old.

℞ Hydrarg. chlor. mitis, gr. i;
Bismuth. subcarb., gr. xii;
Pepsini sacch., gr. xxiv.
M. Ft. chart. xii.
Sig.—One every two hours.

The diet should receive careful supervision and be graded to the requirements of each case. Mild aperient mixtures should be ordered for the bowels as indicated, and the lungs carefully examined daily. As soon as a sense of oppression or tightness about the chest is complained of, hot poultices or fomentations should be applied. The more serious of my cases were painted with a mixture of equal parts of chloroform and tincture of iodine, to which occasionally a few drops of tincture of aconite-root were added. When the cough becomes troublesome, it should be treated by the usual expectorant mixtures.[1] Patients presenting laryngeal complications must be subjected to constant steam-inhalations, together with the application of heat and moisture externally over the larynx.

---

[1] I have found the following to be a serviceable combination:

℞ Ammoniæ muriat., ʒi;
Vin. ipecac., fʒii;
Tinct. opii camph., fʒiiss;
Syr. senegæ, fʒvi;
Aquæ, q. s. ad f℥iv.
M. Teaspoonful every two hours.

Many cases will require, in addition, a general stimulant treatment by digitalis, carbonate of ammonium, wine, or brandy, and liberal liquid nourishment frequently administered. I would strongly recommend the use of an oleaginous preparation to the skin during the stages of eruption and desquamation: in the former stage for the comfort of the patient, allaying itching and aiding in the reduction of the temperature; in the latter to prevent contagion, as it may be by these fine scales that the contagion is carried. For this purpose we may use either olive oil, carbolated cold cream, or cod-liver oil, in this way contributing also to the general nutrition of our patient.

Complications are to be treated as they arise. During convalescence much care should be exercised to guard against colds. The patients should be placed upon general tonics, quinine, iron, and cod-liver oil. Suitable clothing must be insisted upon, with flannel next to the skin.

PERTUSSIS.

By T. M. DOLAN, M.D., F.R.C.P.

Synonymes.—Whooping-cough, Hooping-cough, Chin-cough, Kin-cough; Latin, Tussis convulsiva; French, Coqueluche; German, Keuchhuisten; Spanish, Tos ferina; Italian, Tusse convulsiva.

Definition.—Whooping-cough may be defined as a communicable disease, depending on a specific poison, prevailing epidemically and sporadically. It is characterized by fever, malaise, irritation of the respiratory tract, catarrh, and subsequently by a hard, dry, convulsive, paroxysmal cough. It attacks both sexes and all ages, but especially children, rarely occurring more than once. Usually it runs a course varying from three weeks to three months. It may be complicated with other lesions, as ulceration of the frænum linguæ, enlargement of the tracheo-bronchial glands, capillary bronchitis, lobular collapse, emphysema, various hemorrhages, paralysis, convulsions, jaundice, catarrhal pneumonia, tubercular meningitis, and other diseases of children.

History and Etiology.—We are told by Mason Good that under the name of *bex theroides* the disease was known to the Greeks, and to the earlier writers on medicine, by whom the convulsive cough was distinguished; but, so far as we can gather from the old writers, they appear to have included under the title many other forms of cough, especially those of a catarrhal nature accompanied by forcible and oft-repeated violent expiratory efforts. We cannot conclude that the disease described as *tussis convulsiva* by the old writers always referred to whooping-cough, though there is no doubt that the disease existed and has come down to us in unbroken succession, like the other diseases of childhood, as measles, scarlet fever, etc. It would be profitless to trace its history further back than the time of Cullen, who has furnished us with an admirable definition and description of the nature, symptoms, and treatment of the disease. According to this learned writer, whooping-cough is *morbus contagiosus, tussis convulsiva, strangulans, cum inspiratione sonora, iterata, sæpe vomitus.* The name in English phraseology has been derived from the sound, and by Cullen it is called hooping-cough from the word "hoop," supposed to signify the peculiar key-note struck by the little patient; other writers call the disease kin-cough, or chin-cough, from the word "kink," or from *kind*, the

German for "child," as it is particularly a malady of childhood. All writers acknowledge that it is a highly contagious disease, proceeding, as Cullen taught, from a contagium of a specific nature and of a singular quality and having a peculiar determination to the lungs.

We must recognize that we have to deal with a *contagium vivum*, and that whooping-cough never arises spontaneously. As surely as a thistle rises from a thistle-seed, a fig from a fig-seed, a grape from a grape-seed, so does whooping-cough arise from antecedent whooping-cough. Primary causation we must leave out of the question; immediate causation alone concerns us, for the larger question is out of the domain of experience. Practically, we know that exposure to the virus of pertussis produces the same disease, and that this virus acts under particular conditions. Thus, for instance, where a number of children are gathered together in schools, nurseries, or ill-ventilated buildings, if a primary case be introduced it will spread with rapidity among the other children, being especially favored by the individual states of health of the children. Experience further tells us that the younger the child the more liable it is to infection. Again, we know that it may be carried by infective material, in clothing, that it may be spread from house to house in this way, and that in all other respects it behaves like the contagia of scarlet fever, measles, etc.

Holding the view that it is a germ-disease, it follows the general laws of affections having a similar origin, though there are numerous objections to be met. Why should some who are exposed escape? Why are the latency, intensity, and period of incubation so various? These are objections applicable to all the zymotic diseases. We are able in a measure to explain away these difficulties by means of known laws or observations regarding the germ-diseases. There is one primary law, that the *materies morbi* of every communicable disease reproduces its kind. This primary law is controlled by objective and subjective laws: the diffusion or dispersion of germs, their static existence, the limited duration of their active existence, their development, maturity, and decay, their intermittent reproduction, depend upon climatic influences, physical forces, and are influenced by locality, latitude, and personal environment. Were it not for these controlling or regulating influences, the zymotic diseases would be much more fatal and more widely diffused than they are. Still more, what is true of reproduction in the animal world is true of disease-production. In the animal world destructive energy is warring against creative energy; millions of ova, at least of fishes, are destroyed without fertilization; and so in the vegetable world the seeds of plants cannot find favorable root, and perish. Spores of disease also perish, through not finding favorable soil, and through the agency of oxygen are rendered harmless. All this is rudimentary, but necessary to a full appreciation of the germ-theory.

In whooping-cough, then, the chain of causation proceeds as follows:

1. The primary case.
2. The primary case becomes a centre of infection, and throws off in-

fective material or spores, which may be carried from place to place in clothes, by ships or vehicles, or from one person to another.

3. The virus has a special predilection for children, and its action is influenced by various agencies, such as foul air and overcrowding, and it especially affects badly-nourished children, and children who are recovering from measles or who are teething. It has its period of incubation, varying from four to fourteen days.

4. It does not pay respect to race, and climate does not appear to control it, though seasonable influences apparently modify it.

A very important question presents itself as to how the contagium finds its mode of entrance, whether it primarily attacks the bronchial tubes and air-passages, or whether it is conveyed thither by the blood. I believe that as a germ-disease it follows the laws of other germ-diseases, that the germs enter by some of the channels by which some of the other contagia enter, and that in the blood they develop, setting up primary fever and other symptoms, subsequently attacking the pulmonary epithelium. From the lungs germs are given off as well as from other parts of the body, and we have the contagium carried about and extended. Isolation of a child suffering from whooping-cough will prevent its spreading; and when we come to consider the mortality we shall see how important it is to recognize the highly contagious nature of the disease and the importance of prophylaxis.

Mortality.—Whooping-cough is most fatal among children who do not attend school, and it has often been remarked that children of school age do not suffer from it. This is easily explained. Whooping-cough is most commonly fatal in children under three years of age. Whooping-cough ranks third in the fatal diseases of infancy in England. The following table represents this in figures:

ANNUAL DEATH-RATE PER MILLION IN ENGLAND AND WALES.

PERIOD.	MEASLES.	SCARLET FEVER.	WHOOPING-COUGH.
1838–40 (three years)	580	770	500
1841–42 and 1847–50 (six years) . .	430	870	490
1851–60 (ten years)	410	990	500
1861–70 (ten years)	440	980	530
1871–80 (ten years)	330	720	510

In London the disease has been more fatal than small-pox or measles.

DEATHS PER THOUSAND FROM ALL CAUSES IN LONDON.

PERIOD.	WHOOPING-COUGH.	MEASLES.	SMALL-POX.
1800–53	29	23	31
1854–71	36	24	16
1872–81	36	24	13

If we come to examine the deaths more particularly, we find that three-fourths of all the deaths occur in children under two years of age, the mortality of the female sex being in excess of that of the male. The mortality is also increased when whooping-cough prevails in cold weather. This is attributable to the exposure and to the absence of proper precautions on the part of parents, who are inclined to look upon whooping-cough as an ailment of little moment and requiring but little treatment. We shall presently have to allude to the importance of prevention in view of the appalling mortality of this infantile disease.

Pathology.—In pure uncomplicated whooping-cough the anatomist fails by rough examination to detect any characteristic or pathognomonic lesion. This is not surprising, and lends support to the views I advocate. The simple disease whooping-cough is rarely fatal. It is the complications that kill, and they are very numerous.

In placing pertussis in the group of diseases caused by protophytic fungi, its pathology can be revealed only by the modern methods of research, which have been so fertile of good results in other diseases of this class. Linnæus foreshadowed modern views when he endeavored to prove that *tussis sicca*, or dry cough, was produced by animalcula or had an insect origin. The insect of Linnæus is the microbe of Pasteur. Thus two great minds arrive at the same conclusion. Other observers, such as Poulet, Letzerich, and Binns, suggested the fungoid nature of pertussis. So far back as 1867, Poulet found in the sputa of pertussic patients little bodies which were then termed infusoria and classed as bacterium bacillus, and Letzerich produced whooping-cough in rabbits by inoculating the trachea with the sputa of human subjects. I have repeated a number of experiments[1] to test the action of the blood and nasal secretions, finding that the blood did not produce any effects, but that sputa and other secretions caused death. Microscopic examination of sputa revealed ordinary bacteroid forms, but in addition I perceived a microbe somewhat resembling the Spirochæte plicatilis Cohn. Yet I was assured by competent microscopists that this microbe was an illusion and was accounted for by the personal equation which must always be allowed for in microscopic work,—viz., the desire of the observer to see the microbe he wishes to see! Since the publication of this essay, great strides have been made in the cultivation and staining of micro-organisms, and other observers have followed on the same lines.

Thus, Dr. Burger, of Bonn, has been working at the same subject, and according to the *Berliner Klinische Wochenschrift*, No. 1, 1883, has described what he considered as the special micro-organisms of pertussis. They appear under an immersion-lens VII., ocular 0, of Seibert and Krafft's, as small elongated elliptical bodies of unequal lengths, the smallest being twice as long as broad. Under a very strong power, transverse subdivision can be detected in the longest specimens. They may form chains or groups, but are generally isolated and scattered singly all over the field. They bear a certain resemblance to *leptothrix buccalis*, the spores of which are often found in whooping-cough sputum; but the latter are larger and stouter, and near them the filiform mature leptothrix is always present. Occasionally some of the specific bacilli are found to be inside the mucus-cells in the sputum. The bacillus is easily prepared; it can be readily recognized if colored in the usual way by watery solutions of aniline. Fuchsin and methyl-violet were employed by Dr. Burger, and, as in the case of the bacillus tuberculosis, this micro-organism is best studied when mounted dry. Dr. Burger concludes that this bacillus is the actual producer of pertussis, because it is so abundantly produced in whooping-cough that its influence cannot be doubted, its numbers increase in direct proportion to the severity of the disease, and the course and symptoms of the affection are best explained by the development of this fungus.

---

[1] Whooping-Cough, its Pathology and Treatment, Fothergillian Prize Essay, 188 p. 16. By Thomas M. Dolan, M.D. London, Baillière, Tindall & Cox.

Dr. Burger's observations lack the test of experimental confirmation. We are, however, carried a step further by M. Afanassieff, who prepared with all the precautions for microscopical experimentation a small portion of the expectoration of a whooping-cough patient, which showed large numbers of short rod bacteria, 0.06 to 2.2 μ in length, part singly, partly in two and of larger chains. M. Afanassieff, availing himself of modern methods, made plate-cultures with them, planting a particle of the sputum upon jelly of beef peptone and beef-peptone agar-agar, of each two plates. After two or three days there appeared upon all the plates numerous, almost similar, colonies of bacteria: round or oval light-brown colonies with smooth borders, which did not liquefy the jelly; round, with slightly-toothed borders and brown centre, constituting one round large coccus. In the first-named colonies were to be seen, through the microscope, pure cultures of the above-described bacterium, which the investigator, after a careful comparison with all bacteria thus far known, was constrained to recognize as a bacterium *sui generis*, and which, inasmuch as cultures derived from the expectoration of still other whooping-cough patients furnished exactly the same bacterium, were now transplanted upon various culture-soils. This rod bacterium grows very rapidly in D'Arsonval's thermostat at the ordinary temperature of the room. It does not at all liquefy the culture-soil, and flourishes most rapidly and numerously upon the potato and upon beef-peptone agar. Upon the second day there is to be seen a distinct pellicle, at first of a transparent gray, later on becoming perfectly white. Similar is the pellicle upon sterilized blood serum, only that here it does not spread out far, but remains stationary at a certain stage of its growth. Upon the potato the pellicle, too, is thinner, gray, with a rough surface and irregular borders, which by the eighth and ninth days is strongly toothed. With pure cultures of these rod bacteria the investigator has made eighteen experimental inoculations upon animals. A solution of this culture upon agar-agar at least eight days old and one-half cubic centimetre of common salt was made and injected into the windpipe or lung of dogs and rabbits, of course under antiseptic precautions. The animals all contracted a disease similar to whooping-cough, often complicated with broncho-pneumonia. Several died, and section showed that the mucous membranes of the bronchi, of the trachea, and even of the nose, are the chief seats of the injected bacteria. This same bacterium was found in the lungs and respiratory mucous membranes of children who died of whooping-cough. M. Afanassieff considers it to be the true cause of whooping-cough, and names it the *bacillus tussis convulsivæ*. We are thus a step further on the way; and, as Schwenker[1] and Wenat[2] have confirmed M. Afanassieff's observations, a great lacuna has been filled up.

Though we do not find any rough pathognomonic change in simple uncomplicated whooping-cough, yet it cannot long persist without leaving some impression on various parts of the frame. I have spoken of one important lesion. The imperfect aëration of the blood, the disturbance of the circulation, the very concussion produced when in a severe paroxysm the child is shaken from head to foot, grasping with instinctive haste any support it can lay hold of to break the force of the concussion, the incessant, teasing, harassing cough, the vomiting, cannot occur without altering in some way either the texture of the mucous lining of the throat, bronchiæ, or bowels, or the structure of the lung, the heart, or the brain and its meninges.

We may briefly mention the morbid changes found in the principal organs.

Brain and Membranes.—The post-mortem appearances are what might naturally be expected from the phenomena, and are appreciable to the eye. The minute vessels are injected and enlarged; there is cerebral engorgement, with effusion of fluid into the ventricles. There are no signs of softening. The vessels of the membranes are frequently in a similar condition, and we find the spinal cord and its coverings also congested. In view of convulsions, especially occurring in infants under one year of age, the congested state of the brain is important.

Mucous Membrane of Eyes, Nose, Bronchi, and Stomach.—We find the mucous membranes in a highly injected and irritable condition, in life the conjunctivæ are frequently seen in a state of intense congestion, and we have hemorrhage from over-distention of the blood-vessels caused by the violence of the paroxysms. The irritable, red, swollen condition is a primary occurrence. Under the influence of irritation, mucus is secreted, and after death we frequently see the bronchi filled with abundance of thick mucus occupying the cavities of the air-tubes. Owing to exposure, inflammation sets up, of which we have the usual signs. Vomiting very frequently occurs; and hence the mucous lining of the stomach shares in the general congested state.

---

[1] Schwenker, Lancet, Jan. 7, 1888.

[2] Wenat, Medical News, June 2, 1888.

Emphysema.—Emphysema, according to Steffen, is seldom absent in the lungs of children who have died of whooping-cough. According to West, during the violent expiratory efforts of the closed glottis which characterize a paroxysm of the cough, the air is driven forcibly towards the upper parts of the circumference of the lungs, and hence its seat is marginal. Emphysema is attendant on expiration. Atrophy also plays a part in its production. The air-cavities are subjected to pressure and strain, owing to the constantly-occurring paroxysms.

Pulmonary Collapse.—In 1830, Sir John Alderson described the anatomical characters of collapse; and we are really indebted to him for our knowledge of this condition. His observations have been corroborated by subsequent observers. He differentiated between collapse and pneumonia. The following short passage embraces his views. Speaking of the appearances usually found, he says, "In the lower and posterior portions of the lungs the structure was rendered very firm and dense; the portions which were the subjects of this change were exactly defined by the septa, of a dull-red color, devoid of air, sinking instantly in water, and thin slices undergoing no change by ablution. The individual lobules were more dense than in hepatized lungs; and the cellular membrane between them, retaining its natural structure, conveyed to the touch the same sensation that is felt on touching the pancreas. . . . I apprehend that the appearances detailed differ from those found in peripneumony. In whooping-cough the lung is always dense and contracted, as if the air had been expelled and from the throwing out of adhesive matter the sides of the air-cells had been agglutinated together, while in hepatization the lung is less dense than in whooping-cough, and is rendered more voluminous than in its natural state. Pulmonary collapse is the result very frequently of bronchitis. Thus, if one or more of the tubes become choked up with mucus during expiration, some air is forced out by the side of the mucus, but each respiration draws the phlegm into a narrower part of the tube. Air is expelled, but none is taken in; the consequence is, that the air-sacs collapse."

Following on collapse we have condensation of the pulmonary tissue, as described by Sir J. Alderson. This lesion is one of the most important in connection with the secondary pathological changes in whooping-cough. It is characteristic.

Capillary Bronchitis.—As the result of exposure to cold or of an unequal temperature, capillary bronchitis is one of the most frequent complications in whooping-cough: there is nothing to distinguish it when combined with pertussis. We have the usual inflammatory state of the large and small air-tubes; we find their mucous lining soft, turgescent, injected, with mucus in abundance, blocking up the air-vessels and interfering with the proper aëration of the blood. With more intense inflammation the bronchioles in place of being filled with mucus exhibit a copious secretion of pus. As the result of bronchitis, the bronchi become dilated, with long-continued cough and expectoration, the elasticity of the bronchial tubes is impaired and their muscular activity slackened, and hence they yield to the distending influence of cough in inspiration and to the accumulated secretions. This dilatation is not unfrequently met with. If uniform, the whole calibre of the tube is affected; but if saccular, a number of bead-like dilatations will be seen, within which may be found some mucus, which, owing to the peculiar form of dilatation, cannot be got rid of: we have bronchiectasis. Dilatation of the minute bronchi may be especially noticed at the periphery of the lung; and if we make a section of the smaller tubes we shall find the connective tissue and epithelium destroyed.

Enlargement of the Tracheo-bronchial Glands.—Enlargement of the tracheal and bronchial glands is very frequently met with in delicate and strumous children in whom there is enlargement of the cervical, inguinal, and other superficial glands. Owing to the occurrence of this enlargement in children who have died of whooping-cough, Dr. Guéneau de Mussy supported the view that whooping-cough was essentially an affection of these glands, a bronchial adenopathy. We find these glands enlarged from various causes in other diseases, especially among children brought up amidst unsanitary surroundings and on bad and impoverished diet. Yet we do not find the usual phenomena of pertussis.

Pneumonia.—As the result of cold and other causes, we have pneumonia, and, associated with it, inflammation of the pleura; but the post-mortem appearances will be found to be exactly identical with those observed when the patient has died from pneumonia uncomplicated with whooping-cough. According to the time at which the patient has died, so shall we find the morbid condition of the lung. We may have one of the four stages of pneumonia: 1, the lung simply congested; 2, engorged; 3, hepatized, with red coloration; 4, hepatized, with gray coloration.

It is unnecessary to enter into the pathology of these conditions or to describe the post-mortem appearances of all these stages, as they are given in all our text-books, and may be found under the article on pneumonia in this work.

State of the Heart.—We know that the circulation is disturbed, and the perfect aëration of the blood interfered with, in pertussis. We have but to look at a child in a paroxysm of coughing to see this. As the result of this disturbance of the balance of circulation and aëration, the heart must be thrown out of gear, and its action made irregular. After a paroxysm the child pants for breath. If you place your hand over the cardiac region in this state, you will find that the child is breathing with renewed energy. You can feel the heart palpitating, pumping away with vigorous strokes the imperfectly-aërated blood. Does this disturbance produce any permanent alteration in the heart? Are the valves affected? Have we to dread mischief in the future? Do we find whooping-cough complicated with pericarditis or endocarditis?

As a rule, we may say that whooping-cough does not leave behind it any permanent cardiac lesion. We may find the coronary arteries filled with blood, distended, changes obvious to the naked eye, but we do not find under the microscope any alteration in the muscular fibres, such as degeneration, proliferation, or infiltration. We may find some slight signs of irritation in the chambers of the heart, and the small vessels in the intermuscular connective tissue may be intensely injected. Consequent upon the irregular action of the heart, we have a corresponding irregularity in the pulse; the rhythm and force are altered; but this trouble soon passes away, and after the attack of whooping-cough has completely vanished the pulse regains its normal character.

State of the Kidneys.—Gibb was, I believe, the first to point out that the urine is frequently in a saccharine state in pertussis. Great stress was laid upon this announcement, and at the time it was believed that this condition threw light upon the pathology, and that the nervous theory was thereby supported.

Is the urine always saccharine?

I believe it to be the exception to find it so. I have examined the urine of fifty children with confirmed whooping-cough, and could find traces of it in only thirteen. It is possible to account for the presence of sugar in the urine of children without ascribing it to the irritation of any nerve, if we remember the kind of diet upon which children live.

In what state are the kidneys?

They share in the general congestive state of the other organs. Steffen says that albumen may be found in the urine at the time of violent seizures or shortly after them, but that investigations are wanting as to whether admixtures of blood are always present in it. I can supply this hiatus. Under the microscope blood-corpuscles are not found. I have never seen hemorrhage in the kidneys during whooping-cough, nor even blood in the urine.

Some other Complications.—We may have general disturbance of the nervous system produced by the long continuance of the cough and the paroxysms. In infants in whom the process of dentition is still going on, this disturbance may lead to formidable convulsive seizures, especially in irritable children. We may occasionally meet with paralysis, or with jaundice.

On the value of a constant lesion found in pertussis, viz., ulceration of the frænum linguæ.—This is a lesion very frequently found in whooping-cough, though there are different opinions as to its value and its relation to the disease. Some consider the lesion as constant, initial, anterior to the kink or cough, and related to the different phases of the malady. I support the view that this lesion is produced by mechanical action,—viz., by contact of the tongue during the seizures with the lower incisors.

On the phenomena of the kink or cough.—In no other disease do we meet with such a cough. It has a character peculiar to itself: it is known to every mother, and the diagnosis is soon made out when once you hear this kink. It is unlike the cough of laryngismus stridulus. How is it caused?

Coughing is a common effort, consisting in the first place of a deep and long-drawn inspiration, by means of which the lungs are well filled with air, this being followed by a complete closure of the glottis, followed again by a sudden and forcible expiration, in the midst of which the glottis suddenly opens and a blast of air is driven through the upper respiratory passages. Coughing is a reflex act. But there is something more in the cough of pertussis. In what does the difference consist? and what are the pathological conclusions, if any? There are two stages in the paroxysm.

In the first stage a number of expiratory efforts are made in quick succession, during which the air is driven out of the lungs in jerks of varying degrees of violence. During this stage no air is taken in to make up for what is lost. The blood is thus imperfectly aërated, and the patient seems on the point of being suffocated. In the second stage there is exhaustion of the paroxysm followed by a long-drawn act of inspiration. At this period the peculiar crow, kink, or whoop so characteristic of the disease is heard. The violent expiratory efforts, followed in turn by inspiratory efforts, recur again and again under the influence of reflex irritation. The paroxysm may go on until the irritation is removed, expectoration or vomiting accomplishing this.

The question naturally arises, In what condition are the lungs during the paroxysm? If the chest be auscultated between the short intervals of expiration and inspiration, you will hear some wheezing or vesicular breathing; but if the ear be applied to the chest during the long-drawn noisy inspiration, there is nothing to be heard. How can we account for this? Several hypotheses have been put forward.

1. It has been supposed to result from the slow and imperfect manner in which the air passes to the lungs through the chink of the glottis, which is spasmodically narrowed.

2. Laennec believed that it depended in part upon a spasmodic condition of the muscular or contractile fibres of the bronchi and their branches. We have no post-mortem evidence to confirm Laennec's view; but if it were possible to examine the lungs in this stage, I am of opinion that it would be found correct, and I am strengthened in this by the more recent researches of Charcot on the minute anatomy of the lung. We must bear in mind the minute anatomy of the lung if we would fully appreciate why all is silent in the chest during the respiratory stage,—the causes at work in the production of emphysema, collapse of the lung, and other secondary conditions in the pathology of whooping-cough. When the respiratory act takes place, the air at first does not penetrate beyond the larger bronchi, and is long before it again freely permeates the pulmonary vesicles. And why? Because, though the larger bronchi are patent, the muscular fibres of the smaller and ultimate bronchioles are closed.

In the production of emphysema I accept the respiratory theory advanced by Sir William Jenner. The minute anatomy is also important in relation to the part played by the pneumogastric nerve in whooping-cough.

Roughly stated, the lungs are supplied from the anterior and posterior pulmonary plexuses, formed chiefly by branches from the sympathetic and pneumogastric. The filaments from these plexuses accompany the bronchial tubes, upon which they are lost. Small ganglia are formed upon these nerves; irritation of these nerves is said to have the effect of producing contraction of the bronchial canals sufficient to expel a certain quantity of air. If this theory be true, it helps us in explaining why the larger, mediate, and smaller bronchi are closed during the expiratory stage of the pertussoid paroxysmal cough. Autenreith suggested that inflammation of the vagus was the primary cause of whooping-cough; but this view is negatived by the practical outcome of post-mortem examinations, the vagi being found perfectly healthy in the majority of cases.

Summary.—A recapitulation of my data on the pathogenesis of pertussis may help the reader to grasp them:

1. Pertussis depends on a specific poison or contagium: this is universally admitted.

2. This contagium is active, highly infective: this is granted by all observers.

3. The contagium is comparable to the contagia which produce splenic fever, scarlet fever, variola, measles, etc.

4. It has a particular determination to the lungs.

5. Like all the other contagia, it has its periods of activity and of decline.

6. The period of greatest activity is in the first and second stages.

7. Pertussis runs a regular course, like measles, scarlet fever, and variola, and rarely attacks a person more than once.

8. It thus must be classed among the zymotic diseases.

9. The fact that there is no primary pathognomonic morbid change supports this view.

I hold the zymotic theory to be the most satisfactory thus far propounded from a pathological light, whilst at the same time it harmonizes with the methods of treatment recommended by the best clinicians, and renders them intelligible. Treatment is even more valuable than are hypotheses about pathogenesis, and the treatment may be correct even though our pathological views be wrong.

Symptomatology.—Authors have divided the disease into three stages, corresponding to the *stadium prodromii*, *stadium convulsivum*, and *stadium decrementi;* but this is an artificial classification, as the stages are not always sharply defined. This division is, however, useful. We do not often see the disease in the first stage, as mothers do not bring their children for treatment until the distinctive paroxysms have appeared, when we hear the whoop or kink which proclaims in crowing accents, as it were of jubilation, "whooping-cough."

This first stage comes on unsuspectedly and insidiously: the child may have some slight fever, malaise, be restless, cross, with some symptoms of catarrh; the mother thinks the child has a cold, which will soon pass away under a little domestic treatment. Castor oil is given, oil or tallow and nutmeg are rubbed on the chest, and the mother has the satisfaction of finding the child better in the morning. It is so well that it is allowed to go out without any extra precaution, or without considering the state of the atmosphere. On its return the cough is worse and the infant exhibits more manifest symptoms: there may be some discharge from the nose, the cough is more urgent and teasing, the child is more restless, uneasy, and cries as if in pain. This stage progresses, and we have still more pronounced symptoms of catarrh. A little more care may then be taken by the mother; the child may be nursed, kept in the house, and there may be again an improvement, whereupon there is a remission of watchfulness on the part of the mother or nurse. The child is taken from a warm to a cold room, or after having been warmly wrapped up the extra clothing is taken off. Again there is a change: the cough returns with intensity, occurring in repeated attacks, during the intervals of which the child pants for breath. Sometimes the paroxysms are so continuous that the conjunctivæ become injected. The second stage is now not far off.

How long does the first stage last? Opinions vary. I have known the whooping stage develop in two days, but I have also known it take fourteen. In some cases there has been no *stadium prodromii*. The statistics of other observers also vary. Burger has estimated it as averaging from eight to fourteen days; Lombard, from four days to six weeks; Wunderlich, from three to six days; West, from two to twenty-five days. In my opinion it must vary in accordance with the child's health, the particular receptivity of the infant, and the general environment.

May the first stage terminate the disease?

I believe not, though I have not the slightest doubt that the second and third stages may be materially modified by the care and attention bestowed in the first stage.

Should a child be removed during the first stage?

If the patient can be placed at home under ordinary favorable sanitary conditions, removal is not advisable. The disease cannot be cut short by sending the patient away to another atmosphere. Where there is overcrowding, or where hygienic arrangements are defective, there can be no question that removal would be beneficial if it could be effected without exposing others to the danger.

The second stage is but too familiar, and when the characteristic whoop is heard the nature of the disease is assured. The child may be playing about, when suddenly it prepares, as it were, for the struggle, by grasping with instinctive haste at a chair or anything else within reach. The pulse becomes rapid, the breathing short, and then the paroxysm commences, the air being forced out in sudden jerks, while a long-drawn whoop is given. We have then a repetition of the phenomena, of varying length, until vomiting ensues or the attack exhausts itself. During the paroxysm the child's face becomes turgid, and there are signs of suffocation: it has an air of distress most pitiable to see, and, as the result of repeated attacks, the face becomes puffy and swollen, the eyes congested, and in many cases there is bleeding from the nose, eyes, ears, mouth, and rectum. Many children are utterly exhausted by the attack, though others are at once able to resume their amusement. The paroxysms may be so severe as to bring on convulsions, hernia, prolapsus ani. The paroxysms are irregular in their occurrence and vary in frequency, but they are more numerous at night.

What do we learn by auscultation?

Before the paroxysm comes on, we find the general indications of catarrh, but during the crowing or whoop no sounds are audible.

How long does the *stadium convulsivum* last?

This will depend very much on the state of health of the child, and on the nature of its surroundings. Children who are in good circumstances, and who can have careful nursing and all that it implies, suffer less than those more unfavorably situated. I have known the disease to last from five to sixty days. Gerhardt fixes it at from two to ten weeks; Stenier, at from three to eight weeks; Burmier, at from four to five weeks; Barthez and Rilliet, at from fifteen to sixty-five days.

After a varying time, the paroxysms become less, and we reach the third stage. There is a gradual diminution in the intensity of the paroxysms, the cough loses its peculiar character, the whoop is less frequently heard, or is absent; the bronchial catarrh persists, but after a time this also disappears, and the course of the disease is at an end. The duration of this period varies, depending very much upon the hygienic conditions under which the patient is placed. In simple cases recovery is complete, leaving no after-effects, but in cases that have been complicated, through the complications long-lasting mischief may result.

Diagnosis and Prognosis.—The diagnosis in the first stage is difficult, but we may be assisted by examining the sputa and *detecting the bacillus tussis convulsivæ Afanassieff.* We may suspect the disease if whooping-cough is prevalent in the neighborhood. The diagnosis is simple when the peculiar cough is present.

The prognosis must depend upon the condition and age of the child. Whooping-cough is popularly supposed to be not a very serious disease: this is a fallacy, as may be seen by referring to the mortality caused by it indirectly. Experience tells us that it causes a high mortality among the poor and among badly-nourished infants. The prognosis will depend very materially upon the care which can be given by parents to the children. We have also to be guided by the severity of the paroxysms.

Treatment.—I. *Prophylaxis.*—Whooping-cough, being a disease of a specific nature and highly infectious, should be classified in the list of diseases, notification of which has to be sent to the sanitary authorities, and penalties should be enforced for wilful exposure of children suffering from whooping-cough, to the danger of other children. Children with whooping-cough play in the streets, travel by trains, cabs, omnibusses, and no notice is taken and no wonder expressed that such a thing should be allowed. It is said, "The child has only got the whooping-cough." Yes, the child has only a disease which causes one-fourth of the annual mortality of children in London,—only a disease from which thousands of children die annually; and yet we wonder at our high infant mortality. There is a great wakening to the truths of sanitary science. We must make some systematic attempt to educate the public on the preventive measures which should be adopted to limit, check, or stamp out whooping-cough.

There are few diseases about which there is more lamentable ignorance and carelessness among the public; though it is popularly believed to be communicable, yet no precautions are taken against infection.

The public, unfortunately, believe that every child must have whooping-cough, measles, and scarlet fever, and that, as it must have these diseases, the sooner it contracts them the better. We must endeavor to enlighten the public. One of the first lessons must be that whooping-cough is not a necessary disease of childhood, that children are not doomed by any laws of Providence to either measles, scarlet fever, or whooping-cough; and next we must insist that children suffering from infectious diseases shall not be allowed to play or in any way consort with other children. Contagium is what we have to contend with in whooping-cough. If it were possible on the same day to isolate all the children suffering from this disease and to keep them in quarantine for a lengthened period, whooping-cough might be stamped out. This is impracticable. We may, however, do much to check and limit it, but without the intelligent assistance of the public we are powerless.

II. *The preventive measures necessary to check the spread of this special contagium,—modified quarantine or isolation.*

Pertussis never arises spontaneously. Spreading, then, by contagium, we must establish a form of quarantine to keep the unhealthy from the healthy.

1. In public institutions where there are a large number of children, and where children are constantly coming in, quarantine is necessary and practicable. It is painful to witness the sufferings of a large number of children in paroxysms of whooping-cough. An illustration from experience will show how much can be done by isolation, combined with hygiene, to limit the spread of whooping-cough among children.

In 1869 I took charge for the first time of an institution in which there were a number of children and infants. There was a nursery, and in this nursery there was a patent cradle, in which six infants could be rocked at the same time, and in this cradle might have been seen six little human waifs, struggling, palpitating, choking, in a close, polluted atmosphere. The nursery was badly constituted. Light and ventilation were bad. As all contagia become more virulent in polluted air, I had ample opportunity of witnessing whooping-cough in some of its most painful forms.

I need not detail all the steps taken to alter the conditions of sanitation. A new nursery was opened; every article of clothing that had been used in the old nursery was destroyed; and only fresh admissions to the institution were allowed in the new nursery. The infants who had whooping-cough were detained in the old nursery, which I improved by ventilators, by the free use of deodorants, and by absolute cleanliness, so that in a little time the disease among the little infants in the old nursery came to an end. As I saw all new patients, I was able to ascertain to a certain extent the existence of the disease, and to isolate the children in another series of rooms, away from the nurseries, which were placed at my disposal by the managers. Under the new conditions, whooping-cough in the intense and aggravated form in which I first saw it became unknown.

In all public institutions, then, isolation should be practised, and hospital provision should be made for the treatment of the disease. It would be well if we could send the children of the very poorest class to a hospital when they have pertussis; I mean the class who live in one room, in which father, mother, and children live and sleep. Such a course, however, is impracticable. This class is the puzzle of sanitarians, and, I may say, of the age. How to reach them? How to help them? We must teach them the best precautions under their conditions; we must impress on them the evil caused by their allowing their children to play with other children when in states of disease. Other simple truths may be urged, as to the value of cleanliness, fresh air, light, etc.

Our difficulties become less when we have to deal with the higher and middle classes. It is possible for those who have large houses to give up a room to the infected child, to keep other children away, and to carry out a form of quarantine.

It has been recommended, when whooping-cough is epidemic, that families in a position to remove should take their children away to the country or the sea-side. This I cannot approve of. The removal is not always efficacious: in too many cases it simply means transferring the disease from one locality to another, the children frequently breaking out on their arrival with the very disease they have been carried away from.

If we could establish among all classes a belief in the value of preventive measures, the disease would become rarer, and when it did occur active steps could be taken to limit it.

Isolation, then, is the first prophylactic, and each family must be taught how to carry it out as fully as their circumstances and their house permit.

2. Clothing and articles used by patients suffering from pertussis may carry the contagion: so that all such articles should be disinfected,—if available, by heat; if not, by some form of disinfectant. The clothing of infants is not expensive, and the cheapest plán in the end is to burn all suspected clothing.

3. I need say only a few words about the general hygienic conditions of households. We know that disease loves dirt, that foul air and sewer-

air are the favorite elements in which contagion thrives: so that we must insist that the children in any infected house shall have all the advantages of cleanliness, pure water, fresh air, light, etc.

4. It will naturally be asked, Do you propose to keep the child in one room during the whole period of whooping-cough? I should not advocate doing so, but in certain cases more harm is done by sending the children out into the air than by the measure of keeping them in. The children affected should have fresh air, and they may advantageously, the wind and weather being favorable, be sent out to breathe pure atmospheric air,—to be disinfected,—care being taken not to bring the little ones in contact with other children. Oxygen is the greatest of disinfectants. Due attention must be paid to the underclothing of children suffering from pertussis; they must be warmly clad.

Oxygen is a destroyer of contagion, and, thanks to this power, the virulence of all the zymotic class of diseases is lessened: so that any objection which might arise on the ground that by sending children out the poison-germs might be wafted away, causing a spread of the disease, is minimized.

Common-sense principles must guide the mother: she must be told that in pertussis the mucous membrane is in a highly irritable condition, that a damp, penetrating wind will convert a simple case of pertussis into a complicated one, that the constitution of the child must be studied, whether strong or delicate, etc. A few days' entire confinement to the house is a less evil than one hour's exposure in unsuitable weather.

III. *Pertussis in Public Institutions.*—In public institutions children must have exercise, but with judicious management this can be effected without bringing the infected in contact with the uninfected. The exercising-ground, if small, can be used when other children are in-doors. In foundling-hospitals and other similar institutions, the ingenuity of the officers will be taxed to provide for isolation and exercise, but they will be well repaid for the trouble.

In those institutions wherein whooping-cough is now not a visitor but one of the family, looked upon as a necessary condition of babies and children, I can imagine that a serious amount of trouble will be involved in any effort at isolation, and managers may stand aghast at any attempt to stamp out, limit, or check it. It is possible to limit it; and it is a work that should be done, even though it may cost time and money.

Within my own recollection, erysipelas, hospital gangrene, and bed-sores were regarded as conditions necessary to surgical operations and hospitalism. We do not now believe such false doctrine. Erysipelas and hospital gangrene followed on dirt, uncleanliness, foul air; bed-sores were produced by bad and careless nursing, by dirt. They stood almost in the relation of cause and effect. They are now unknown in well-managed hospitals, and if they occur we may take it for granted there is some defect in the hygienic arrangements or in the nursing. Whooping-cough will flourish and thrive in a colony of infants, it will remain there, and will be handed down to the next colony, unless some steps be taken to destroy it.

It may be difficult to provide isolation for a number of infants; in certain institutions there is such a large annual supply, such an influx of infants, that the accommodation required might be thought to be excessive.

Curative and Palliative Measures.—The scientific physician does not pretend to cure scarlet fever, typhoid fever, measles, small-pox, etc., but, like the helmsman at the helm of a ship, he strives to guide their course, to pilot his patients safe to land,—to recovery,—avoiding the rocks and reefs, or, in other words, the complications which may arise. Great skill is required in this process, even though all the above diseases naturally tend to recovery. Like these diseases, whooping-cough, as a rule, runs a regular course; and the efforts of the physician must be directed to steering safely through the perils which surround it. His aid is valuable, even though he does not pretend to cure in the sense in which the word "cure" is commonly understood.

Many specifics have been introduced claiming the power of arresting the disease; most of them have proved worthless. Moreover, it would not be far from truth to say that nearly all the drugs in the Pharmacopœia have been tried: arsenic, alum, acetic acid, antimony, benzine, belladonna, bryony, chloral, cannabis indica, cantharides, cochineal, croton oil, chloroform, carbolic acid, drosera, ether, hydrocyanic acid, hyoscyamus, ipecacuanha, iodide of silver, lobelia, laudanum, morphia, nux vomica, nitric acid, petroleum, potassium salts, turpentine, salicylic acid, quinine, have been recommended and praised for their efficacy by various writers. Blistering and bleeding have had their advocates.

We have previously referred to the various hypotheses on the nature of pertussis. The treatment by the light of what I regard as the most tenable theory does not enable us to abort the disease. We must treat the disease according to the stages.

1. The first stage (*stadium prodromii*), coming on insidiously and with he ordinary symptoms of catarrh, induces parents to treat the disease hemselves, so that we do not often have an opportunity of seeing this stage. The indications for treatment are simple, and depend very much upon the constitution of the child, state of the weather, and condition of the household. If possible, and diagnosis be verifiable, precautions must be aken to protect other children from infection. Then next the child must be kept in an equable atmosphere, and be warmly clad, proper attention at he same time being paid to diet, which should be plain but nourishing. A mild aperient may be required, and some simple saline mixture may be given with advantage. We have found no advantage in departing from he old-fashioned plan of treatment in this first stage. We have not seen any case aborted by either an old- or a new-fashioned remedy. If in the first stage there has been great irritation and the cough is troublesome, a simple mixture of ipecacuanha with syrup of squill will be found of great

service. We must remember that the leading indications are to allay irritation and prevent complications. In this stage it will be found useful to disinfect the room by burning a carbon cone charged with sulphurous acid, the child being taken temporarily to another room; or, if a cone be not available, ordinary sulphur may be ignited. Other agents may be employed, as carbolic acid, eucalyptol, or thymol, to purify the atmosphere.

2. When the first stage is passed, and the peculiar paroxysm so characteristic of the disease is established, I strongly recommend the succus belladonnæ in large doses. Here the chief indications are—first, to palliate or cut short the paroxysm; secondly, to relieve the irritability of the lungs; and, thirdly, to assist expectoration. The belladonna appears to achieve its end by relieving spasm. Chlorate of potassium may be of great service, but the preference I should give to belladonna, and infants, fortunately, appear to have a tolerance for it. To keep the bronchial tubes free from the accumulation of mucus, we can call to our aid the vinum ipecacuanhæ, with syrup of squill and carbonate of potassium.

3. After a varying time the paroxysms diminish, and we reach the third stage. Here change of air—especially sea-air—is most beneficial, in some cases acting with an almost magical influence. If the cough persist, an alum mixture offers us one of the simplest and best forms of checking it. Half a grain, or even a grain, may be given three or four times a day, to a child a year old, associated with the extract of belladonna in one-thirtieth-grain dose.

Much advantage will be derived in this last stage from the use of phosphate of iron, or of cod-liver oil and malt.

The plan of treatment I have sketched will generally be sufficient to guide our little patient through pertussis when the case is simple. But we must be armed in readiness for the disturbing elements which may interfere with the safe conduct to health of the little vessel we are guiding; we must be prepared to deal with children of peculiar constitutions, with neurotic tendencies, with idiosyncrasies, and we shall have to study all these characteristics and then fix upon a medicine which will efficiently cope with them.

Thus, we may have to resort to the bromide of potassium, or the bromide of ammonium, to allay nervous irritability; in another case we may have to give hydrocyanic acid, to check vomiting; in still another we may have to fall back upon the use of quinine, not in the hope of cutting short the disease, but to sustain the organism during the struggle which is going on. We may again have to ring the changes, substituting one medicine for another, or mingling them so as to make a compound mixture in which shall be combined belladonna, bromide of potassium, bromide of ammonium, or maybe using quinine with hydrobromic acid, etc. In some cases it may be advisable to try croton chloral, or some other powerful sedative; whilst chloroform-inhalation may also be required.

Spraying the fauces has been praised, and various substances have been recommended for the purpose, as cocaine, quinine, resorcin, benzol, etc.

The latest favorite as a specific appears to be antipyrin, given in small doses in a little syrup. Much advantage will be derived from the nasal douche, using for this purpose some alkaline water with some mild disinfectant, as thymol, or permanganate of potassium: the passages are thus kept cleared from irritating matter.

J. Lewis Smith has found carbolic-acid vapor very useful. He advises "three teaspoonfuls of the saturated solution of carbolic acid in water enough to cover the bottom of the croup-kettle to the depth of two inches, and when this is brought nearly to the boiling-point the vapor is inhaled through the tubes every hour or second hour for three to five minutes." The steam atomizer can be used with a solution like the following:

℞ Acid. carbolic., ʒss;
Potass. chlorat.,
Potass. bromid., āā ʒii;
Glycerinæ, ℥ii;
Aquæ, ℥vi.[1]
M.

If the steam atomizer is not practicable, the room in which the child is confined can be saturated with steam by means of an ordinary kettle containing the solution advised by Poulet:[2]

Spt. thymol,	10	grammes.
Alcohol,	250	"
Water,	750	"

The treatment must be directed to meeting symptoms, and, by efficient surveillance, to prevent complications. The general indications during convalescence are simple: fresh air, tonics, sea-bathing, good nourishment, proper protection of the body, are all required, and these can be best secured, of course, by the richest class.

Great attention should be paid to the diet of a child with whooping-cough. The food should be in the most digestible form, in small amounts, and given at short intervals. Milk, eggs, soup, puddings, are especially indicated, and if the child's strength be much exhausted by its constant vomiting, or by the strain from the paroxysms, stimulants and tonics should be employed.

The complications which arise in whooping-cough are very numerous. I have enumerated what they are, and for their treatment must refer to the special headings under which they are described in this work. I could have wished to lay down a specific treatment for pertussis and to write out its formula, but, unfortunately, there is no specific, though we can do a great deal. We can alleviate, we can palliate, and we can prevent.

---

[1] J. Lewis Smith.

[2] Lond. Med. Record, May 15, 1884.

VARIOLA.

By A. D. BLACKADER, M.D.

Definition.—Variola is an acute fébrile and highly contagious disease affecting the whole system. It commences abruptly, after a definite period of incubation, with an initial fever of from two to four days' duration. This is followed by a cutaneous eruption passing, in determinate sequence, through the several stages of papule, vesicle, and pustule, finally desiccating and leaving small permanent cicatrices wherever the suppuration has invaded the deeper tissues of the skin. It is one of the most loathsome of diseases, and is still much dreaded, not only because of the high rate of mortality incident to it, but also on account of the liability to deformity and to impairment of function which frequently result from an attack. In the great majority of cases one attack destroys the susceptibility to subsequent contagion.

Synonymes.—Small-pox; German, Blattern, Pocken; French, Petite vérole, Picotte.

History.—The earliest records of the disease are said to be found in China, where writings on the subject date back as far as B.C. 1122 (Haeser). It appears to have been unknown to the early Greeks and Romans, although a few think that the great plague of Athens (B.C. 430–425) was none other than small-pox. Excluding this, the first appearance in Europe of a plague that we can recognize as resembling small-pox in its symptoms occurred in the sixth century and ravaged the countries along the shores of the Mediterranean. During the times of the Crusades it again appeared in epidemic form and made frightful havoc in many of the more southern districts of Europe. In England it appeared in the thirteenth century, and in Northern Germany in the fifteenth. It was conveyed to this country shortly after its discovery, and in Mexico in one year its victims were computed by millions (Curschmann). It was universally regarded as the greatest scourge of mankind, and was estimated as causing one-tenth of all the deaths among the human race (La Condamine), "while leaving on those whose lives it spared the hideous traces of its power." It was for a long time confounded with the measles, with the pest, and with other papular and pustular skin-eruptions (Curschmann). The first amelioration of the disease came through inoculation introduced from the East into England (A.D. 1717), whence the practice shortly found its way into Northern Europe and America (1721), and was of much service in limiting the dire effects of the disease. Upon the discovery of vaccination, in 1796, inoculation fell into desuetude, and was outlawed in England in 1841, as tending to propagate the disease. Since Jenner's discovery small-pox has lost the prominent place which it held in medicine, but recurring epidemics from time to time warn us that the plague has lost none of its power, and that the necessity for careful vaccination is still imperative.

Etiology.—Of the first origin of small-pox we have no knowledge. In our day every case is referred to a previous one, from which the contagion has been received either by infection or by inoculation. It is very readily conveyed through the air. According to recent opinion, it may be communicated in this way to great distances, especially from small-pox hospitals (Power). The contagium appears to be of a very clinging nature: clothing, bedding-material, and such-like, attainted by the secretions or exhalations of the body, retain it in an active condition for a long time, and, unless very carefully disinfected, may become the means of propagating the disease months, or even years, afterwards. It is liable also to be spread by persons so slightly affected by the disease that its true nature is overlooked and they are allowed to attend to their daily business and to associate with others. In children, also, cases have occurred of so mild a nature that no eruption appeared, yet were they the means of communicating the distinct disease to others (Collie). Unless extreme care be taken, it is also liable, perhaps more than any other disease, to be carried by physicians and nurses from one patient to another. The contagium is most likely communicable from the onset of the initiatory fever. Probably all the secretions and excretions of the body contain it more or less, but it is present in its most active form in the contents of the vesicles just before they become sero-purulent. The patient is infectious so long as any particle of the original eruption remains adherent to his body. Conditions of soil and climate have no effect on it. Wherever predisposed persons are subject to its contagium, there will it break out. This predisposition to it, though not equally strong in all, is very general, except when annulled by efficient vaccination or by a previous attack. It is said that a very few can boast immunity from it; but this may be considered doubtful. "Susceptible persons almost invariably contract it on their first exposure, even if the exposure be of short duration" (Collie). No period of life is exempt, although it is stated that the predisposition to it is less in the earlier months than after the first year. Even uterine life does not exclude the disease. Infants have been born either already ill with small-pox, or with traces of having gone through the disease; but this is rare. "As a rule, the fœtus escapes, even if the mother be suffering from small-pox; and if the new-born infant be vaccinated within a few hours of its birth it usually escapes, even where it is born in a small-pox ward" (Collie). Sex makes no difference in susceptibility; race does. Colored races in general are much more prone to the disease than the white; the negro is especially susceptible. It is less frequently met with in the children of the more educated classes, among whom vaccination is carefully performed, than in the poorer and less educated, who are apt to be careless. The presence of chronic disease exerts no influence on susceptibility, but children ill with another infectious disease are tolerably secure for the time being. They become again susceptible, however, during convalescence. As a rule, one attack removes the liability to contract the disease, but it is asserted that certain conditions, such as puberty, and change of climate, favor the reawakening of the predisposition. The contagium of small-pox exhibits much variation in different epidemics as regards its intensity, its course, and the appearance of special symptoms during the progress of the disease.

Pathology and Pathological Anatomy.—The present state of our knowledge does not warrant any definite statement as to the exact nature of the contagium, for, so far, it has baffled the researches of the most careful investigators. Analogy would point to the presence of some micro-organism, and it is probable that ere long some special micrococcus will be discovered bearing specific connection with the disease. The latest investigations of Guttmann have resulted only in finding in the pock-lymph the well-known staphylococcus pyogenes and micrococcus albus, neither of which has any definite connection with the specific process. The colonies of bacteria also found by investigators in the internal organs (lymph-glands, kidneys, liver, and spleen) are due to septic processes complicating small-pox, and are not peculiar to it (Baginsky).[1] The characteristic anatomical lesion (the pock) of small-pox is found in the skin, where it first appears as a discrete macule due to a circumscribed hyperæmia of the corium. Associated with this congestion, changes take place in the adjoining cells of the rete Malpighii, swelling them, and raising the outer layers of the epiderm in a hard papule. In the cells towards the centre of the lesion these changes assume a necrobiotic character, transforming the cells into cloudy flakes destitute of nuclei, while towards the periphery active proliferation takes place. At the same time an exudation of clear serum occurs from the upper layer of the corium, between these altered cells of the rete Malpighii, separating and compressing them, and gradually converting the papule into a vesicle, which increases in size under the continued exudation, until a width of two or three lines is reached. Many of these papules are formed around the orifices of a hair-follicle or sweat-gland, and the umbilication in such is produced by the less yielding nature of the epiderm at such points;

---

[1] Recently Marotta, of Naples, asserts that he has obtained a special micrococcus (micrococcus tetragonus) by making cultures in a slightly alkaline medium. The colonies thus procured have a fine orange color, and produce complete fusion of the serum or gelatin in from twenty days to a month. These solutions have an intense alkaline reaction,—a fact which he thinks may have a therapeutical bearing. Inoculations on calves, even with cultures of the seventh generation, produce pustules identical with those of vaccinia. (*Rev. Mens. des Mal. de l'Enfance*, January, 1887.) These conclusions, however, have not yet been verified by other observers.

but in others it is due to the coagulation-necrosis of the central cells which form unyielding bands, while the surrounding epithelial cells undergo proliferation and become raised with the exudation-serum. By about the sixth day the pock has become fully developed, the central bands have given way, and it is now full and rounded, while its contents have lost their opalescent character and are puriform. The surrounding tissues in the mean time have participated more or less in the inflammatory reaction, and a red and swollen areola surrounds the pock. When the pustule is mature, desiccation commences. Should the epiderm break, the contents escape, and crusts are formed, but frequently merely desiccation takes place and by the falling in of the epiderm the appearance of secondary umbilication is produced. Cicatrization goes on underneath these crusts and scabs, which are gradually shed, leaving the new tissue of a light-brownish or purplish hue. The amount of subsequent pitting depends on the loss of substance suffered by the underlying papillæ during suppuration. Similar lesions to those found on the skin, but not so typical, are found on the mucous membranes,—principally on those which are exposed to the air. Those of the mouth, pharynx, nares, larynx, and trachea are the most frequently affected, but in severe cases they are found extending into the bronchi of the second or third order, also in the upper part of the œsophagus, in the rectum near the anus, in the vulva, in the vagina, and, very rarely, in the urethra close to its orifice. The blood in fatal cases is dark and coagulates imperfectly. There is congestion of all the internal organs. The mucous membrane of the mouth and upper air-passages is hyperæmic, its surface covered with brown tenacious mucus, under which may be seen the characteristic lesions of the disease in the stage at which death occurred. In the more severe cases patches of diphtheroid membrane are not infrequently seen covering extensive ulcerations. In the lungs in children signs of bronchitis are generally present, and catarrhal pneumonia is not infrequent as a sequence. Croupous pneumonia during the eruptive stage, and pleurisy, are also met with. The exudation in pleurisy is sero-purulent at first, but it very rapidly becomes purulent. Pericarditis is sometimes observed. Endocarditis is more rare. Acute fatty degeneration of the cardiac muscular tissue has been reported by Desnos and Huchard. On the gastro-intestinal mucous membrane the follicles are found enlarged. Peyer's patches in children are swollen and congested, with the exception of those near the valves, which escape (Collie). Acute articular inflammation, with sero-purulent effusion affecting principally the large joints, is not uncommon. In the cutaneous tissues abscesses are frequent. Erysipelas is not generally met with outside of the hospitals. Gangrene of the vulva is occasionally seen. Septicæmia and pyæmia, associated with metastatic abscesses, are frequent causes of death in the later stages of the disease. In the nervous system we find few pathological changes corresponding to the severe derangements manifested. Westphal has demonstrated numerous disseminate centres of inflammation in the spinal cord as the cause of the paralysis, or ataxia, which occurs occasionally as a sequence. Collie mentions two cases where extensive hemorrhage was found in the brain, and Curschmann one where an effusion of blood took place into the nerve-sheaths. The true pustule has never been known to occur on the cornea; on the palpebral conjunctiva it is rarely if ever seen, but on the ocular conjunctiva it is not very infrequent. Its favorite position here is about half-way between the cornea and the inner canthus, and, more rarely, about midway between the cornea and the outer canthus. There are seldom more than two or three pustules. They are small and surrounded by a deep injection; their epithelial covering is soon lost, leaving a round yellowish-white ulcer, resembling an ordinary phlyctenula. The more-to-be-dreaded keratitis sets in about the fourteenth day as a small ulcer on the margin of the cornea, leading to perforation with total destruction of the eye. Iritis and inflammation of the deeper structures are very rarely met with except in connection with the above. The ear is said to suffer more frequently even than the eye, but, as a rule, no pustules extend beyond the cartilaginous portion of the auditory canal. Hyperæmia and inflammatory swelling extend, however, frequently into the deeper parts, and occasionally spread to the membrana tympani. Inflammatory manifestations have also been found on the mucous membrane of the middle ear, but never any pustules. In the slighter hemorrhagic forms we have more or less sanguineous exudations into some or many of the pocks, and into the skin immediately beneath the papillary layer. In the severer forms large and small extravasations of blood take place into the skin and underneath the vesicles or pustules, and from many of the mucous membranes. In the most malignant form, purpura variolosa, the true papules of the disease are said to be wanting; but Osler[1] says that frequently even in these cases, if carefully looked for upon the forehead and wrists, they may be discovered at the end of the second or early in the third day, but the rapidly-extending ecchymoses soon hide them, and it may afterwards be difficult even to feel them. Post-mortem examination reveals large and small hemorrhages into many of the viscera, ecchymoses under the serous membranes on the surface of the brain, heart, lungs, liver, and kidneys, and extravasations into many of the mucous membranes. There is almost invariably an extensive effusion of blood behind the kidneys into the retro-peritoneal tissues and along the course of the ureter. In addition, Ponfick and Osler call attention to the firm dense condition of the heart and abdominal glands in these forms of the disease, in contrast to the soft and friable condition of these organs found in variola vera.

Symptomatology.—*The period of incubation.* From the time of the reception of the contagium to the first appearance of the initial fever the child, as a rule, presents no abnormal symptoms, and appears to be in its ordinary health. Occasionally an amount of irritability and peevishness unusual to it may be noticed, and some authors have observed a marked pallor in the face; but such symptoms are infrequent. The length of this stage is in general from ten to thirteen days. Very rarely is it shortened to eight days or lengthened to fourteen or fifteen. In severe hemorrhagic types it is said to be from six to nine. *The stage of invasion* generally comes on suddenly with symptoms of severe fever. The young child becomes fretful and restless; its skin is hot and may be either dry or perspiring; vomiting sets in early, and is generally persistent; there may be constipation, but in young children, and in severe cases, diarrhœa generally prevails for at least the first four or five days. The respiration is hurried, drowsiness comes on, and, if old enough, the child complains of severe headache and constant pain in the loins. Frequently there is abdominal pain of a colicky character, which is increased by pressure in the epigastric region. The drowsiness may deepen into stupor, and convulsions or delirium set in. The first onset, in severe cases, may be with a convulsion, from which the child passes into a state of stupor only to be broken by repeated convulsions. In older children the first complaint is generally of chilliness, with or without a distinct rigor; this is followed by pyrexia, great prostration, vomiting, and continuous backache. Sometimes we meet with a temporary paraplegia of the lower limbs, with complaint of a feeling of numbness, and not infrequently with an incontinence of urine and fæces, which passes off in a few days. The tongue is coated, the tip and edges being of a deep red; the pharynx in many cases is congested, but not to the same extent that it is in scarlatina. There is much variation in the degree of fever. The temperature in the axilla may vary from 102° to 105° Fahr. The pulse is full and frequent, and ranges from 120 to 160. These symptoms last until the appearance of the rash, which generally takes place on the third day, though it is sometimes delayed until the fourth. Sydenham, quoted by Trousseau, says the longer the eruption is delayed the milder will the attack be; but Curschmann thinks this inference unwarranted for all epidemics. Certain it is that the violence and intensity of this stage are no reliable indications of the severity of the attack, but are often much influenced by the temperament of the child. Frequently the most violent symptoms at this stage in a nervous child eventuate in a harmless varioloid, but sometimes the tender constitution of the infant may fail beneath the severity of the onset, and death ensue before the eruption can make the diagnosis certain. In a very few the invasion-symptoms may be so mild as to be quite overlooked by the mother. One such case occurred to myself, where no cross-questioning of the mother could elicit any previous symptoms, and yet the papules were well out, and ran the distinct course of a discrete variola. Eustace Smith and Day mention similar cases. During this stage, more frequently in children than in adults, certain *temporary, or initial, rashes* occasionally make their appearance. They are apt to be misleading, and therefore require careful attention. They generally occur about the second day, but may be a little earlier or later. When erythematous in character they may generally be classed under one of two varieties,—the scarlatiniform, resembling an erysipelatous or scarlatinal rash, and the macular, closely resembling the eruption of measles. Either of these may more or less cover the whole body. In general the scarlatiniform is most marked on the lower abdominal region, in the triangle made by including the hypogastric region, the groins, and the inner surface of the thighs (crural triangle of Simon), and on the lateral thoracic, including the inner side of the arm, the axilla, and the pectoral region (brachial triangle of Simon). The macular is most common on the chest, on the extensor surface of the extremities, "especially in the neighborhood of the knees and elbows, the backs of the hands and feet, on the genitals, and, lastly, as a streak extending from the ankle upwards along the line of the extensor proprius pollicis." These rashes may occur singly or together; they generally fade as the true eruption comes out, but sometimes they more or less persist. Little prognostic importance is to be given to them. They vary much in frequency in different epidemics, and are said to precede more frequently attacks of varioloid. Curschmann states that in most instances the number of pustules was in inverse ratio to the extent of the initial rash. Often these rashes are accompanied by petechiæ, of size varying from that of a pin's head to that of a bean. Such *purpuric* rashes are almost invariably limited to the triangles of Simon, and are of much more importance than the simple erythematous rashes. Curschmann says that the only pathognomonic symptom in the initial stage is the appearance of such a hemorrhagic exanthem in Simon's crural triangle. The prognosis of such cases is in general more grave, but not always, as the subsequent attack is sometimes of the milder form. In forming an opinion we must take into account the general symptoms, and not, as Sydenham says, "go by the external appearance only."

---

[1] Canada Med. and Surg. Jour., vol. v. p. 297.

Stage of eruption. On the third day, as a rule, the true eruption of the disease makes its appearance. Coincidently with it the temperature begins to fall, the pulse becomes quieter, and an amelioration of all the symptoms takes place, except in the severer forms of the disease, when this relief is very partial and the fall in temperature is comparatively slight. The eruption in most cases may be first noticed on the face, and its earliest situations there are on the upper lip, round the alæ of the nose, on the forehead, and on the chin. Sometimes in very young children it makes its first appearance about the genitals and in the fold of the groin or about the lower part of the loin or on the thighs; but such cases are not frequent. It is rarely seen on the back of the wrists and on the neck, and spreads consecutively, in the course of the following twenty-four to forty-eight hours, over the chest, back, arms, lower part of the trunk, and, lastly, on the lower extremities; some of the papules may almost always be seen on the palmar and plantar surfaces. It is most abundant on the face and back of the hands, next on the neck and arms, least on the trunk. Any part of the cuticle that has been recently irritated will probably be earlier and more abundantly covered with the papules than elsewhere. The eruption appears as small, slightly elevated maculæ, rapidly developing into conical papules about the size of a pin's head, or a little larger, pale red in color, and distinctly indurated to the touch. Sometimes, owing to an areola which may surround it at this stage, it presents an appearance, especially upon the trunk, not unlike the developing stage of measles. On the second day these papules have become of a deeper red, larger, and more elevated, and new ones have come out in the intervening spaces, so that they appear more numerous than on the first day. Between the papules the skin may still be of normal color, but if the papules be very close there may be a diffused redness especially over the face, and the skin there may have a granular appearance. An inspection made at this time of the mouth, throat, and upper air-passages will reveal spots of deep congestion on the mucous membranes which, simultaneously with the eruption on the skin, develop into small elevations and afterwards become vesicular. While the number of the papules is to a certain extent an indication of the severity of the attack, yet their behavior, as to whether they remain separate or coalesce, is a still more important one. Hence upon this fact two types of the disease have been distinguished:

1. *Discrete small-pox*, in which the pustules, when fully developed, remain distinct, and in which the disease is generally of the milder type.

2. *Confluent small-pox*, in which the lesions coalesce, sometimes towards the end of the papular stage, but more frequently when the vesicles are changing to pustules. In this type the disease always assumes a severe character.

In addition to these we distinguish,—

3. *Hemorrhagic small-pox*, in which extravasations into the pock or underneath the skin and hemorrhages from the mucous membranes become a prominent feature. Such cases are almost invariably fatal.

4. *Modified small-pox*, in which the symptoms have been mitigated and the course of the disease more or less modified and shortened by previous vaccination or inoculation, or by a previous attack.

Other forms have been distinguished by different authors, of which the more important are *coherent or semi-confluent small-pox*, in which the pustules touch but do not coalesce, and which generally runs a favorable course; and *corymbose small-pox*, a somewhat singular and rare form of the disease, in which the eruption appears in close-set clusters about the size of the palm of the hand, or smaller, while the skin surrounding these patches is for some distance free from the disease. The rate of mortality in this type is very high. These latter forms, however, do not require special description.

We shall first describe the course of the *discrete* form, as the most typical. On the third day of the eruption a minute drop of pellucid serum may be seen at the apex of the older papules. This rapidly increases in amount, and converts the conical papules into somewhat flattened vesicles with clear opaline contents. "In the great majority early in their development an apicial depression can be seen, which later on deepens into the characteristic umbilication. This is more than a mere depression of the summit, and consists in a fixing or puckering of the peripheral part of the roof-wall, giving the lesion a crenated appearance. It may be regarded as pathognomonic of variola.[1] By the fifth day a slight turbidity may be noticed in the contents of the vesicles, and on the following day (the eighth of the illness) they are distinctly pustular on the face and hands. The eruption is now said to be mature, the pustule is pea-sized, multilocular, and more or less spherical, its umbilication having been removed by the continued exudation into it. The pock on the lower limbs, as it appears later, runs through all its stages one or two days behind that on the face. On the mucous surfaces the eruption during this stage becomes a source of much discomfort, and, in young children, of danger. It always, if at all abundant, gives rise to great irritation. After the vesicular stage is reached, the roof-wall of the lesion is very apt to give way, leaving a superficial erosion or ulceration, which may become covered with pseudo-membrane. More or less inflammation of the surrounding tissues takes place, causing pain and difficulty in swallowing. Should the larynx be implicated, there will be hoarseness and a metallic or croupy cough. In the very young, sudden attacks of suffocation, speedily ending fatally, may ensue. Even when slightly involved, the nasal passages in children become blocked and increase the difficulty of breathing. Blepharitis and slight conjunctivitis are frequently present, and the œdematous lids are with difficulty opened. In discrete cases there may be little fever during this stage, the appetite may return to some extent, the nervous symptoms abate, and a certain amount of quiet sleep be obtained.

When the contents of the pock become puriform, the disease enters upon the *stage of maturation*. The inflammatory reaction around the pock now increases, the temperature rises, and the child again becomes very restless. This is the period of the *secondary fever*. It is liable to fluctuations, and declines gradually. Its severity and duration are much dependent on the number of the papules and the type of the disease. In the milder cases it is very slight, and lasts but two or three days, but in the more severe the temperature rises very high (104°–106° F.). We may have active delirium with a full and somewhat hard pulse of 120–140, or the patient may sink into a typhoid condition with a low delirium or stupor, feeble quick pulse, and subsultus tendinum, and easily succumb to one of the many complications liable at this time. By the eleventh or twelfth day of the disease, sometimes a day or two earlier or later, the maturation is completed, and the *stage of desiccation or decline* commences. The fever now gradually abates, the tumefaction of the skin subsides, and the contents of the pustules have a tendency to dry. In most of the pustules the outer wall is ruptured, either by pressure or friction, and its contents escape and form thick yellowish crusts which afterwards deepen in color. On the face when the pustules are numerous these crusts are frequently very large. Cicatrization goes on beneath. Should the ulceration have been deep, the detachment of these crusts is liable to be slow, and frequently new crusts form with wearisome persistence after the old ones have been removed (Eustace Smith). Brown or purplish-colored cicatrices are left behind, which in time change to dead-white depressed scars with uneven edges and an irregular floor. The entire course of the illness covers a variable period of from three to five weeks.

---

[1] J. Nevins Hyde, Pepper's System of Medicine, vol. i. p. 458.

In the *confluent* type of the disease all the symptoms are more severe and show a tendency to develop more quickly than in the discrete form, which we have just described. The incubation-period is relatively short, and the initial stage is ushered in by a severe chill or convulsion. The fever runs high, and the remission on the appearance of the eruption is only slight. Even before the eruption develops, the skin shows deep congestion. The papules appear early and come out quickly over the whole body. They are dense, deeply set, and in severe cases may, even on the first day, be adherent on the face and hands. Sometimes large flattened papules appear which coalesce during the vesiculo-pustular stage to form extensive and irregularly-shaped bullæ. Even on the trunk, where there is not the same tendency to coalesce as elsewhere, the pustules frequently do not develop or fill up well, but remain flat with an ill-defined edge. This is always a bad indication. About this time the appearance of the whole face is much altered, owing to the great swelling of the skin: the lips are protuberant, the eyelids œdematous, and the nose enlarged and broadened. Thick crusts of secretion block the nares. The mucous membranes suffer severely. The eruption on them is always abundant, with much surrounding hyperæmia. The fauces are generally much swollen, and the tongue is enlarged and sometimes inflamed. There is much viscid secretion in the throat, and in many of these cases a pseudo-membrane forms on the fauces which may extend into the respiratory passages. Deglutition is extremely painful, sometimes almost impossible, and respiration is very seriously impeded. Should the larynx be attacked, rapid œdema of the glottis may take place, and should it escape that, perichondritis with necrosis is not uncommon. With the stage of maturation the fever, which has never subsided, rises still higher, there is great thirst, and often intolerable itching of the skin. Albuminuria is usually present. Delirium of a violent or low muttering form may set in. Sometimes the child remains in a half-comatose condition, with a feeble, fluttering pulse. Complications in this type of the disease are very frequent. Some of the more severe cases never properly enter the pustular stage. The vesicles instead of swelling and becoming pustular remain flat. The face assumes a dirty-white, pasty appearance, and profound exhaustion sets in. In others the eruption appears but slightly, the skin is dusky red, the circulation is very feeble, and the urine is scanty or suppressed. Such cases have been described under the term malignant. In them death usually takes place between the seventh and the ninth day.

Hemorrhagic Small-pox.—Two types of this most malignant form of the disease have been described. In the first and most severe type (purpura variolosa) the poison appears to act so gravely upon the blood as to develop an intense purpuric condition with an overwhelming prostration of the vital powers. The characteristic eruption fails to appear. In such cases, after a brief incubation-period, we meet with a violent explosion of symptoms at the commencement of the initial fever. A deep scarlatiniform, sometimes almost purplish, rash appears on the trunk and extremities, generally leaving the face free. In this rash petechiæ and vibices rapidly appear; on the extremities these remain discrete, but on the trunk they coalesce into large irregular-shaped figures of deep-black hue. At the same time blood is effused under the conjunctiva, hemorrhages occur from many of the mucous membranes, and there is frequently hæmaturia. Diphtheroid exudations take place in the pharynx, and a horrible stench adds to the frightfulness of the case. The temperature generally remains low, rising perhaps just before death. The mental faculties are frequently clear. The end may occur in a few hours, but generally two or three days elapse. In the second form, hemorrhagic symptoms set in during some stage of the eruption. The earlier such symptoms occur, the more intense is the blood-poisoning and the more grave the general condition. When they appear during the papular stage the eruption is generally sparse and the disease runs a course not unlike the previous form just described, but not so rapid. Death usually takes place in five or six days. In other cases the hemorrhagic condition occurs during the vesicular stage, and in others not until the pustular stage is reached. In such the effusion takes place into the pock, and more or less into the subjacent cellular tissue. In the more severe cases there may be hæmatemesis, hæmaturia, or melæna. Death takes place between the sixth and ninth days. In cases less grave, "if the hemorrhage be limited it is not necessarily a fatal sign, although the patient may die of small-pox, irrespective of the hemorrhage; but if it be extensive, in particular if it be largely into the skin, the case will be fatal, vaccination notwithstanding. In these cases there is usually more or less mental confusion or delirium. It is important, however, not to confound this dangerous form of the disease with an effusion of sanguinolent fluid into some of the pocks only," the result probably of some passive engorgement of the tissues (Collie).

Varioloid is the somewhat incorrect term which we apply to small-pox modified by vaccination. Inoculation and a previous attack may both produce a similar modification, but the former is now never employed in this country, and a second attack in childhood is unknown. Previous sufficient and effectual vaccination in infancy is in the majority of cases a complete protection against an attack up to the age of eight or ten years. In a very few before that age varioloid makes its appearance even after efficient vaccination, but the further we pass it the more frequent do the cases become. In varioloid the disease is always lessened in its intensity, and may be altered both in its course and in its duration. Morrow[1] says vaccination denaturalizes small-pox, deranges the regular order of its evolution, and effaces its most distinctive features. The initial stage is frequently as severe as in the unmodified form, and sometimes longer, but as often it is very mild. The temporary rashes are frequently seen. Bartholow says that the better defined they are the milder the attack will be; but this is probably liable to variation in different epidemics. Defervescence is rapid and generally complete with the commencement of the eruptive stage. The eruption of varioloid is apt to be very irregular in its appearance and its course. Instead of beginning on the face, it may make its first appearance on the back, chest, or extremities. In general the number of the pocks is much lessened, frequently under twenty altogether. Their abortion may take place at any period, convalescence ensuing rapidly. Occasionally the course may be almost typical, but the symptoms throughout be of a mild form. In most cases it is shortened and the secondary fever quite absent. Slight permanent cicatrices may be left if the pustule has fully developed. In the milder cases the danger is lest the disease be overlooked till it spread to other members of the household.

Complications and Sequelæ.—Speaking generally, we may say that there are fewer complications and sequelæ met with in this disease than in most of the specific fevers. Among those efficiently vaccinated, complications of all kinds are generally absent. The previous health of the child seems to have a strong predisposing influence, and weakly and strumous children with a tendency to pharyngeal catarrh, bronchitis, enteritis, strumous ophthalmia, etc., are very liable to suffer in these parts should the attack prove at all severe. Most of the complications met with are intimately connected with the local affection and appear towards the end of the eruptive stage. Among the most important are those of the *respiratory tract.* Pharyngitis, and œdematous and diphtheritic laryngitis, may early assume a very threatening character. As soon as dyspnœa sets in, relief should be given by tracheotomy or intubation. More or less bronchitis occurs in all the severe cases in childhood, and not infrequently is accompanied with broncho-pneumonia. Pleurisy in children is common and very fatal (Eustace Smith). It commences suddenly, runs a very rapid course, and terminates as an empyema almost invariably fatally in three or four days. Pneumonia of a passive form is liable to come on insidiously. It is slower in its progress, and not so uniformly fatal. Trousseau records an interesting case of a child of twenty months, who on the third day of the eruption was seized with dyspnœa. At the moment of performing tracheotomy two false membranes were thrown out through the wound. The child died a few hours after the operation. An autopsy showed that the pseudo-membrane extended to the larger bronchial tubes. On the right side were isolated masses of purulent pneumonia, and a small quantity of purulent effusion.

---

[1] Journal of Cutaneous and Venereal Diseases, March, 1886.

The *heart* in children occasionally suffers. Cases of pericarditis and, more rarely, endocarditis are recorded. Acute fatty degeneration of the myocardium may be a cause of sudden death. In the *digestive tract* serious complications are perhaps less frequent in children than in adults. Salivation is rare, and when it occurs is not severe and passes off in four or five days (Lewis Smith). Parotitis and glossitis are extremely rare in childhood. Obstinate vomiting and a persistent catarrhal diarrhœa are not infrequent, and add much to the exhaustion. Peritonitis is very rare. A mild orchitis or ovaritis is said to be met with sometimes. They subside coincidently with the eruption. Although albuminuria is frequent, it rarely leaves chronic mischief behind. Erysipelas is an infrequent complication. Gangrene of the vulva is liable to occur in children with catarrhal leucorrhœa, and gangrene of the finger-tips and toes is of rare occurrence. One of the most frequent of the sequelæ is a series of small boils or superficial abscesses in the subcutaneous cellular tissue. More important are the deep-seated abscesses which may occur in the extremities and seriously interrupt the progress towards recovery. They sometimes give rise to pyæmia. Periostitis may develop and be followed by caries or necrosis. Arthritis of a suppurative character is not uncommon in the larger joints. It may involve the surrounding tissues and lead to much damage, if not to actual loss of the joint. Septicæmia and pyæmia are not infrequent towards the later stages, and are a frequent cause of death. After all severe attacks the convalescent is left in a weak and very anæmic condition. In a very few, damage to the nervous system may result. Partial paralysis, aphasia, and tremors are reported, and sometimes the intellectual powers may remain weakened for many months.[1] For the following description of the special complications met with in the eye and the ear, I am indebted to Dr. J. J. Gardner, oculist, who had charge of a department of the Civic Hospital, Montreal, during the epidemic of 1885:

"*Complications met with in the eye.*—Almost every part of the eye may suffer, the lids, lachrymal sac, conjunctiva, cornea, choroid, and even the retina and extrinsic muscles. These complications may occur either during the course of the disease or afterwards. They are much more apt to occur if the type of the disease be severe, or if the patient be debilitated. Conjunctival affections are common. Some hyperæmia is always seen when the eruption occurs on the lids, but is of little moment. Occasionally we meet with a simple catarrhal ophthalmia, or more rarely a purulent conjunctivitis with great chemosis, sometimes so severe as to be mistaken for gonorrhœal ophthalmia. The variolous form, however, is not so painful, and there is less swelling of the palpebral conjunctiva, and less tendency to corneal ulceration. Keratitis may develop from a purulent conjunctivitis, or quite independently of this, but never earlier than the twelfth day (Adler). It may occur either as a circumscribed superficial infiltration, which heals under atropine and hot fomentations, or may take the form of an ulceration very dangerous to the eye. De Wecker regards this as in many cases a 'neuroparalytic keratitis.' It generally begins near the margin of the cornea, is very irritable, and seems to have a greater tendency to spread superficially than to penetrate: so that we rarely have perforation till the greater part of the cornea is destroyed. Hypopyon, with iritis, usually accompanies it, and ultimately panophthalmitis. The prognosis depends very much upon the period at which it begins, and on the severity of the general symptoms. The earlier its appearance in the course of the disease, the more dangerous it is to the eye. Complete resolution is very exceptional. The more favorable termination is a *leucoma corneæ adherens*, which is frequently total. The treatment does not differ from the general treatment of corneal ulcer, except that it must be prompt. Early and frequent examination of the cornea should be made, to note any morbid process. Owing to the great swelling of the lids, this is by no means an easy matter, but with the aid of lid-retractors it can generally be accomplished. It is best to begin treatment with frequent fomentations with a hot saturated solution of boric acid, and instillations of atropine or eserine three or four times daily. The ulcer may be dusted with a little powdered iodoform. If under this treatment the inflammation increases, the edges of the ulcer should be thoroughly cauterized with the galvano-cautery, and, if necessary, paracentesis corneæ performed. Gentle pressure should be made with a bandage. If there is much discharge the bandage must be removed very frequently and the parts cleansed, and once or twice daily the everted lids should be brushed with a solution of nitrate of silver (gr. v–x to ℥j). De Wecker claims to have had better success with eserine than with atropine. If there be no iritis it may be freely used. In lachrymal obstruction the duct must be opened. *In the ear*, according to Wendt, complications are more frequent than in the eye. The milder forms of hyperæmia are generally overlooked, as they cause no complaint. Catarrh of the middle ear is common, and is generally directly due to swelling of the naso-pharyngeal mucous membrane closing the Eustachian tubes. Sometimes this goes on to acute inflammation of the middle ear, which may end in extensive destruction of the soft parts and ossicles, with subsequent permanent deafness. The pain during the formation of pus is very intense, but subsides as soon as the membrane bursts or is incised. This complication requires careful and early treatment. One or two leeches at the outset may be applied in front of the tragus, and be followed by frequent hot bathing. This is best accomplished in the absence of a douche by allowing hot water to drop from a sponge held a few inches from the ear, the water flowing off into a basin beneath. If the pain be not quickly subdued and the membrana tympani be found bulging, an incision should be made in the most prominent point. Gentle inflation afterwards by Politzer's method daily is of great service. The ear must be cleansed by syringing with some antiseptic lotion. The naso-pharynx should at the same time receive treatment. *In the throat* the presence of the eruption gives rise to much irritation, and is the source of perhaps the commonest complaint of small-pox patients. The accompanying viscid secretion occasions dysphagia, nausea, and sometimes dyspnœa and a troublesome cough, which is very trying to weakly children. Sometimes a diphtheritic membrane appears in the soft palate and tonsils, increasing the gravity of all the symptoms. Œdema is not infreqent in children. Severe ulceration of the mucous membrane of the larynx with perichondritis and necrosis of the cartilages may occur, and be associated with abscess in surrounding tissues. The treatment of all throat complications is very unsatisfactory, especially in children. The pain and discomfort I found most relieved by warm chlorate of potash gargles followed by a spray of a weak solution of cocaine of a strength of one-half to two per cent.; but this requires caution in very young children. The parts should be kept moist by the frequent administration of small quantities of fluids, or ice. Œdema, if sufficient to cause dyspnœa, requires scarification or prompt tracheotomy, or intubation. If the suffocation be due to an abscess, it must be opened."

---

[1] H. Tomkyns, M.D., Leicester, Eng.

Mode of Death.—Death during the first few days is generally due to variolous toxæmia. Later on in the course of the disease it is most frequently owing to exhaustion. In young infants and children, with their small larynx and defective respiratory power, it not infrequently takes place by apnœa. Sometimes cerebral symptoms set in, and death follows with coma or convulsions, and rarely we have it occurring suddenly, with signs of rapid heart-failure, when the previous symptoms had apparently been favorable. It is a notable fact that, when discrete small-pox proves fatal, death generally occurs between the eighth and ninth days of the disease, while in the confluent form the most fatal period is not till between the eleventh and thirteenth days.

Prognosis.—In the unvaccinated, the younger the child the greater the danger. Even when the attack is discrete in character, almost all under one year die, and a large proportion of those under two years. In such, even when convalescence seems to have set in, a sudden change may occur about the fourteenth or fifteenth day, and death ensue. Above the third year the discrete variety generally terminates favorably, but the confluent is very fatal in children of all ages. The severity of the initial symptoms bears no absolute relation to the course afterwards, but the child's previous health has a very important bearing. Any enfeebling disease, such as scrofula, phthisis, or syphilis, renders the prognosis bad. The amount of eruption governs the prognosis to a great degree, as also the extent to which the mucous membranes are implicated. During the development of the pock, any cessation or irregularity in its course is to be dreaded. Any sudden fading of the eruption or unusual pallor of the skin, any failure to become full and swell out about the eighth day, or any sudden shrinking of the pock, as if by absorption of its contents, is of the gravest import, and is generally followed by death, frequently within twenty-four hours. On the other hand, a good defervescence on the appearance of the eruption, a bright and rosy areola, with a moderate eruption filling out well about the eighth day, a fair return of the appetite, and a moderate secondary fever with no complications, are all of favorable import. In the hemorrhagic forms of the disease the prognosis is always very bad. A few cases in which hemorrhagic symptoms set in during the pustular stage may recover, but in general death is certain. "Laryngitis, if severe enough to give rise to distinct difficulty of breathing, is mostly fatal, even if tracheotomy be performed" (Collie). Complications should be looked for if the secondary fever run high. Different epidemics vary much in their mortality. Those occurring in summer are generally more dangerous than those occurring in winter (Curschmann), and the mortality is usually less at the end of an epidemic than at its commencement. Varioloid is rarely fatal, and has no complications.

Diagnosis.—It is very important for all concerned that a true diagnosis should be made in every case of variola and varioloid as promptly as possible. An error either way exposes the physician to merited blame, which, in general, his patients will not be slow to bestow upon him. Where there is any suspicion that a case is this dreaded disease, it is well to have definite knowledge on the following points. Are there other cases of variola in the neighborhood? Has the child marks of successful vaccination? If so, how many distinct marks, and how long a time has elapsed since they were made? If not, has the child been inoculated, or has it had a previous attack of small-pox? Has there been any possibility of exposure? Although it is very improbable that an attack of varioloid should occur in an efficiently vaccinated child under ten years of age, yet it is by no means impossible, as many instances during our Montreal epidemic attested. On the other hand, it is possible for a child to have a very mild attack of variola without previous vaccination. During the initial stage it is impossible to make an absolute diagnosis, but in the absence of effectual vaccination, and with the possibility of previous exposure, we should regard with suspicion the symptoms of this stage appearing without other sufficient cause. While it is remembered that vomiting with pyrexia may usher in many of the ailments of childhood, in the present disease the vomiting is more apt to be persistent. Pain in the back is a symptom which it is difficult to elicit or to estimate the severity of in young children, but should the child lose control of its sphincters small-pox may be suspected. In cases of repeated convulsions with intervening somnolence or stupor, occurring in very young children, the possibility of small-pox as the cause should not be overlooked. Except in cases of known exposure, a physician is, however, not justified in speaking absolutely until the characteristic eruption fully appears in the form of small, distinct, "shotty" papules, seen first on the face and forehead, and perhaps on the back of the wrists, and successively invading the neck, trunk, arms, and lower extremities, and visible also on the mucous membrane of the mouth and fauces. Should there be any irregularity in the appearance, or doubt about the symptoms, the proper course is to wait another twenty-four hours, until the papules on the face become vesicular. At this time a diagnosis ought to be made with certainty. Before this only one symptom is said to be pathognomonic, the hemorrhagic initial exanthem in Simon's crural triangle; but this appears only in the few.

Morbilli in children is probably the disease most likely to be confounded with small-pox, especially when the former is of the papular variety. In some cases it is impossible to distinguish between the two rashes for the first day. The points of difference are as follows. In measles during the invasion-stage we have catarrhal symptoms appearing early in the conjunctival, nasal, and bronchial mucous membranes. These are absent in small-pox. The rash of measles appears on the fourth day almost simultaneously on the back and face, and the papules are larger, smoother, softer, and brighter red, as opposed to the paler papules of variola, which communicate a more harsh and indurated, or shotty, sensation to the finger. "In cases of small-pox severe enough to simulate measles, on passing the hand over the face the feeling is that of hardness and furrowed roughness, like that produced by passing the hand over a piece of corduroy, whereas in raised confluent measles the sensation is like that produced by passing the hand over a piece of velvet" (Collie). The range of temperature during the initial stage of measles is lower than in variola, but there is no defervescence on the appearance of the exanthem; on the contrary, the temperature may continue to rise, and it attains its maximum during the height of the eruption. Variola and varioloid are to be distinguished from *varicella* by the stage of invasion lasting two or three days. In varicella this is generally absent, and very rarely does it exceed twenty-four hours,—any constitutional disturbance met with usually following, not preceding, the rash. The vesicle, in varicella, follows the papule within a few hours, never exceeding twenty-four, and attains its full development in one or two days. It is soft, globular, and superficial, rarely umbilicated. Its contents may be evacuated by a single puncture. The eruption is generally best seen on the back of the shoulders. Small isolated papules, which do not form vesicles, are probably due to varioloid. In *scarlet fever* the rash appears within the first twenty-four hours, and the throat is invariably implicated. The appearance of a papular rash on the third day would remove all doubt. Only in hemorrhagic small-pox might the diagnosis be obscure. Sometimes the symptoms during the invasion-stage may resemble those of *pneumonia;* but the appearance of the rash would, of course, put that diagnosis aside.

Papular eczema may be mistaken for the commencing eruption of small-pox, but it is very rarely that there are any preceding febrile symptoms, and if so they are of short duration. The red itchy character of the papules, the irregular distribution, the non-implication of the mucous membranes, should serve to distinguish it; but in any case of doubt the assistance of time should be sought. After forty-eight hours the variolous papule becomes distinctly vesicular. *Sudamina, pemphigus, herpes, ery-*

thema, *urticaria*, and *acne* have been mistaken for variola, owing to hasty and ill-considered diagnoses. If attention be paid to the element of time, the anomalous forms of small-pox need give no difficulty. "A scarlatinoid rash followed on the third day by an eruption of papules is small-pox. If such rash be of dark color, with purpuric and inky spots, the case is small-pox. Speaking generally, hemorrhagic effusions into the skin, purpuric and black spots, hemorrhage into the conjunctiva, and scarlatinoid measly rashes, preceded by initial symptoms, are small-pox" (Collie).

Treatment.—In any case of sickness in children when the sudden onset and general character of the symptoms would indicate specific disease, it is of importance that isolation in a large well-ventilated room in the upper part of the house should take place at once. As soon as the physician has determined the possibility of the attack being one of small-pox, arrangements should be made to insure the strictest isolation, and, at the same time, as perfect hygienic surroundings and as careful nursing as can be obtained. The room must be large, free of all unnecessary furniture, and possess means for thorough ventilation and free access of air. Its temperature should be kept moderate, about 66° F. Strong light should be excluded, lest it increase the tendency to pitting on the face and hands. The diet should be light, yet eminently nutritious. In some instances it would be the better of being partially pre-digested, or of having digestants, such as some good preparation of pepsin or pancreatin, associated with it. During the invasion-stage there is almost always complete anorexia, and digestion and assimilation are in abeyance. Only the blandest fluids should be permitted at this period; but during the eruptive stage feeding must be pressed, especially if the case be severe. Much tact and assiduous coaxing may be required to induce the little one to attempt to swallow, but it is important that as much nourishment as possible should be taken. In general it will be best to give it in small quantities at short intervals day and night. During the stage of suppuration, with its severe drain on the system, and its tendency to exhaustion, stimulants will be required in addition to the most nutritious and assimilable food we can give. In regard to its therapeutic management, it should be remembered that, with our present knowledge, we have no drug that will control or modify the course of variola. It is a self-limited disease, and, as Rilliet and Barthez long ago pointed out, all treatment tending to disturb the normal course of the illness is harmful. Sydenham's repressive treatment, designed to retard the development of the eruption, by keeping the skin cool and using saline laxatives, and the course of treatment adopted before his time, of encouraging derivation to the skin by promoting perspiration in every way, have both been found injurious. The disease runs a more favorable course when the eruption appears and develops naturally and regularly. Many antiseptic and antizymotic remedies have been tried, chief among which are quinine, salicylic acid, carbolic acid, and the sulpho-carbolates, but so far the results have not been in any way encouraging. It is well, therefore, to recognize fully at the outset that our therapeutics, at least for the present, should be confined to sustaining and palliative measures, and to the combating of any complications that may arise. Depressing measures of all kinds should be avoided in children, and especially in infants. During the stage of invasion severe nervous symptoms may call for treatment. Chloral hydrate, associated with one of the bromide salts, has been used with good results. Antipyrin or antifebrin in cautious doses may be tried. At present they promise to be of much service, both in reducing the hyperpyrexia of this period and in relieving many of the nervous symptoms. If the temperature run high and the head be hot, cold applications may be made to the scalp, and the body gently sponged with tepid water. For the vomiting I have found small doses of cocaine afford much relief. Should it fail, other gastric sedatives, such as soda, bismuth, and hydrocyanic acid, may be tried. In cases of older childen one of the effervescing citrates may prove very grateful. Should the bowels be confined at this time, a gentle laxative may be given. During the entire period of the eruption one important indication seems to be to relieve the irritation of the skin and mucous membrane. Very numerous are the different applications that have been made use of at one time or another in the hope of limiting the surrounding inflammation and lessening the tendency to subsequent pitting. It seems very doubtful if any of them are of great value. Still, it is in most cases desirable that some attempt to prevent the scarring should be made, especially on the faces of young girls. Perhaps the most successful method is that recommended by the Germans, of keeping cloths wet with water spread over the face and arms. Curschmann advises that the water be cool, but Kaposi recommends that it be used as warm as may be comfortable. If desired, some antiseptic or deodorizer may be added to the water. Hyde uses a solution containing one drachm of boracic acid with a drachm or two of glycerin to a pint of water as warm as may be comfortably borne. Cloths wrung out of this should be constantly applied, changing them as they cool. During the night-time, or when the patient is sleeping, they may be covered with oiled silk to retain the heat and moisture. If the eruption be very profuse over the body, and the irritation very great, the Vienna plan, of immersion for two or three hours daily, may be tried with older children. The water should be maintained as nearly as possible at a temperature of 98° F. Dr. Welch recommends a mixture of olive oil and lime-water in equal parts, to be from time to time painted over the surface with a large camel's-hair brush. Dr. Tomkyns writes me that he has used with much success in the Fever Hospital, Manchester, England, a thin solution of common starch, glycerin, and tincture of iodine (glyc., ℥ss; tinct. iodini, ʒii; sol. amyli, Oss) to relieve the dermatitis and prevent pitting. During the epidemic in Montreal in 1885, at the Civic Hospital tincture of iodine was painted once or twice daily over the face and hands while the eruption was in the papular stage. It was thought to diminish the pitting. A solution of nitrate of silver has been used similarly on the first appearance of the papules on the face. Schwimmer strongly recommends the use of the following paste: carbolic acid, 4–10 parts; olive oil, 40 parts; prepared chalk, 60 parts. Make a soft paste to be spread on soft linen, and with this cover the face and arms. The linen should be changed every twelve hours. To diminish the intolerable itching and fetor of the later stages, and to lessen the contagion, the body should be sponged once or twice daily with tepid water to which a small quantity of permanganate of potash has been added, and afterwards anointed with a weak carbolated oil. To relieve the irritation of the mucous membranes, sprays and gargles of chlorate of potash should frequently be used. They may be either warm or cool, as may be most grateful. Sometimes a little biborate of soda may be advantageously added to this solution. Mucilaginous drinks may be allowed freely, and several times a day the nurse should gently cleanse the mouth and fauces with a small swab of absorbent cotton soaked in the borax solution. The mouth and entrance to the nares should be kept carefully cleansed from mucus, and the nostrils should be sprayed at least twice daily with an alkaline spray containing a small amount of carbolic acid. During the stage of decrustation warm baths should be given daily. Should the scabs be extensive, it is important that pus be not allowed to collect in quantity underneath them. On the forehead and face means should be taken to get rid of them as quickly as possible. On the scalp the crusts are apt to persist, and numerous poultices may be required to remove them. Should suppuration continue, it may be checked by the application of carbolated zinc ointment. In children the itching at this time is often intolerable and may call for some gentle form of restraint for their hands. As the pustule approaches maturation, the physician must be on the watch for any symptoms indicating failure of development. Such would require prompt stimulation. Should laryngitis threaten, inhalations of medicated steam should be commenced at once, and poultices be applied externally. Should dyspnœa set in, an emetic, if the child be strong enough, may first be tried. If it fail, resort must be had to tracheotomy or intubation. Severe insomnia or delirium may require the cautious use of bromides, opium, or chloral. Throughout the whole course of the illness the strength of the patient must be conserved. Complications must be treated on general principles, avoiding anything approaching systemic depletion, and interfering surgically when it is apparent that such interference is necessary. During the stage of convalescence iron tonics are generally required, and one of the best is the muriated tincture of iron. It is rendered less irritating to the stomach, and more palatable, by being combined with a considerable amount of glycerin. For the severe hemorrhagic forms of the disease no treatment has been found of any avail. In the milder forms, stimulants with tonics offer us the best hope. My own predilections are strongly in favor of the use of iron in large quantities.

In this disease, however, our duty is not finished with the mere treatment of our patient. We have to consider other members of the household and the public generally. When small-pox enters a house, vaccination and re-vaccination must be promptly and effectually performed. In such cases, as in every case where known exposure has taken place, the duty of the physician is to give as prompt protection as possible. We believe that this can best be done by using fresh humanized lymph, which produces a more rapid and certain development of the vesicle than does bovine lymph, and therefore more quickly secures protection. The patient may be considered free from infection when every particle of scab and crust is removed and he has received several thorough subsequent baths. The most complete disinfection of everything that has come into contact with him or that has been in his room must be insisted upon, and should be performed under public authority and supervision.

VACCINATION.

By W. T. PLANT, M.D.

Definition.—The conveyance of cow-pox or vaccinia from the animal to man or from one person to another for the purpose of protecting the system against small-pox.

History.—No chapter of medical history is more interesting than that of the discovery of vaccination, the most beneficent result the world has known of human thought and effort.

For several centuries small-pox had been, more than any other pestilence, or even war, the foe of man. It had spread to all climes and over all countries, carrying off, on an average, fully one-sixth of all those attacked by it, and inflicting incurable blindness or deafness and frightful disfigurement upon great numbers of the survivors. Nothing could exceed its fury when it visited a country for the first time. Its introduction into Mexico in 1520 was followed by the loss of three millions and a half of people. In 1707 it made its first entry into Iceland, and more than one-fourth of the whole population fell victims to it; crossing into Greenland a quarter of a century later, that island was speedily almost depopulated.[1] Society was everywhere largely made up of two classes, those who were pock-marked or otherwise mutilated and those who were still liable to become so.

Small-pox was especially fatal among the young. In the larger cities of Great Britain about one-third of the deaths under fourteen years were due to it, and the mortality under five years was about fifty per cent.

Such was the forlorn condition over the known world when in 1716 the Hon. Edward Wortley Montagu received appointment as the English ambassador to the Ottoman court. The letters of his accomplished wife, Lady Mary, who accompanied him to Constantinople, were so instructive and entertaining as to gain for her a lasting celebrity. In one of these, written from Belgrade in 1717, she describes in sprightly terms the Turkish custom of "engrafting" small-pox, and declares her intention of trying it on her young son.

Returning to England, she seriously undertook the introduction of the art there, and evinced her faith in its safety and virtue by having her little daughter inoculated with small-pox matter. This was in April, 1721. The expediency of the operation being questioned by the scientists of the day, government extended remission of sentence to six prisoners condemned to death, on condition that they would submit to inoculation. The experiment was successful, and the pardons were cheaply bought. After this the operation was taken into royal favor, and two daughters of the Princess of Wales in 1722 received variolous infection after the Turkish method.

---

[1] Chambers's Encyclopædia.

The same year that it was introduced into England—1721—it began to be practised in Boston, where an epidemic of great virulence was raging. But of two hundred and forty-four persons inoculated by Dr. Boylston within six months, six died. About the same time there were in England two conspicuous examples of death from inoculated small-pox.[1]

These deaths served, both here and in Europe, to prevent the new practice from being received with that instant favor which it otherwise would have enjoyed. It was near the middle of the century before it met with anything like general acceptance. In 1746 the Small-Pox and Inoculation Hospital was founded in London, in order that the poor as well as the rich might share in the benefits of the operation.

In Great Britain and America inoculation or the "engrafting" of small-pox may be said to have had a reign of about fifty years,—the latter half of the eighteenth century. People then made appointments with the small-pox physicians as we now do with dentists. During our war for independence the wife of General Washington improved the occasion of a brief sojourn in Philadelphia by undergoing inoculation, and she had, according to history, "a very favorable time."[2]

The efficacy of this operation in mitigating the severity and danger of small-pox was certainly very great. The proportion of deaths following it was, on an average, about three in a thousand,—a very gratifying contrast to the mortality of the disease communicated in the usual way. But there was one fatal drawback. However light the engrafted disease might be, it was still small-pox; and the more it was conveyed in this way, the more were centres of infection multiplied from which those not protected were liable to contract the disease in its worst form. To individuals inoculation was a great blessing; to society at large it was a great curse. In the early part of the eighteenth century, before inoculation, about one-fourteenth of the deaths in Great Britain were from small-pox; in the latter part, after inoculation had become quite general, about one-tenth of the deaths were from that disorder.[3]

But no sooner had variolous inoculation reached a position where it could claim for itself in this country and Europe something like general recognition and practice, than one was born who was to introduce a new method, entirely devoid of danger to the individual and free from the fatal defect of spreading small-pox.

---

[1] Rees's Encyclopædia.

[2] Irving's Life of Washington.

[3] Seaton, Hand-Book of Vaccination.

Edward Jenner, the son of an English clergyman, was born in 1749 at Berkeley, in the county of Gloucester,—a county celebrated then, as now, for its dairies. Having early fixed upon medicine for a vocation, he joined himself to a surgeon in a neighboring village for instruction. About this time the confident assertion of a young countrywoman that, having had cow-pox, she was proof against small-pox, made an impression on his mind that was lasting. In his twenty-first year he went to London to finish his pupilage under the great John Hunter. After two years in the metropolis, he went back to his native village of Berkeley and began practice. On renewing his acquaintance with the dairy-people, their belief in an antagonism between cow-pox and small-pox was again brought to his notice. He began to look into the matter. By degrees, as evidence accumulated, he became convinced that there was something in it. He conceived that a disorder that could be conveyed from the cow to the human system by rare chance in milking might be more surely communicated by methodical operation, that it might also be carried from person to person without losing its protective power, and that in this way, the whole human family being made secure against small-pox, that loathsome disease would be driven out of the world from sheer lack of living accommodations. After this manner we find Jenner talking to his friends in 1780.

From the scarcity of true cow-pox, he was not yet able to bring these magnificent and inspiring thoughts to the touchstone of actual experiment. It was not until May 14, 1796, more than a quarter of a century after his thoughts were first turned that way, that Jenner made his first vaccination. The subject was a lad of eight years, named James Phipps. Several weeks afterwards the boy was inoculated with small-pox matter, and, as Jenner had predicted, no result followed. Two years passed away before he was able to repeat the experiment, owing to the disappearance of cow-pox from the dairies.[1] Having at length made himself certain of the truth and importance of his discovery, he published a pamphlet entitled "An Inquiry into the Causes and Effects of the Variolæ Vaccinæ," and sent it out to the profession. At first considerable incredulity was manifested, but it could not last, for the proofs were at hand and more were forthcoming. Within a year from the first public announcement of the discovery, seventy of the most distinguished physicians of London signed a declaration of their entire confidence in it.

With so much impetus gained, the spread of vaccination was marvellously rapid. In 1800 it began to be practised in this country, and a year later in France. In 1803 the court of Spain sent out an expedition for the purpose of carrying vaccination to all the Spanish possessions of the Old and the New World. It returned after three years, having made the circuit of the globe.[1] Within six years after it was first given to the public, the knowledge and practice of this beneficent operation had spread over all the world. All nations hailed it with demonstrations of joy and gladness. In Russia, the Empress gave the first child vaccinated the name of "Vaccinoff" and made its education a public charge.

---

[1] Encyclopædia Britannica.

In the universal thankfulness for this unparalleled blessing, he through whom, under Providence, it had been secured was not forgotten. Honors were conferred on Jenner by foreign courts, and he was voted honorary membership in many learned societies. The anniversary of his birth and that of his first vaccination were for many years celebrated in Germany as feast-days. In 1802 Parliament voted him fifty thousand dollars, and five years later a hundred thousand more.[2]

With the modesty that ever characterizes true greatness, Jenner continued to reside among his life-long friends in and around the little village of Berkeley. He did, it is true, soon after he became famous, try London life and practice for a time, but he soon tired of them and returned to his more congenial country home. Here he passed the evening of his life in the practice of his profession, in correspondence relating to his great discovery, and in the peaceful enjoyment of the society of his friends. He died of apoplexy in 1823, and his remains were laid away in the parish church. Of all the benefactors of mankind, not one has ever lived to see the world so abundantly blessed through his labors as Edward Jenner.

Nature of Vaccinia.—What is cow-pox, and whence does it derive its royal power over small-pox? Jenner regarded the two diseases as essentially the same. In 1801, only three years after the great discovery was promulgated, Gassner, of Günsberg, inoculated cows with small-pox matter. Lymph from the resulting vesicles was conveyed to four children, and the ordinary phenomena of vaccination followed.[3] Later than that,—1830,—Dr. Sonderland, of Barmen, in Prussia, blanketed some cows with the bed-clothing of a deceased small-pox patient. Other coverings from the same bed he suspended around their heads. In a few days the animals became sick and feverish, and an eruption like that of genuine cow-pox appeared. Lymph from these vesicles produced genuine vaccine in the human subject.[4] These experiments in their essential features have been many times repeated in this country as well as in Europe, with like result. Vaccination, then, is the conveyance of small-pox, but of a small-pox wonderfully modified and stripped of its terrors by passing through the animal. In some way it has parted with its contagious property, so that vaccinated small-pox is not constantly spreading the disease, as was the case with inoculated small-pox. The horse as well as the cow, though perhaps less readily, may be made the subject of variolous infection. Jenner used lymph from the horse quite largely, but since his day the cow has been almost the only source of original supply, so that the term vaccination, from *vacca*, "a cow," is entirely appropriate.

---

[1] Encyclopædia Britannica.

[2] Ibid.

[3] Seaton's Hand-Book of Vaccination.

[4] Eberle on Children.

Kinds of Lymph.—The material for vaccinating is now taken from both the heifer and the child: hence we speak of bovine and human virus. Until recently the latter was in almost exclusive use. Previous to 1870 bovine lymph was seldom procurable, because epidemics of cow-pox were of infrequent occurrence. But about that time, responding to a clamorous popular demand, its production on a large scale by vaccinating young cattle began to be followed as a business. There are now in this country quite a number of establishments for the propagation of kine-pox. Any locality with telegraph and railroad communication can now be supplied with little delay with recent bovine virus. Within a few years the use of human lymph has greatly lessened. But, notwithstanding the extended and still-spreading popularity of bovine virus, there are very many in the profession who believe that failures with it are much more frequent than with the humanized, and that it is less regular and reliable and often more severe in its working,—a view that my own observations would tend to sustain. A certain lack of affinity between lymph from the cow and the human system is certainly quite possible.

Conveyance of other Diseases.—An asserted advantage of animal matter over human is immunity from the liability of conveying other diseases. This is a moving consideration with multitudes among the laity, who believe that all sorts of eruptions and blood-disorders are chargeable to human lymph.

It is doubtless true that a vaccination may be followed by an erythematous, an erysipelatous, or an eczematous eruption caused by the local and constitutional irritation consequent on the operation; but I can find no reliable evidence to warrant the opinion that chronic enlargement of glands, or scrofula, or consumption, or any of the ordinary skin-eruptions, have ever been transmitted from person to person through vaccination.

But how is it with syphilis? That is an inoculable disease: except when inherited, it is had only through inoculation. May it be conveyed with vaccinia? There can be no doubt of it. So many cases have now been recorded in which cow-pox and syphilis have been simultaneously imparted by vaccination, that they cannot be explained away, and vaccino-syphilis must be accepted as a fact. It was thought by many that in all such instances there was an admixture of blood with the lymph, and that care to exclude that would obviate danger. But one of the experiments of Dr. Robert Cory, chief vaccinator to the National Vaccine Establishment, England, would seem, even if there were no other proof, to decide the question. To test this point,—the possibility of conveying syphilis with clear lymph,—that gentleman, with a scientific devotion which we admire but could never imitate, carried out a series of experiments on his own person. He repeatedly vaccinated himself from children who were plainly and some of them actively syphilitic, using with greatest care only the clear lymph. One of these trials, made July 6, 1881, was not, as all the previous ones had been, barren of result. On this occasion he vaccinated himself in three places from a three-months-old child that had an eruption and sores which were evidently syphilitic. In three weeks syphilitic papules appeared at the seat of two of the punctures. These were followed in due course by sore throat, roseola, and other indubitable evidences of constitutional syphilis.[1] From this it would seem to be proved that clear, limpid lymph *may* be a medium of carrying that disease from one person to another. But, conceding this, it is to be remarked that this child was selected for this experiment because it had syphilis in an aggravated form, and that no medical man would ever think of taking lymph from such a source. There is no reason in all this for the rejection of all human lymph, but rather cause for careful scrutiny of the antecedents and present condition of every proposed vaccinifer.

Degeneration of Lymph.—It has been claimed that lymph gradually parts with its protective virtue by passing successively through the human system, and that if we would make the protection against small-pox complete we must use the bovine product altogether or return to it at short intervals.

In 1816, Jenner wrote that the vesicles he was then producing were in every respect as perfect as in the first year of vaccination, though, to the best of his knowledge, the matter from which they were derived was taken from the cow about sixteen years before. Prof. Hebra asserts that in the principal vaccine establishment at Vienna lymph has been carried down without interruption from the first vaccination at the beginning of the century, and that it now "takes" as well and is as protective as at first.[1] Dr. Charles V. Chapin, writing from the office of the Superintendent of Health, Providence, Rhode Island, December, 1885, says that they were then using stock that had been maintained by continuous transmission from child to child since 1856, without being once renewed from the cow; that nearly forty-seven thousand persons had been vaccinated from it; and that it had shown itself not inferior to the bovine product in protective power, besides being more certain to take and less liable to produce troublesome sores.[3] We believe that lymph may degenerate, not because of its transmission through the human body, however many times the transmission may be made, but through lack of care to choose stock from healthy and vigorous subjects. Let a farmer gather seed-corn from stunted ears growing on poor soil, and he will soon be raising degenerated crops. This is a general fact of vegetable and animal propagation, to which vaccination is no exception.

---

[1] Boston Med. and Surg. Jour., vol. cxi. p. 188.

[2] Manual of Skin Diseases.

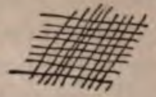
[3] Annual Report Supt. of Health, Providence, Rhode Island, 1886.

Forms of Lymph.—The lymph used in vaccinating, whether human or bovine, is either fluid or dry. As a fluid it is conveyed by the lancet directly from the vesicle to another person, as in "arm-to-arm" vaccination. When numbers are to be operated on at once, as in schools and factories, this is the surest and most convenient way. Fluid lymph may also be taken up from the vesicles in capillary tubes, which, after closure of the ends, are put aside for future use.

The use of dried lymph is, in this country at least, much more common. At the proper time the vesicle is punctured and the exuding fluid collected on ivory or quill points, and permitted to dry; or the vesicle may be left to mature. In the latter case the lymph dries down into a dark-brown crust, that falls from the arm about three weeks after vaccination. The bovine crust is notoriously unreliable, and is not very much used.

Selection and Preservation.—Vaccine lymph is a perishable substance, soon losing its virtue if exposed to light, air, warmth, or moisture. Under opposite conditions it may be kept indefinitely. It is not injured by any degree of cold. The quills and ivory points now supplied by various establishments for propagating bovine virus are sometimes in boxes, sometimes in small bundles wrapped around with rubber cloth. To give satisfaction they must be used without much delay. The same is true of human lymph put up in the same way. When it is desired to preserve the stock for some time, I think the human crust is preferable. In selecting lymph our first thought is of purity. We usually take it from children, as they are not likely to have acquired blood-disorders. Unless we have full knowledge of the family, it is best not to vaccinate from a child of less than three months, because it might have latent syphilis. The child that is to serve as a vaccinifer should have favoring antecedents, should be in perfect health and wholly free from skin-eruptions, and should have a *perfect* vesicle. If the crust is taken, it should be freed with the utmost care from all extraneous matters by paring away the thin edge and scraping the under surface. A good way to preserve the crust for future use is to divide it into three or four pieces and to wrap these separately in a bit of thin rubber cloth. Drop these into a very small vial, cover them with absorbent cotton, and cork tightly. Enclose this bottle in a larger one, cover with cotton, and cork tightly. Label with name, age, and date. Then wrap the whole in rubber cloth or other dark, impervious material, and put in a cellar or an ice-chest,—preferably the latter. So treated, the crust may be kept for many months, ready for use at any time.

Operation.—The only instrument needed is a lancet. This should be perfectly clean. It is a good rule to plunge in hot water and wipe dry just before using. The outer aspect of the left arm at or near the insertion of the deltoid muscle is, by almost universal custom, the place chosen. The chief ways of preparing the arm now in vogue are by abrasion and scarification or cross-scratching. In the first the cuticle is scraped by the lancet over a surface of a quarter to a half inch square until a little serum appears.

This prolonged scraping, while not painful, is not agreeable. In the second, linear incisions are made from half a line to a line apart, and these are crossed by others, after this manner:  This plan was, I believe, first proposed by Dr. D. Francis Condie, of Philadelphia, not far from 1830.[1] It appears to me to be the best yet devised, not only because it presents so many lines of absorbing surface that it is very certain, but also because it is scarcely at all painful or unpleasant. I have repeatedly, after this way, vaccinated sleeping infants without their waking. The incisions should be quite superficial. The little blood that follows may be wiped off, or absorbed by a blotter. It is well to prepare two or three of these places, an inch or more apart. This being done, the arm is ready for the application. If the quill or ivory point is to be used, first moisten the lymph-covered surface with a drop of clean, cold water. If bovine matter is being used, remember that it is less soluble than the other kind, and it is well, with lancet or with penknife, to rub it up with the water. Now apply the moistened quill or ivory to the abraded or scratched areas, and rub and press well in, to insure absorption.

In using the dried crust, only a diminutive piece is needed for one vaccination. This is to be mashed down with the knife-blade, or, better, between two small squares of glass. Add a drop of water and rub into a paste. This is to be taken up by the lancet and applied in the same way as the quill or ivory. The secrets of successful vaccination are care in the selection and preservation of lymph and cleanliness in every detail.

Phenomena.—After the operation, there is a period of latency of about three days' duration, sometimes a little less, frequently rather more, and in rare instances as much as a week. This brief incubative period of vaccinia is a valuable characteristic, as it enables us to forestall small-pox after known exposure.

On or about the third day a small, hard pimple appears at the point of operation. With two days' growth it becomes a vesicle. In another day it has become umbilicated and divided into eight or ten cells, like the small-pox vesicle. By the eighth day, or ninth at most, the vesicle has attained complete development. It is raised prominently above the surface, and is distended with transparent fluid. At this time, or earlier, but by no means later, lymph may be taken for use. The next phenomenon is the appearance of a belt of inflammatory redness around the vesicle,—or pustule, as it has now become. This is the areola, and is quite characteristic of vaccinal activity. With a day or two of growth the areola attains a diameter of two to three inches. It is now of a bright-red or livid color, and the flesh under it is commonly hard, itchy, and painful. With the areola, some constitutional symptoms, as headache, rigors, fever, and general aching, appear. The whole arm is apt to be lame and the axillary glands swollen.

After the tenth day there is rapid decline. The constitutional symptoms abate; local pain and itching subside; the areola fades away; the fluid of the vesicle becomes opaque and concrete and soon dries down into a dark crust. About the twenty-first day the crust falls, leaving a circular, depressed scar with pits and radiating lines that correspond with the cells and partitions of the former vesicle. The scar, red at first, gradually becomes white, and commonly remains through life.

---

[1] Condie on Diseases of Children, 5th ed., p. 544.

Deviations from Regular Course.—These are not very frequent. The phenomena are sometimes retarded, especially if bovine lymph is used, but the protection is not less complete, provided the symptoms succeed one another in due course with characteristic vesicle and areola. Physicians have sometimes been accused of using impure stock because the operation has been followed by an eczema or other eruption or by enlarged lymphatic glands. If we consider how often the irritation of coming teeth causes like symptoms, we shall not be surprised that the "working" of vaccinia should now and then put out these signals of systemic perturbation.

Doubtless diseases and tendencies that have been hitherto latent may be stirred into activity by this operation. If this is true, may it not be supposed that syphilis was present in some of the instances reported as syphilitic inoculation, but was unsuspected until the constitutional commotion brought about by vaccination spurred it into activity?

Traumatic erysipelas may follow vaccination. I was once called to prescribe for a frugal woman who had performed auto-vaccination, using liberally of the crust that had been shed by her son, without scraping or paring. Her whole arm was erysipelatous; large abscesses formed, and permanent disfigurement resulted. Fatal pyæmia has been known, it is said, after vaccination. Doubtless it is owing to the care that physicians, as a rule, exercise, that these disorders so seldom occur.

Degree of Protection.—To what extent does a single vaccination insure against small-pox? Certainly with the majority the security is complete and lasting. Comparatively few of those who can show a characteristic scar contract variola if exposed to it. But there are some for whom the operation is but a partial safeguard,—some in whom there is liability to a mitigated small-pox. It is probable that in some of these the original vaccinal impression on the system was not all that it might have been, and that a second operation done soon after the first had run its course might still have produced the characteristic "working," and so might have conferred a more nearly complete security.

It is certain that in many persons the protection afforded by a vaccination becomes progressively less with lapse of time. Especially is this true of some who, vaccinated in infancy, have reached mature life. There can be no doubt that the vaccinal influence diminishes with the constitutional changes that take place at puberty.

But it is to be remembered that, while this operation does not in all cases confer complete immunity, neither does small-pox itself do it. In numerous instances persons have had that disease twice, and a few even more than twice. Abundant observation has proved the justness of Jenner's claim for vaccination made in these words: "Duly and efficiently performed, it will protect the constitution from subsequent attacks of small-pox as much as that disease itself will. I never expected it would do more; and it will not, I believe, do less."[1]

In a word, then, this is the protection afforded by thorough vaccination: complete and lasting to the vast majority; to a small minority partial; to all, with scarcely an exception, security against great suffering, extreme disfigurement, and death.

Susceptibility Universal.—Entire insusceptibility to the infection of cow-pox is very infrequent, if indeed it ever occurs. Often, to be sure, a child is vaccinated two, three, or more times without result; but I am of the opinion that the failure is generally due to the lymph or the operator rather than to the subject. Of upwards of nine thousand operations done at the Blackfriars Station of the National Vaccine Establishment since 1859, there was but *one* case which on a second trial was unsuccessful.[2] Doubtless there are times in the lives of many, perhaps of most, when the operation succeeds less readily than at others; probably some have seasons of complete insusceptibility, as *may* be the case when other serious diseases are present in the system; but that any of the race are through life wholly insensible to the influence of vaccinia there is good reason to doubt. Skilled operators with fresh lymph, especially, I think, with humanized lymph, have seldom to report failures in primary vaccinations.

Age and Season.—Evidently, if immunity from so terrible a disease as small-pox can be conferred by means so simple and harmless, the blessing should be extended to early infancy. All authorities recommend, and the laws of many countries require, the operation to be done before the end of the first year. In England the Vaccination Act of 1867 requires the operation to be done "within three months of birth, or as soon afterwards as the public arrangements of the district in which the family lives will afford opportunity of obtaining gratuitous vaccination."[3] I believe the best time is between the first and fourth months. It is then well over before dentition begins. If the child is sickly or has any skin-eruption, it should be got into good condition beforehand.

If, however, small-pox is prevailing, there is no age, however early, and no degree of poorliness, that should bar out the operation. In imminence of danger, as when variola is in the same street or house, I would vaccinate an infant immediately on its birth. Experience has shown these early operations to be as safe and protective as later ones.

Books have something to say about the best time of year for vaccinating, but I think it matters little. In winter and in summer the course of vaccinia is the same: the important thing is not to neglect or defer it when it ought to be done.

---

[1] Barron's Life of Jenner, vol. ii. p. 135.

[2] Seaton, Hand-Book of Vaccination.

[3] Ibid.

Revaccination.—The liability to loss or diminution of protection with lapse of time creates a frequent necessity for revaccination. Since we know from statistics that small-pox after successful revaccination is an exceedingly rare event, public safety would be promoted by requiring all persons, without regard to the time of the primary operation, to renew it at or soon after puberty. Where it is not a requirement of law, this duty is too much neglected. There can be no harm, either,—on the contrary, there may be much good,—in revaccinating with every near approach of small-pox.

Doubtless in very many cases of vaccination repeated no result will be manifest. In a large proportion, however, the ordinary phenomena appear, but in an irregular way. The vesicle is apt to be small, acuminated, and without a central depression. It usually takes a swift course, reaching full growth by the sixth day instead of the eighth, and then rapidly declines. The crust is likely to be small and imperfect, and should *never* be used to vaccinate from, nor should lymph for this purpose ever be taken from a secondary vesicle.

VARICELLA.

BY CHARLES G. JENNINGS, M.D.

Definition.—An acute, specific, infectious disease, peculiar to infancy and childhood, characterized by a short febrile period, and a vesicular eruption distributed over the whole surface of the body. The vesicles appear in successive crops and disappear by desiccation in from three to five days, occasionally leaving permanent cicatrices.

History.—The recognition of varicella as a distinct disease dates from the latter part of the seventeenth century. A few clear descriptions of the disease written before this time leave no doubt as to its existence from a very early period. Rhazes (A.D. 910), the earliest writer on small-pox, imperfectly described it. Ingrassias (1550) and a contemporary, Vidus Vidius, also described the disease, the latter author under the term *crystalli*, a name often used to the present day. According to this writer, the Italians were familiar with the disease, and called it *ravaglione*, the name they still use. With these few exceptions, no mention is made of the disease by the early writers, and its history is inseparably connected with that of variola.

To Sydenham (1675) is due the honor of finally separating measles and variola; but this writer makes no mention of varicella as a distinct disease. A few years after Sydenham's publication, Morton and another English physician (1690) wrote of several cases of varicella under the title of *variola maxime benigna*. At this time the disease was popularly distinguished from variola and termed chicken-pox. Morton introduced this term into medical literature (Gregory). Gee and Fagge give the derivation of this word to be from *cicer*, "chick-pea," through the French *chiche*. The name varicella, first given to the disease by Vogel (1764), is a diminutive of *varus*, a "pimple." Almost all the various names given to the disease by different writers refer to its resemblance to variola.

These early writers observed that an attack of chicken-pox gave no protection against variola; and it was in all probability this fact that led many to separate it from that disease. With but an occasional exception, the physicians of this period doubted the distinct character of varicella and classed it with the mild forms of varioloid. Considering the close resemblance of the two diseases, and the fact that at this time medical knowledge was so cloudy that measles and small-pox, diseases that are now so easily distinguished from each other, were confounded, it is not remarkable that this confusion should have existed.

The introduction of inoculation for the prevention of small-pox in the early part of the seventeenth century drew the attention of observers to the mild forms of varioloid disease. The opposition to inoculation was strong, and those who opposed it brought forward all cases of varioloid and varicella to prove the inefficacy of the operation. It became necessary for the adherents of the practice of inoculation to establish the specific character of varicella; and this led to its very careful study.

Although in 1730 Fuller made what appears to be the earliest assertion of the non-identity of variola and varicella, urging in the plainest terms that chicken-pox could not be produced by the contagium of any other specific disease, Dr. Heberden[1] gave to the world the first full and accurate description of varicella. He fully discussed the pathological relations of the disease, and from his observations he came to the conclusion that varicella was a disease distinct from variola. Dr. Heberden was a physician of large experience and a careful and accurate observer, and his work was looked upon as a standard by the practitioners of his day.

In Germany, Dr. Franck, of Vienna (1805), and a few years later Dr. Heim, of Berlin, made important contributions to the literature of the disease. Writers of this period caused much confusion by the multiplication of names and by the frequent inclusion of cases of mild varioloid. Heim affirmed the specific nature and the inoculability of varicella, and called attention to the distinguishing characteristics of the cicatrices of variola and varicella.

In 1806, Willan wrote of varicella and gave particular attention to the minute description of the cutaneous lesion. According to their form, he termed the varicellous vesicles *lenticular*, *conoidal*, and *globate*. These distinctions are still occasionally to be found in medical works, but such multiplication of descriptive terms has become obsolete.

From the appearance of Heberden's essay until about the first part of the present century the opinion that varicella was a disease distinct from variola was quite uniform. The defenders of inoculation for the mitigation of variola were able to prove by abundant observation that the varioloid eruption that was so frequently seen after inoculation was the distinct disease varicella, thus removing doubts that were raised of the protective power of the operation.

In 1798, Jenner communicated to the world his great discovery of vaccination, and the practice of this preventive of variola quickly supplanted inoculation. With the general introduction of vaccination, and the resulting modification of the disease, came many cases of varioloid. The close resemblance between mild varioloid and varicella and the simultaneous prevalence of epidemics of the two diseases again brought doubts to the minds of physicians as to their distinct character. The epidemic that prevailed in Scotland in the years 1818 and 1819 was carefully observed and studied by Dr. Thompson, of Edinburgh. In his work[1] upon the subject he reopened the question of the specific character of varicella. His observations led him to believe that varicella was simply a mild manifestation of the variolous poison. Thompson's views of the pathology of the disease found a number of supporters, and to the present day there are a few eminent writers who hold to the same doctrine.

---

[1] Transactions of the Royal College of Physicians, vol. i. p. 427, 1767.

The most influential supporter of the theory of the etiological identity of variola and varicella in recent years was Hebra. As a great teacher of dermatology, his influence was wide-spread, and his ideas have had a controlling effect upon the minds of many of the physicians of Europe and America. Among very recent writers upon varicella, Kaposi and Bruyelle follow the theory of Hebra.

American literature on varicella is very meagre, but American physicians with but rarely an exception have given no credence to the theory of identity. To the writer's knowledge, the only author who has unqualifiedly upheld Thompson's views is Eberle.[2] He thought the evidence adduced by Dr. Thompson conclusively proved the common origin of the two diseases. Dr. J. Nevins Hyde[3] is inclined to consider the two diseases identical. His views are peculiar, and he formulates them thus: "Practically and clinically, it is useful to regard these disorders as of a distinct nature. The arguments, however, in favor of such absolute distinction are not irrefutable. There is probably in both forms of disease but a single virus, that of variola; but this, modified by evolution among generations of vaccinated children, has, in this process of natural cultivation or attenuation, produced a malady of tender years whose attacks do not protect from variola and occur irrespective of vaccination."

Etiology.—Varicella is a disease of infancy and childhood. Infants under the age of six months enjoy a certain immunity, but it is not so marked as in the case of scarlatina and measles. Congenital varicella has not been recorded. Senator observed a case in an infant eleven days old. In children over ten years of age the disease is rare, while in adult life it is so infrequent that many observers of large experience have not met with it. The writer has observed one case in a mulatto aged twenty-three years. The influence of age is well illustrated by five hundred and eighty-four cases reported by Baader,[4] of Basle:

CASES.	AGE.	CASES.	AGE.
382	1 to 5 years.	2	16 to 20 years.
191	6 to 10 "	2 (?)	20 to 40 "
7	11 to 15 "		

---

[1] Account of the Varioloid Epidemic in Scotland, 1820.

[2] A Treatise on the Diseases of Children, 1833, p. 419.

[3] System of Medicine, Pepper, 1885, vol. i. p. 481.

[4] Jahrbuch f. Kinderheilk., xvii. 104.

The immunity of older children and of adults has been ascribed to the heavier quality of the skin. The disease, however, is so wide-spread, and such liberty is allowed to those affected by it, that very few individuals pass through early childhood without contracting it. These exceptional individuals may enjoy a peculiar insusceptibility which persists through life.

As with the other exanthemata, varicella occurs, as a rule, but once in the lifetime of an individual. Sex has no influence.

Varicella occurs sporadically and in epidemics. In large communities it is always present. Thomas has observed that in large towns epidemics are not separated by intervals of several years, as is the case with measles and small-pox, but occur once every year or every half-year. In Leipsic epidemics occur regularly a short time after the opening of the infant-schools. Epidemics of varicella often precede or follow epidemics of other contagious diseases. There is no evidence that the disease is unusually prevalent during an epidemic of small-pox. Seasons and meteorological conditions do not influence the spread of the disease.

The medium of contagion is in all probability the respired air.

Many experiments have been made in the inoculation of varicella, but the successful cases have been very few, and it must be admitted that the disease can be inoculated only with great difficulty. That it can be transmitted at all by inoculation is denied by many writers. Boyce,[1] of Edinburgh, made many attempts at inoculation, but always failed. Bateman[2] states that it may be transmitted by inoculation, but does not give experimental evidence. Hesse[3] collected eighty-seven cases of inoculation. Of these, nine were followed by general disease and seventeen by local manifestation only. The result in the other cases was negative. Thomas obtained negative results in similar experiments, and states that Heim, Vetter, Czakert, and Fleischmann were equally unsuccessful. In this country Dr. J. Lewis Smith[4] endeavored to communicate the disease in this way, but failed. More recent experiments by Steiner[5] tend to confirm the inoculability of varicella. He inoculated ten children, in eight of whom varicella developed. The stage of incubation in the eight successful cases was eight days. Unless these favorable results fail to be confirmed by subsequent observation, or the experiments are proved to be faulty and the exposure to have taken place in some other way, it must be conceded that, although it often fails, the disease may be transmitted by inoculation.

In all probability the disease may be carried by a third person, although so little attention is paid to it that the origin of a particular case is rarely traced.

The nature of the specific virus of varicella is as yet unknown. Dr.

---

[1] Thompson on Varioloid Diseases, p. 74.

[2] On Cutaneous Diseases.

[3] Ueber Varicellen, Leipsic, 1829.

[4] Diseases of Children, 5th ed., p. 46.

[5] Wien. Med. Wochenschrift, No. 16, 1875.

A. Tschamer[1] claims to have cultivated the virus and to have obtained a hitherto unknown micrococcus. He states that the organism is distinct from the microbe that he obtained from the cultivation of variola and vaccinia, thus, according to him, proving the distinct character of varicella. He did not carry his experiments with varicella further than to isolate the microbe.

As stated above, there are a few writers at the present time, especially on the continent of Europe, who do not give to varicella a place as a distinct disease. Hebra applied the term varicella to cases of variola in which the rash is very scanty, and which run a favorable course and always terminate in recovery; and this is about the opinion of those who now hold to the doctrine of identity. Manifestly, such a classification will include under the term varicella not only all cases of this disease, but also many cases of mild or modified small-pox.

Those who believe in the non-identity of the two diseases exclude from varicella all cases, regardless of their clinical features, that originate from exposure to variola or are capable of transmitting variola to another individual by inoculation or otherwise; and, unless it can be shown that the disease which prevails almost continuously in our large communities regardless of the presence of small-pox, and which alone is entitled to the name varicella, can generate true variola, its claims to a distinct position among the specific diseases must be granted.

In favor of the non-identity of variola and varicella are the following arguments, modified from Gee:[2]

1. Varicella and variola are not interchangeable. There is not a single authentic instance where either of the two diseases was the result of exposure to the other. When the two diseases prevail together in a community, and there is free exposure to both, instances may arise that appear to negative this proposition, but, with the opportunities for error that such circumstances offer, they are of little value. In the United States varicella is one of the commonest of diseases, while small-pox, except in the large seaboard cities, is very rare. So rare is it that in many parts of the country practitioners of large experience pass many years without seeing a case. When variola appears in a community previously free from it, it bears no relation to the presence or absence of varicella, and it can, with but few exceptions, be traced to outside origin.

Chicken-pox often prevails as an epidemic isolated completely from cases of variola. Mohl first brought forward this fact. According to this writer, between the years 1809 and 1823 small-pox was entirely absent from Copenhagen, while cases of chicken-pox were met with every year. This is now the every-day experience of all American physicians in general practice. Now, an epidemic of varioloid free from concurrent cases of non-modified small-pox has never yet been seen, and it is highly improbable and opposed to medical experience that varicella, if it were but modified small-pox, should never give rise to a distinct case of variola. Furthermore, chicken-pox is inoculable with difficulty, and the few successful inoculations have invariably produced typical varicella. Inoculated small-pox, whether modified or not, has never yet been proved to have caused chicken-pox.

---

[1] Arch. f. Kinderheilkunde, B. ii. H. 3 (abst. in Amer. Jour. of Obstet., vol. xv. p. 247).

[2] Reynolds, System of Medicine, Amer. ed., vol. i. p. 124.

2. Varicella and variola are not mutually prophylactic.

This is a fact acknowledged by all writers, and it is too well known to require fuller consideration.

3. Varicella and vaccinia are not mutually prophylactic. This, also, is every-day experience.

4. Varicella is a disease only of very early life. Variola is equally common at all ages.

In view of these great pathological differences, varicella must be considered a disease *sui generis*.

The theory of Dr. Hyde, cited above, holding to the common origin of variola and varicella, is open to criticism. That the diseases may have had a common ancestor in remote times is not improbable, but Dr. Hyde places the time of beginning differentiation at too recent a date. The existence of varicella before the introduction of vaccination is proved by the statements of many writers, and varicella was as distinctly differentiated from variola one hundred years ago as it is to-day. No steps in the evolution can be traced. One of the most characteristic features of varicella is its almost exclusive appearance in infants and very young children. There is no evidence that the mild and modified variola that occurs after vaccination has any peculiar tendency to attack young subjects. The only modification of variola that is produced by vaccination is in its severity: every other pathological characteristic is retained.

Pathological Anatomy.—Death from varicella being almost unknown, opportunity to study the pathological anatomy of the cutaneous lesion rarely presents. Our knowledge of the condition is largely a matter of inference. Dr. Hyde describes it thus: "Manifestly, the exanthem is exudative in type, the serum in circumscribed areas lifting the superficial layer of the epidermis from the deeper parts of the derm. Unquestionably, septa occur in typically-developed varicella chambers, similar to those seen in variola,—a pathological fact which is the corner-stone of the doctrine relating to the unity of the disorders. The serum contained in these septa possesses an alkaline reaction. The formation of a cicatrix is evidently due to the intensity of the process in certain exceptional lesions, as a result of which the papillæ of the corium are superficially destroyed. These sequelæ are often due to the picking and scratching of the lesions."

Symptomatology.—Varicella has a period of incubation that is very long and is more variable than that of variola or of measles. It is differently stated by various observers, but some of the most careful clinicians (Thomas, Trousseau, Hesse) give it as from thirteen to seventeen days. In the cases successfully inoculated by Steiner the period of incubation was uniformly eight days. Ordinarily there are no symptoms during this period, although occasionally, as in the other exanthemata, there may be slight deviations from the perfectly healthy state.

The onset of the disease is first made known, usually, by the appearance of the characteristic rash. Watchful mothers will rarely have their attention called to any symptoms preceding the eruption, and it is very seldom that the physician is called until the formation of the vesicles is well under way. In numbers of observations in institutions the beginning of the eruption and the onset of the fever have been simultaneous. Hyde observed the evolution of the disease in twenty children in the Chicago Home of the Friendless, no one of whom was recognized as ailing before the eruption appeared. There may be, however, a premonitory stage of a few hours' duration and marked by slight constitutional disturbance. More rarely there is greater perturbation of the functions. Inflammation of the mucous membranes, high fever, and severe nervous symptoms have been noted. The writer has observed one case that was ushered in by a convulsion. A fleeting erythema sometimes precedes the vesicles. It should be borne in mind that these unusual symptoms often may have a cause other than the specific virus. Henoch regards all symptoms during the invasion stage as accidental.

The eruption of varicella generally appears first upon the upper half of the body or upon the chest. As a rule, according to the writer's observation, mothers first note the rash upon the upper part of the back. Thomas, however, gives the face as the part first invaded. From the place where the rash begins the eruption rapidly spreads over the body, face, hairy scalp, and extremities. Upon the face the eruption is usually most abundant and characteristic on the forehead and near the temples. The distribution is variable. There may be only a few small vesicles, scattered over a limited region, or, in typical and well-developed cases, no portion of the cutaneous surface may escape.

The rash begins as a number of small, red, slightly-elevated macules, which Trousseau very aptly compared to the rose-rash of typhoid fever. These macules, according to Dr. Gee, disappear when the skin is stretched, —this being a proof that there is no exudation into the cutis, simply a hyperæmia. In a few hours a small vesicle forms in the centre of each macule, and it quickly enlarges to its full size. The vesicles are round or oval in form, and vary in size from a pin-head to a small pea. They are quite superficial, being covered only by the outer layers of the epidermis, but tense and surrounded usually by a narrow inflammatory zone. According to Fagge, they are sometimes seated upon a perfectly colorless surface, so that the patient looks exactly as though he had been sprinkled with drops of clear water. The vesicles are discrete, and vary in number from twenty or thirty to two or three hundred. Thomas has noted as many as seven or eight hundred. Confluence of adjacent vesicles occurs very rarely.

Thomas says that the vesicles sometimes are congregated into small groups, making the eruption resemble zoster. The fluid contained in the vesicles is at first clear and colorless and of an alkaline reaction. As the vesicles mature, many become cloudy and the contents slightly tinged with yellow from the presence of a few pus-cells, but they never become purulent (Fox).

Within a few hours after the formation of the first vesicles, a fresh crop of macules form, which by the next morning have developed into typical vesicles. This may be repeated two or more times, so that with the development of the new and the fading of the old lesions the eruption appears to come in successive crops. As a rule, only at the beginning of the disease are the vesicles well developed and characteristic. Many of the macules that come late abort before reaching the vesicular stage. They remain as macules for a few hours and then fade. Others may form small and imperfect vesicles. Exceptional contents of the vesicles are rarely seen. Eichhorst mentions blood and air as sometimes present.

The eruption begins to decline on the second or third day. Some of the vesicles become flaccid by partial absorption of their contents; others grow tense and burst, or they are ruptured by the scratching of the patient. They then dry up and form thin, yellowish or brownish crusts. Some of the vesicles late in the disease may enlarge and form veritable bullæ, which show a slight umbilication in the centre when the lesion begins to dry. In very many cases two or three of the usual-sized vesicles also are slightly umbilicated.

The crusts fall or are scratched off in a few days, leaving small, circular, and slightly-depressed patches of reddened skin. When the skin is delicate, the large vesicles, or those injured by violence, may leave cicatrices that persist through life. The scars are circular, and very soft and white.

The mucous membranes also are the seat of the eruption. Vesicles form in the mouth and throat. They are thickest upon the hard and soft palates. The vesicles quickly lose their epidermal covering and become excoriated and resemble aphthous ulcers. Vesicles form upon the prepuce in boys and in the vagina in girls, and when present in these localities cause difficulty in urinating. The involvement of the internal organs in the eruption has not been certainly determined. That it may occur is made probable by a specimen presented to the New York Pathological Society, May 9, 1887, by Dr. Partridge. The patient died of varicella complicated with broncho-pneumonia. Diarrhœa was present during life. In the large intestine were found a number of excoriations that appeared like varicella vesicles. That a copious eruption in the intestine does not often occur is proved by the infrequency of intestinal catarrh as an accompaniment of the disease, but it is also true that a few vesicles could be present—as thick as they appear in the mouth—without symptoms of intestinal irritation.

The constitutional symptoms of the stage of eruption are variable in duration and intensity. In a few cases the disease runs its course without fever. As a rule, however, with the onset of the eruption, or preceding it by a few hours, there is a mild febrile state, with the usual symptoms of that condition. The fever may be ushered in by a slight chill or chilly sensations. The rise of temperature is generally not great, rarely going above 101° or 102° F. The fever is remittent in type, with evening exacerbations; or the morning temperature may be normal and a slight rise occur towards evening. Occasionally the fever may be so high as to be a matter of concern. Thomas reports a case in which it rose to 106.8° F. but quickly fell.

The febrile period continues for two or three days, or, in cases in which the eruption is prolonged by successive crops of vesicles, it may extend over a longer period. Persistence of the febrile state beyond the ordinary time should warn the physician to be on his guard for complications.

In uncomplicated varicella no symptoms other than those attendant upon this mild febrile movement are commonly present. Rarely the throat may be a little sore and the cervical glands enlarged and tender. With the desiccation of the vesicles and the decline of the fever convalescence is established. The duration of the disease from the initial symptoms to the last falling off of the crusts is eight or ten days.

In healthy children the disease does not show much variation in type.

Varicella Gangrænosa.—Under this name physicians of England have called attention to a peculiar and grave manifestation of varicella. Mr. J. Hutchinson was the first to describe this dangerous form of the disease. It "is not confined to weakly, ill-nourished children, but is most common in them. It is no doubt connected with the curious tendency to spontaneous gangrene sometimes met with in children" (Eustace Smith). According to several observers, this condition often attends acute miliary tuberculosis.

"In gangrenous varicella the vesicles, instead of drying up in the ordinary way, become black and get larger, so that a number of rounded black scabs, with a diameter of half an inch to an inch, are scattered over the surface of the body. If a scab be removed it is seen to cover a deep ulcer. Around it the skin is of a dusky-red color. All the vesicles do not take on the gangrenous action, so that we find many varicellous scabs of ordinary appearance mixed up with the blackened crusts. The gangrenous process often penetrates deeply through the skin to the muscles, but under some of the scabs the ulceration is more shallow. These cases are very fatal. Mr. Warrington Haward has reported the case of a weakly baby twelve months old, who weighed only six pounds and a half. The child was attacked with gangrenous varicella and died in a few days of pyæmia with secondary abscesses in the lungs."[1]

According to Dr. Crocker,[2] the gangrenous eruption does not always appear to come from the varicellous eruption, but occurs in parts not the seat of the varicellous rash. According to Hutchinson, loss of sight may result in these cases, from purulent irido-choroiditis.

---

[1] Disease in Children, Eustace Smith, New York, 1884, p. 49.

[2] London Lancet, May 30, 1885.

Complications.—Varicella has no complications that are directly dependent upon it. Various diseases, however, have at times been seen to accompany it. Among those that have been noted are erysipelas, otitis, and peritonitis. Measles and scarlatina have been reported in this country.

Sequelæ.—Not infrequently after varicella an anæmic condition is left which may continue, unless properly treated, for some time. Pemphigus and urticaria have been noted as sequelæ. Meigs and Pepper mention severe bronchitis or broncho-pneumonia as liable to result from exposure after varicella.

According to Powell,[1] it often leaves behind troublesome sores about the head and body which are very likely to lead to glandular enlargement, and secondarily to tuberculosis; and the utmost care should be taken to protect from the air by means of collodion all vesicles that are large and likely to ulcerate.

Henoch[2] reports four cases of acute nephritis following varicella, and urges the necessity of examining the urine for a few days following the exanthem. The relation of this grave disease to varicella is further confirmed by two cases reported, one by Janssen[3] and the other by Oppenheim.[4] Both of these writers urge the necessity of urine-testing following the disease. So far as the writer is aware, nephritis as a sequel to varicella has not been reported in the United States, but the knowledge of the relation may establish the etiology of cases attributed to other causes. According to the published reports of the above writers, nephritis develops in from three to twelve days after the decline of the rash. With the exception of one case, which terminated in death and in which post-mortem examination revealed parenchymatous nephritis, the clinical histories of the cases have been those of mild tubular nephritis.

Diagnosis.—Great interest is attached to the diagnosis of varicella, especially to its clinical separation from variola and varioloid. The prompt recognition of the benign character of the disease is of great importance, both to the patient and to the community; as failure on the part of the physician to diagnosticate it correctly may either subject a patient to an isolation made doubly disastrous by exposure to the infected air of a small-pox hospital, or expose a community to the danger of wide-spread infection from a variolous subject. Either mistake is a grave one, and certainly would involve the physician in its disastrous results.

Typically-developed variola differs so much from varicella in its clinical history that a mistake in diagnosis is hardly possible; but, as the result of the very general vaccination that has taken place in civilized countries, imperfect and vaguely-defined cases of varioloid frequently occur and cause much difficulty in recognition, particularly when the two diseases prevail together. The difficulty is so great that epidemics have occurred which for a time have baffled the diagnostic skill of able physicians. A remarkable example of such an epidemic was reported by Dr. Charles A. Lee in the *American Journal of the Medical Sciences* for July, 1853. This epidemic of varioloid prevailed in the townships of Gorham and Phelps, Ontario County, New York. According to Dr. Lee, the eruption bore all the distinguishing marks of varicella, pemphigus, purpura, and even erysipelas. Some of the physicians—one a practitioner with a large experience with small-pox—were so confident that the disease was chicken-pox that they refused to advise vaccination for the exposed persons. This epidemic was undoubtedly one of variola, but with unusual manifestations. Cases of typical small-pox soon occurred, and rigid inquiry traced every case directly to a case of variola imported from a neighboring city. Of the occasional difficulty in diagnosis Dr. Hyde says, "The sooner it is generally understood that intermediate forms occur which cannot be positively assigned to the one or to the other category, the better it will be for both the profession and the laity."[1]

---

[1] On Diseases of the Lungs and Pleuræ, New York, 1886, p. 278.

[2] Berlin. Klin. Wochenschr., Jan. 14, 1884.

[3] Ibid., Nov. 28, 1887.

[4] Ibid., Dec. 26, 1887.

The clinical characteristics of varicella as distinguished from variola are—

1. The age of the patients attacked by the disease.

Although variola attacks persons regardless of age, varicella is particularly a disease of infancy and early childhood. Any varicella-like eruption in an adult should be looked upon with the gravest suspicion, and the patient strictly isolated until, by the history of the case, its source, and the course of the disease, all doubt as to the diagnosis is dispelled.

2. The short period of invasion.

The eruption of varicella is not, as a rule, preceded by a distinct period of invasion: the appearance of the rash is the first indication of ill health that the child manifests. When an invasion period is present, the symptoms are of an ill-defined character and rarely continue more than one day. The invasion period of variola is three days in duration, and is marked by characteristic symptoms. It is ushered in by a chill, which is quickly followed by high fever, vomiting, and intense headache and backache. These symptoms are never met with in varicella. Even very mild cases of varioloid present a distinct and moderately severe period of invasion. Occasionally, however, it is fleeting and hardly noticeable.

3. The superficial and vesicular character of the cutaneous lesion.

Examination of the varicellous vesicle shows it to be located beneath the most superficial layers of the epidermis. The macular stage is of short duration, and the macule soft and but slightly elevated above the surface of the skin. A small vesicle quickly forms in the centre of the papule, remains a vesicle filled with clear or opalescent fluid for twenty-four or forty-eight hours, and then dries into a light, easily-detached crust. The variolous eruption passes through a distinct papular stage lasting three or four days. The papules are well developed, raised markedly above the skin level, and by the hard, shotty character of the base are shown to be situated deep in the cutis vera. The papules become vesicular on the sixth or seventh day, and by the ninth day the vesicles are transformed into umbilicated pustules.

---

[1] Pepper's System of Medicine.

4. The abundance of the eruption upon the body.

In varicella the eruption is most abundantly and characteristically developed upon the back. The face, hands, and feet show but few vesicles. In variola the eruption is thickest and apt to become confluent upon the face, hands, and feet.

5. The transient febrile stage.

The constitutional symptoms of varicella have been seen to be very insignificant: very rarely does the febrile period continue over three or four days. The temperature is commonly highest at the beginning of the eruption and declines as the vesicles desiccate. There is no secondary fever. In variola the temperature, after maintaining a high point during the invasion period, suddenly falls with the appearance of the rash, and when the eruption becomes pustular the secondary fever manifests itself.

Although typical variola and well-developed varioloid differ thus widely from varicella, very mild and abortive cases of varioloid occur in which a diagnosis presents the greatest difficulty. The invasion stage may be short and so mild as to escape observation; the eruption may be arrested in its evolution, never reaching the characteristic pustular stage of typical variola; and the febrile stage may be transient. When such irregularities occur, only the greatest care will save the physician from error. No one symptom or manifestation can be relied upon, but all the points in the history and development of a given case must be carefully weighed.

Mr. Makuna[1] states that the varicella vesicles are always unilocular and can be emptied by one puncture, and that the contents are serous and watery. The variolous pustules are always multilocular, cannot be emptied by one puncture, and contain a plastic and viscous fluid. This observation is not confirmed by other authorities. The varicellous vesicle is undoubtedly occasionally umbilicated, and often contains delicate trabeculæ which make it multilocular.

Impetigo and impetigo contagiosa may be confounded with varicella. The latter disease is distinguished by the accompanying febrile disturbance, the numerous vesicles, the vesicular type of the lesion, and the halo surrounding it.

According to Dr. Ashby,[2] a vesicular syphilitic eruption may simulate varicella. He cites a case reported by his colleague Dr. Hutton in which "an eruption (apparently syphilitic) of vesicles, somewhat hard and shotty, seated on an inflamed base, made their appearance in a child of three years; the vesicles appeared in crops for ten days, each vesicle lasting about six days, leaving some staining." The diagnosis between varicella and syphilis would be made by the history of the case, the temperature, which would not be elevated in syphilis, and the presence and distribution of other forms of the specific eruption, as bullæ or papules.

---

[1] London Lancet, Sept. 6, 1879.

[2] Archives of Pediatrics, vol. iii. p. 101.

Prognosis.—Varicella is the most benign of the exanthemata. When uncomplicated,—and complications are very rare in the United States,—the prognosis is always favorable, and the profession and the laity look upon it as a trifling disorder. Were it not for the suspicions that the eruption excites, physicians would rarely be called to see the disease. This fact makes the study of varicella outside of institutions very difficult.

In children of feeble constitution or debilitated by bad hygienic influences or recent disease, varicella may leave a condition of impaired health that will favor the development of scrofula.

Eczematous eruptions after varicella with concurrent swollen glands may lead, according to Powell, to the development of phthisis.

The prognosis of varicella gangrænosa is grave, many of the cases terminating rapidly in death.

When complications occur, the prognosis will depend rather upon their character than upon the primary disease. The cutaneous lesion terminates, with but few exceptions, in the complete restoration of the continuity of the skin. Upon the face, when the skin is delicate, or because of violence, a few vesicles may leave permanent cicatrices.

Treatment.—Commonly no prophylactic treatment of varicella by isolation of the affected person is necessary, although children enfeebled from any cause, and to whom any febrile disease is a matter of concern, should be protected from exposure.

The child with varicella should be kept quiet in bed or upon the sofa during the febrile period, and the indications of the febrile state met as occasion demands. Rarely is any treatment demanded other than quiet and light food and the proper regulation of the temperature of the room.

The lesions upon the face, particularly in girls, should be watched and care taken to protect them from violence. It is advisable to empty the large vesicles upon the face by a puncture, and bathe them with a mild antiseptic lotion to favor their rapid resolution.

The treatment of the grave form of the disease known as varicella gangrænosa should be the supporting treatment of gangrenous conditions from other causes.

The continued anæmia sometimes following the disease should be met by iodide of iron and a bitter tonic; and in children in whom there is left a tendency to cutaneous eruptions attended by glandular enlargements, a course of cod-liver oil and the careful regulation of the diet are of great importance.

PAROTITIS.

By OLIVER P. REX, M.D.

Derivation and Synonymes.—From the Greek *παρά*, "near," *οὖς*, *ὠτὸς*, the "ear," and the suffix *itis*, denoting inflammation. Latin, Phlegmone parotidæa, Inflammatio parotidum, Cynanche parotidæa; Italian, Parotide; Spanish, Parotiditis; French, Parotide; German, Ohrspeicheldrüsenentzündung,—vulg., Ziegenpeter, Mumps, Bauerwetzel, Tölpelkrankheit, Kehlsucht, Klirren; English, Parotiditis, Mumps.

Definition.—A contagious epidemic inflammation and enlargement of the parotid gland, generally occurring in youth, acute in its origin and course, accompanied by fever and fever-symptoms, followed in some cases by an abscess of the gland, but usually subsiding within a week or ten days, without leaving any trace.

Varieties.—A condition of tumefaction and inflammation may be set up in the parotid by a blow or some external injury, and, following such a trauma, epidemic parotitis may arise. Though this may occur, a class division of traumatic parotitis is hardly necessary or within the terms of the definition. The same may be said of those retention-cysts which arise from occlusion of the duct by a foreign body or as a result of local inflammations. It may be questioned also if those cases of so-called mumps that have been reported as caused by local diseases, stomatitis, extensive disease of the mouth or teeth, diphtheria, etc., are not in reality examples of duct-occlusion as a result of cicatrices or local injury. The epidemic and contagious nature of parotitis must make us examine such cases carefully and prevent us from multiplying our varieties of parotitis proper. We have remaining two divisions:

1. Idiopathic, or, more properly, epidemic parotitis;

2. Secondary, symptomatic, metastatic, deuteropathic, malignant, or suppurative parotitis, or that following typhus, dysentery, scarlet fever, small-pox, measles, etc.

The secondary form of the disease differs in no essential respect from the idiopathic, except perhaps in tendency to proceed to suppuration of the gland. This is doubtless due to the fact that, as a result of the previous or intercurrent disease, not only is the system weakened, and the tissues rendered less able to resist attack, but there is also an excess of the morbific material in the body, which acts injuriously upon the parotid gland.

My own experience, however, is that upon opening the suppurating organ there is far less pus in it than its appearance indicated.

Besides the evidently necessary greater attention to the systemic conditions and disease, the treatment of secondary parotitis is the same as that for the idiopathic form that is given below.

IDIOPATHIC OR EPIDEMIC PAROTITIS.

Etiology.—We know nothing of the essential nature of the origin of the disease. That it is distinctly though not intensely contagious, there can be no doubt. Its epidemic nature is more evident in the country than in cities. The law of its spreading is not clear. In some localities it will disappear for twenty or thirty years, whilst in others it seems to be almost endemic. It is rarely sporadic, and the epidemics are usually in the spring or fall. Infants are seldom attacked, and the affection is confined to the period of childhood and early youth, though adults who have not previously had it are sometimes the subjects. Females are more exempt than males. One attack gives almost certain immunity from others. Virchow, Niemeyer, and others hold that it arises as a catarrhal inflammation of the ducts proceeding thence to the acini of the gland.

Pathological Anatomy.—From the fact of its non-fatality, it is evident that but few post-mortem examinations of the gland during its disease have been made. From these, however, it is reasonably clear that the pathological process consists essentially of a hyperæmia and a serous infiltration of the acini. If not absorbed, this exudation may lead to permanent enlargement of the gland, or, by compression, to its atrophy. This hyperæmia and exudation are also present in different degrees in the tissues in the neighborhood of the gland, may extend to the lymphatics of the neck, and occasionally to the submaxillary glands.

Symptomatology.—The period of incubation is variously given at from six days to two weeks. The prodromic symptoms usually appear about a week after exposure, and consist in feelings of languor, malaise, loss of appetite, irritability, slight feverishness, etc. These signs may be so slight as to escape notice, or in the weak and nervous may become so marked as to demand great care or lead to a mistake in diagnosis. Cases have been recorded where convulsions have occurred during this initial stage, or where vomiting, diarrhœa, delirium, and a tendency to syncope have been alarming.

The local exhibition of the disease, the pain and swelling of the parotid, may appear within a day after the prodromes, or not for a week thereafter. Its first indication is shooting pains beneath the lobe of the ear during motion of the jaw. A deep-seated swelling will soon be found at this place, which gradually increases and extends till the side of the face and neck are implicated. The swelling is at first upon one side, but is usually followed by swelling of the other side within one or two days. The head is at first held towards the affected side, to avoid tension of the affected muscles and tissues, but when the affection is bilateral the head is held rigidly erect. The appearance of the patient is somewhat ludicrous, owing to the swollen neck, the immovable head and muscles of expression, the staring eyes, and the changed, foolish expression of the face. This impression upon the bystander has been so great as to stamp itself in language, numerous derisive epithets for the disease existing in most languages. To the patient it is a very different matter, since he will suffer considerable hunger and thirst rather than endure the pain that results upon attempting to chew or swallow. The disorders of function extend to impaired enunciation, hypersecretion of saliva, or sometimes its reverse, with great dryness of the mouth, pains and ringing in the ears, imperfect hearing, etc. As a result of the difficulty of eating, loss of appetite, vomiting, constipation, etc., may be set up; whilst cerebral hyperæmia may in rare cases follow from pressure upon the cervical veins.

The constitutional symptoms are those of the prodromous stage exaggerated: the fever may rise to 104° F., but commonly does not go above 101° to 102°, with the increase of pulse and the gastro-intestinal and cerebral symptoms that are concomitants of the elevation of temperature.

All the symptoms of mumps may sometimes be lacking to such a degree as to be out of all proportion to the swelling present.

A striking peculiarity of this affection is its occasional metastasis to the genital organs. In boys there may occur an orchitis, or swelling of the testicle (usually of the same side, and this the left), with scrotal œdema, whilst in girls the ovary, vulva, or mammæ are similarly affected. This inflammation is not generally severe, and runs about the same course, as regards time, as the parotitis. It may even disappear from these parts and be followed by an exacerbation of the parotid symptoms which, at the appearance of the orchitis, etc., had already notably lessened or disappeared.[1]

Diagnosis.—The inception of the malady may be mistaken for that of one of the exanthematous fevers, but the mistake would be apt to have no very serious consequences, since, as we know of no specific prophylaxis, the expectant treatment would apply equally to both. With the appearance of the tumor and its local manifestations, hardly to be mistaken for an enlarged cervical gland, sarcoma, etc., all doubt is set at rest, though a retention-cyst from occlusion of the duct-orifice, by local disease or a foreign body, may cause all the symptoms of epidemic parotitis. In such cases the lack of a history of exposure or of an existing epidemic will lead to an examination of the mouth, and more particularly of the duct. Occlusion by such almost imperceptible substances as a bit of tooth-brush bristle or spicule of a nut-bur may require the most careful examination to reveal their presence. If there be local disease of the mouth, it is necessary to keep in mind the possibility of a mechanical occlusion by the results of inflammation, by cicatrices, etc. External injury may also set up a swelling of the gland like the real parotitis in many respects.

---

[1] One of the interesting curiosities of this subject is the case detailed by Schmalle (Inaug. Diss., Greifswald, 1886), and shows that though the metastasis is generally from the parotid as an original source, this reflex neurosis may start out from a far-distant point and end in a pronounced parotitis. Three days after a surgical operation for hemorrhoids with the thermo-cautery, a severe attack of acute parotitis with fever broke forth, and with such extensive suppuration of the gland that three incisions (in the gland, behind the ear, and in the external auditory meatus) were required to give vent to the large quantities of pus.

Prognosis.—The prognosis is good. No case of death has been reported due directly to the disease itself. The duration of the disease is about ten days, though if, as usual, one side of the face is affected some time after the first, the whole duration of the illness is proportionately lengthened. The scrofulous and otherwise weak may not recover so quickly. Atrophy of the gland seldom follows a severe case, and still more rarely is there atrophy of the testicle in metastatic orchitis. Cases of meningitis due to parotitis have been reported.

Treatment.—The disease being self-limited, not dangerous, of short duration, and its specific cause unknown, the indications of treatment are of course manifest. Rest, even some days of confinement to bed, or at least to the house, is advisable. The fever, if slight, does not demand active antipyretic treatment. Cooling drinks and a light nourishing diet are recommended. If irregularities of the digestive system exist, they should be corrected according to the judgment of the physician. Saline laxatives will control the tendency to constipation. Owing to the difficulty of chewing and swallowing, liquid food, milk, beaten eggs, broths, etc., will be required. If there be great restlessness or marked cerebral symptoms, it will be well to apply cold to the head, or give light doses of aconite; chloral may be required, but morphine only in rare and extreme cases. The patient may find it very grateful to swallow bits of ice, or to allow the same to melt in the mouth. The giving of an emetic in cases of bilateral attack or in those of orchitis, as recommended by some authors, is not, in my estimation, advisable for children.

Topically, a mixture of opium and sweet oil, one drachm to the ounce, should be rubbed upon the tumor, which is then protected by a light dressing. Cold applications, though perhaps useful, are not liked by the patient. If a tendency to suppuration is noticed, shown by a tenseness and redness of the skin, a leech or two may be applied behind the ear. Should an abscess become inevitable, its formation should be hastened by poultices, and when formed it should be opened and its contents thoroughly evacuated, to prevent complete disorganization of the gland, or a possible perforation of the cavity of the tympanum.

ERYSIPELAS.

By J. O. HIRSCHFELDER, M.D.

Synonymes.—English, St. Anthony's fire, The Rose (Scotland); Latin, Febris erysipelatosa, Ignis sacer; German, Erysipel, Rose, Rothlauf; French, Érysipèle; Italian, Risipola.

Definition.—Erysipelas may be defined to be a dermatitis having the tendency to spread rapidly, accompanied by comparatively severe constitutional symptoms, with rapid resolution and complete return to the normal condition.

The etymology of the term is obscure, the most probable derivation being from *ἐρύω*, to "draw," and *πέλας*, "near," the wandering character of the disease being thereby indicated.

History.—Knowledge of this disease dates back to the time of Hippocrates, who speaks of it in his writings. It is likewise referred to by Galen, who supposed that a bilious humor in its efforts to escape from the blood through the skin occasioned erysipelas. This belief was long entertained, and was succeeded by various theories, depending upon the state of medical philosophy at different periods. At one time looked upon as the outward manifestation of alterations of the blood, at another as a simple inflammation of the skin, again as a member of the family of exanthematous diseases, it has finally come to be regarded by the best investigators as the result of the invasion of the body by a specific germ.

Etiology.—We find erysipelas occurring with greater or less frequency at all places, at all seasons, and under most varying external conditions; sometimes showing itself only here and there as sporadic erysipelas, at other times the cases becoming so numerous as to merit the name of epidemic. It may follow injuries or operations, as surgical or traumatic erysipelas, which form we shall not consider in this treatise except in so far as the lessons taught by it may lead to a better understanding of the disease in general. On the other hand, erysipelas may arise without any traumatism, as the medical or idiopathic form. This distinction between the traumatic and the idiopathic erysipelas has been found to be more apparent than real; for investigation has shown in the latter cases that a primary lesion—a point of entrance of the poison—can be found in almost every instance in which the examination is made with sufficient care. The erysipelas may arise from a slight abrasion of the skin, an intertrigo, a small pustule, an eczema, an ulceration, a leech-bite, or some such trivial lesion that under ordinary circumstances might escape attention.

The causes that produce erysipelas are the direct and the predisposing. It has long been maintained that erysipelas is a specific disease produced by a specific germ; but until lately that germ had not been isolated and its causal relation to the disease under consideration established. The investigations which led to this conclusive proof were instituted by Fehleisen, who discovered micrococci 0.0004 mm. in length occurring in chains of from six to twelve in the erysipelatous tissue,—micrococci which he isolated and cultivated. With the culture-fluids he made inoculations and produced the characteristic signs. There has been much discussion upon the subject of micro-organisms and the *rôle* they play in the etiology of disease, and much has been assumed by the bacteriologists that has not been proved. On the other hand, however, the antagonists of the germ-theory have displayed a scepticism which contrasts strongly with the credulity with which recommendations of therapeutic measures are received and which in many instances passes far beyond the limits of the allowable. It certainly behooves us to deal cautiously with so vital a question; but in the individual disease a germ must be accepted to be the etiological factor when it is invariably found in every case and when inoculations of the well-isolated culture are followed by the characteristic signs of the disease in question. Such conclusive evidence of the micrococcus of Fehleisen being the cause of erysipelas has been brought forth. We have even been able successfully to inoculate human beings with such cultures for the purpose of curing other diseases. It had frequently been observed that various tumors, nævi, etc., upon becoming the seat of erysipelas disappeared. Thus, Busch related in 1866 that he saw multiple sarcomata disappear after erysipelas following extirpation of one of them. In a case of sarcoma of a lymphatic gland of the neck which extended from the clavicle to the parotid, erysipelas supervened upon an injection of morphine, and the gland diminished to one-half its previous size, at the same time becoming soft. The patient died of collapse, and at the post-mortem examination Rindfleisch found that the tumor had become changed to an emulsified fluid filling a reticulum.

These therapeutical experiments were repeated by Fehleisen upon a larger scale. He successfully inoculated six individuals affected with sarcoma, carcinoma, and lupus with cultures of the fourth, ninth, fourteenth, fifteenth, sixteenth, and seventeenth generations, producing erysipelas. The shortest period of incubation was fifteen hours, the longest sixty-one hours.

Janicke inoculated a case of cancer of the breast with Fehleisen's cocci. In seven or eight hours a chill occurred, with a temperature of 104.5° F. Twelve hours later erysipelas set in, extending over the entire body, producing death on the fourth day. At the post-mortem examination the cancer was found to be softened and infiltrated with the cocci.

Whatever may be the therapeutic value of these experiments, they certainly prove that erysipelas is caused by the micrococcus.

Sex.—It is frequently stated that females are more disposed to erysipelas than males. This statement is, however, erroneous.

Age.—It is more frequent in the first year, and after that age occurs as often in adults as in children.

Season.—Erysipelas is supposed to occur more frequently during the cold than during the warmer months. This is not invariably the case, as one of the severest epidemics, that of Paris in 1861, took place during the summer. Hirsch found erysipelas most frequent during sudden changes of the weather.

Contagion.—There cannot be the slightest doubt that under certain circumstances erysipelas is directly contagious from person to person. It does not act at a distance, as do other infectious diseases, for a number of cases are not affected at once. This contagion is not always evident, but in many instances it has been firmly established. In all the works upon the subject conclusive cases have been cited in great numbers. One of the most classic instances is that communicated by Dr. Blin to the Paris Academy in April, 1864. Dr. Paintevin, assistant of Voillemier in the Lariboisière, contracted erysipelas from two cases under his charge in the ward. Dr. Testart, from Guise, a place in which up to the time there had been no erysipelas, visited him during the acme of the attack. Dr. Testart returned to Guise, and three days after leaving Paris became affected with erysipelas. Dr. Testart infected a servant who waited upon him, and a relative who visited him from a distance of about twenty-four miles and who manifested the disease two days later. The wife of the latter patient then became infected, and also three members of a family named Lefranc who had called upon the invalids. A relative of Lefranc, aged seventy, living in a neighboring village, became infected after a visit, and likewise two Sisters of Mercy who waited upon the Lefrancs. Upon returning to their convent they infected others. The physician who attended them died of erysipelas, and his daughter became infected eight days after his return home, the infection in the latter case having its origin in leech-bites that had been made over enlarged submaxillary glands.

Epidemics of erysipelas have been recorded in various hospitals, and have been most frequent in the obstetric wards in which puerperal fever existed. These epidemics differ from those of the exanthemata which spread over a large area, inasmuch as they are limited to one hospital or, it may be, to one bed. The rise and disappearance of these hospital epidemics are somewhat mysterious. They are most frequently observed in crowded, filthy, and ill-ventilated wards, but have also occurred where the hygienic conditions were the most favorable imaginable. On the other hand, the most unfavorable states have not always been productive of erysipelas, as, for example, during the Crimean War, in which the sanitary measures were almost entirely neglected, hardly any cases of the disease were observed.

In some instances a direct relation to sewer-gas poisoning could be traced, as in the notorious case of the Middlesex Hospital. Here during a long period a number of cases of erysipelas occurred in two beds, whereas no other cases were observed in the ward. No reason for this peculiarity was discovered until a defective drain-pipe from a privy was found to run behind the plaster between these beds. The defect was remedied, and no more cases occurred until ten years later, when the same faulty condition of the drainage again led to erysipelas in the same beds, and again ceased upon repair of the pipes.

Vaccination is a frequent etiological factor. Thus, Dr. Doepp, of St. Petersburg, vaccinated nine children from one who developed erysipelas on the following day. All of the nine individuals became affected with erysipelas. In 1850 vaccination in and around Boston was so frequently followed by erysipelas that the vaccination was discontinued. A cachectic condition of the individual seems to predispose to the disease.

Erysipelas not infrequently returns in the same patient every year or oftener. This is especially the case when the face is the seat of the disease. In such instances chronic rhinitis, eczema, or some other form of chronic inflammation exists, from which the infection occurs.

Symptoms.—Erysipelas in children over six months of age very much resembles that of adults, differing from it only in slight respects. That of the new-born, however, is quite distinct. We shall first describe the form occurring in older children, and then consider the affection of the new-born.

Erysipelas may be introduced by a prodromal stage, which is, however, often absent. When present the prodromes are not characteristic, being those common to many other diseases. We find the child drowsy or restless. There is a general feeling of malaise and aching of the limbs. There may be more or less fever, and in some instances oppressed breathing may exist for a few days previous to the manifest signs of the disease.

The disease proper frequently begins with a chill, which may be more or less severe, and which in younger children is often replaced by convulsions and vomiting. Following these there is a rise of temperature, which may reach as high as 105° F. Usually the local manifestations begin to develop immediately; there is a feeling of warmth, tension, and pain in the affected part, which becomes mottled pink and somewhat œdematous. The patches gradually become more intensely red, and coalesce to form a single fiery patch, whose color disappears upon pressure, to reappear when the pressure is removed. At the same time the swelling of the affected skin increases, especially if the part be one in which the subcutaneous tissue is loose. The edge of the patch is elevated on one side and terminates sharply towards the healthy skin. From this elevated edge the erysipelatous infiltration rapidly extends to the neighboring skin. The extension usually first begins by the formation of small branches followed by the line of infiltration; as the disease progresses, the parts behind gradually become paler, and within two or three days have all the appearances of healthy skin. The extension may be more or less rapid and may involve a smaller or a larger territory. The fever during the progress of the disease remains high, and is accompanied by more or less severe constitutional symptoms. The appetite is lost. Nausea, vomiting, intense headache, and thirst are present. The tongue is covered with a thick, dry coat. The urine is passed in small quantities, and not infrequently contains albumen. Sleep is much disturbed, or may even be entirely absent. In some cases delirium or a soporous condition may be present. The mucous membranes adjoining the skin are oftentimes likewise involved in the process. In the cases terminating in recovery the progress of the skin-affection ceases, the redness gradually pales, the swelling subsides, and the fever disappears. Where death ensues, it usually occurs with a high temperature.

Seat of Commencement.—Erysipelas begins most frequently in the face. Some local affection of the skin determines its point of origin. This is most frequently situated where the skin passes into the mucous membrane at the nose, mouth, eyelid, or ear. It may arise at the genitals or the rectum, and not infrequently proceeds from intertrigo, eczema, a pustule or acne eruption, or some other affection of the skin which occasions a solution of continuity. Erysipelas may arise in the mucous membrane, as, for example, in the pharynx, and may then extend outwards to the skin and from there run its usual course. In fact, many of the cases of facial erysipelas in which no point of origin can be found, and which were formerly thought to be idiopathic, proceed from the interior of the nose. These internal forms may pass outward upon one or more of the following routes: 1, to the lip; 2, through the choanæ and nostrils; 3, through the nasal cavity and lachrymal ducts; 4, through the Eustachian tube, passing through the middle ear to the external ear: the tympanum offers no obstruction. Many of the cases of erysipelas which appear at the root of the nose pass through the lachrymal duct from the interior of the nose. Under such circumstances the lachrymal sac appears distended, as when obstruction of the passage occurs. This sign may precede the external erysipelas by some days, and through it we may prognosticate the approaching affection. Erysipelas occurs with greatest frequency on the head, next on the trunk, and least often on the extremities.

Integument.—Upon the surface of the skin erysipelas usually begins as a mottled pink patch, which rapidly becomes darker and confluent. From this initial focus the redness rapidly extends, running in the direction of those lines of least tension of the tissue to which attention has been called by Langer. At parts where the subcutaneous tissue is firm and adherent the progress of the erysipelas is arrested, as, for example, at the base of the skull, over Poupart's ligament, etc. The surface of the skin may be smooth and glistening, but is often covered with vesicles that vary in size from the very smallest, which require a lens to observe them, to large bullæ. The skin often feels rough like shagreen leather. The blebs are sometimes tinged with blood, and gradually become turbid from admixture of pus and epithelial cells with the serum. There is always more or less swelling present, depending in degree upon the severity of the affection and the looseness of the areolar tissue. In certain parts, as the face and the genitals, the intumescence may reach enormous dimensions. On the third to the fifth day the redness and swelling in the part affected gradually fade; vesicles that are present are either absorbed, or burst, or dry to yellowish crusts. In the subsequent desquamation the cuticle either is shed in a fine scurf or peels off in layers. The skin rapidly returns to its normal appearance. At times in parts abscesses develop, or gangrene may ensue. The latter lesion is preceded by the development of bloody blebs. It has been stated that the gangrenous process may be prevented by the early pricking of these sanguineous vesicles.

Mucous Membranes.—The membranes most frequently the seat of erysipelas are those of the respiratory tract. The erysipelas may be primary, or may extend from the skin. In either case we find the surface intensely red, and glistening as if varnished. There is but little swelling. According to Todd, the muscles of the pharynx do not react to irritation, so that when the patient swallows solids do not pass into the œsophagus, but remain in the larynx. The paralysis is both motor and sensory. The process may extend to the glottis and produce death by suffocation through œdema glottidis, or may pass onward to the lungs, giving rise to a peculiar form of pneumonia. We find slight dulness, with subcrepitant rather than crepitant râles, changing their seat from day to day, differing from the ordinary form of pneumonia in the rapid extension of the lesion from one part to the other. In such cases both Strauss and Denuce found no fibrinous exudation in the alveoli, but instead leucocytes and granular cells. The lymph-glands supplied by the affected region rapidly swell, and are very painful. In erysipelas of the face swelling of the submaxillary glands may precede the external appearance upon the skin by several days. In such cases we have some hidden origin of erysipelas, as in the nose.

Digestive Tract.—We find a thick, dry coat upon the tongue, which may even be crusty. The ordinary symptoms accompanying fever are present.

Kidneys.—The urine is similar to that of other febrile affections. It is claimed by some that albumen is usually present. According to Blechmann, albuminuria reaches its greatest height from the second to the sixth day, and may last from nine to twenty-two days, or may be chronic. Da Costa finds it in every severe case of erysipelas, and often transitorily even in slight cases. The quantity of albumen, according to this author, is usually small. He not unfrequently finds red and white blood-cells, but rarely blood-casts. The albuminuria may be due to parenchymatous changes of the kidney, such as accompany all high fevers, or to specific nephritis.

Cerebral Symptoms.—These may be the ordinary febrile disturbances of the brain, headache, hebetude, etc., or we may have wild delirium or a comatose condition. The cerebral signs are more intense in affection of the scalp, and were formerly incorrectly ascribed to meningitis. Trousseau and others following him have found nothing pathological within the cranial cavity. The disturbances are to be ascribed in part to the fever, in part to a specific alteration of the blood, and in part to impeded return of venous blood from the brain through the pressure of the œdematous scalp.

Fever is usually introduced by a chill, or at least by a sensation of chilliness. The temperature rapidly rises to a high degree, and falls somewhat with the appearance of the eruption. Following this is a further rise, the temperature remaining high during the progress of the erysipelas. Fall usually occurs by crisis in from twenty-four to forty-eight hours, but may be more gradual,—by lysis, as it is termed. The critical fall may be preceded by a precritical rise.

The pulse goes hand in hand with the temperature, except in very feeble individuals, in whom it is proportionately more rapid.

Erysipelas in the new-born presents a very different picture from that witnessed in older children. We do not usually find the same violent onset. It arises insidiously, and the fatal disease has generally developed before the attendants are aware of the fact.

In the new-born, erysipelas usually begins about the navel or in the region of the genitals. On the first day all that is seen is a slight blush of the affected parts. The infant suckles well, and may have no fever. If it be robust, this condition may continue for as long as three or four days. Soon, however, a change in the scene occurs. High fever develops; the child no longer takes the breast willingly; it nurses irregularly, and vomits that which it had taken. It becomes agitated, restless, sleepless, cries continuously. The pulse is irregular, small, and frequent. Diarrhœa with stools that are yellow occurs in the beginning, but later the dejecta become green and liquid. At the same time the affected skin becomes enormously distended and glistening. The tension of the part is so great that it is difficult to make an impression, and this when made rapidly disappears. The appearance has been well compared to that of a urinary infiltration. Phlegmonous inflammation with the development of subcutaneous abscesses is very frequent. In many cases gangrene of the affected parts occurs. Death almost invariably results. The child either becomes more and more soporous and finally passes away in a condition of coma, or death may be ushered in by convulsions. The course of erysipelas in the new-born is more erratic than in adults, but the progress of the disease is not attended with the same exacerbations of fever. The fever does not usually reach the height which it attains in older children. The disease generally lasts from five to fifteen days; but the progress of convalescence is often retarded by abscesses or gangrene. Death may be hastened by complications, which readily ensue, especially peritonitis, which is apt to be produced by extension of the inflammatory process through the umbilical vein. Meningitis, pleuritis, and pulmonary complications are by no means rare.

Course.—Erysipelas is an acute disease, and runs its course, as a rule, in from ten to fourteen days. The duration is longest where the trunk is involved, and shortest where it is localized in the lower extremity. Relapses of erysipelas are comparatively frequent. The second attack usually has the same seat and runs the same course as the first, but is apt to be lighter. Certain individuals are predisposed to the disease, and are the victims of an habitual erysipelas which occurs yearly or even more frequently. Such habitual erysipelas is usually facial, and has its origin in persistent affections of the mucous membranes of the head. It is, therefore, more frequent in scrofulous subjects.

Complications and Sequelæ.—In cases of long duration, especially such as are attended with enfeeblement of the patient, hypostatic pneumonia is very apt to ensue. Croupous pneumonia likewise occurs.

Pleuritis is a very frequent complication. It is maintained by some observers that it is due to a direct continuation of erysipelas of the skin which covers the thorax; but it has also been found in some cases in which the chest is not otherwise involved, as, for example, in erysipelas of the face.

Peritonitis is a complication which occurs with moderate frequency. It is especially apt to occur in the erysipelas of the new-born, in whom purulent inflammation of the umbilical vein is the connecting link between the external and the internal affection.

Affection of the endocardium has lately been frequently assumed by French authors as a concomitant of erysipelas. It is certainly not so frequent as they suppose, the murmurs leading to the diagnosis probably being more frequently due to anæmia than to affection of the valves.

Pyæmia is most apt to occur in traumatic cases and in such as are accompanied by gangrene.

Various affections of the eyes have been observed in erysipelas, from simple conjunctivitis to perforation by corneal ulcers. Amaurosis has been produced by retrobulbar abscess.

Pathological Anatomy.—The histological changes that occur in the skin in erysipelas are similar to those observed in a burn or those occasioned by an application of iodine. There is excessive congestion of the blood-vessels where the erysipelatous process is at its height, together with transudation of a large quantity of plasma, so that the affected tissue is more or less œdematous. At the same time we find numerous leucocytes surrounding these blood-vessels like a sheath. This infiltration of small cells is most evident in the deeper layers of the corium and in the subcutaneous cellular tissue enveloping the fat-cells. The tissue-elements themselves are not affected: there is no increase in the connective-tissue cells. The epidermis is loosened from its connection with the corium, so that the dried cuticle can be readily removed, leaving the swollen papillæ exposed.

In the skin immediately in advance of the red margin, and extending for a distance of about one centimetre corresponding to the line of advance, Fehleisen found the tissue infiltrated with the specific micrococcus. These little bodies, 0.0004 mm. in size, are of high refraction and occur in chains of from six to twelve. They fill the lymph-passages of the corium, especially in its superficial layers, and are but few in number in the subcutaneous tissue. None are found in the blood-vessels. They rapidly increase in numbers by fission.

In the red zone Fehleisen finds signs of inflammatory reaction. Here, as described above, a large number of wandering white blood-corpuscles are found among the micrococci, repelling them and in part enclosing them.

In the part immediately behind the red zone only the leucocytes are found, but no micrococci.

When the erysipelatous redness begins to fade, the leucocytes rapidly degenerate, changing to a finely granular mass, so that in a few hours all that remains is a fine detritus. Within a day or two this also disappears, and the skin resumes its normal condition. This rapid *restitutio ad integrum* is characteristic of erysipelas, and is due to the fact that the tissue-elements are not themselves the seat of any lesion. In some cases, however, they do not entirely escape. The white blood-corpuscles wander out in such numbers that the nutrition of the tissue-elements suffers, and a breaking down occurs, leading to the formation of abscesses. Such minute abscesses develop most frequently at the apices of the papillæ.

Respiratory Tract.—Inflammation of all parts of the respiratory tract is found in erysipelas, from congestion of the mucous membrane of the nose to an erysipelatous infiltration of the tissue of the lungs differing from croupous pneumonia by the absence of fibrinous exudation into the alveoli. Hypostatic congestion of the lungs is frequently found post mortem; also, at times, pleuritis, with serous or even purulent fluid. In the intestinal tract inflammatory changes have been observed, especially in the duodenum, in which small ulcers sometimes occur. These are similar in character to those occurring in extensive burns of the skin, and are probably similarly produced by reflex hyperæmia of the mucous membrane.

The spleen is usually found to be enlarged and soft. The kidneys, the heart, and the liver have been described by Ponfick to be in a state of parenchymatous degeneration, as in all cases of continued high temperature. Nothing characteristic of erysipelas has been found in these organs.

Diagnosis.—Where the disease has developed upon the skin, as a general thing the diagnosis presents no difficulty. The sharply-defined redness with intumescence gradually extending forward, with rapid retrogression in the older portions, and the severe constitutional signs, render its recognition easy. Before the eruption has manifested itself, however, the diagnosis may be more difficult. Chill, fever, and depression are signs of many diseases; but the painful swelling of the lymphatic glands of the region about to be involved offers a sign of some value. In erysipelas of the face the glands of the neck are swollen, in that of the upper extremity, those of the axilla; and in erysipelas of the lower extremity the lymphatics of the groin will be found tumefied and sensitive. Swelling of the lachrymal sac is an important diagnostic aid in approaching cases of facial erysipelas. Primary erysipelas of the mucous membrane is sometimes very difficult to diagnosticate before it has progressed to the skin. The glistening, varnished-like appearance of the membrane is characteristic, together with the painful enlargement of the glands and the severe constitutional affection. These are especially indicative during an epidemic of erysipelas.

Eczema rubrum may be recognized by the red color, with œdema and small vesicles, but without fever.

In *simple erythema* we have a brighter red color of the skin, without tumefaction or sensibility on pressure, and without fever.

In *urticaria* we have a whealy eruption, with simultaneous affection of various parts of the body. There is likewise manifest absence of general symptoms.

With the new-born the commencement of *sclerema* is sometimes taken for erysipelas. In the latter the induration is confined to the skin and the subcutaneous tissue, whereas in sclerema it occupies the entire thickness of the member, involving the muscles likewise. The tumefaction is firm and hard, without redness, and with a depression rather than a rise of temperature.

In *superficial lymphangitis* the redness is streaked and follows the lymphatics, which may be felt as hard cords, or disseminated nodules may be found along the course of the lymphatics.

In *phlegmonous inflammation*, or *pseudo-erysipelas*, the swelling of the skin is more board-like, and usually terminates in abscesses. The redness of the skin is darker than in erysipelas, and is not so sharply defined.

Prognosis.—Erysipelas occurring in older children, as a rule, runs a favorable course. While this is true in ordinary cases, there are certain epidemics of erysipelas in which the prognosis is far more unfavorable. Age is a very important prognostic element. Erysipelas of the new-born is a most malignant disease. All observers unite in the statement that almost every child under three weeks that becomes affected with erysipelas dies. This excessive mortality is thought to be due not so much to any specific character of the disease as to the feebleness and vulnerability of the infantile organism, although it has been maintained by Trousseau, among others, that the erysipelas of the new-born is not ordinary erysipelas, but a puerperal form.

With increasing age of the child the prognosis becomes more favorable. After the second year a child is in but little more danger than an adult. The feebler the organism, other things being equal, the greater the danger.

High temperature of itself is not a specially unfavorable sign, unless it be of long duration.

Ambulatory erysipelas is dangerous on account of its lengthened course and the consequent effects upon the patient. Severe delirium is likewise an unfavorable symptom.

Affection of the mucous membranes increases the danger, as was known as far back as the time of Hippocrates, who stated that it was an unfavorable sign when erysipelas extended inward.

Complications, of whatever kinds, render the prognosis of erysipelas more unfavorable.

Treatment.—*Prophylaxis.* For the prevention of the spread of erysipelas, isolation of cases is absolutely necessary, as the contagious character of the disease has been established beyond a possibility of doubt. The experience of all hospitals in which erysipelas has been prevalent proves the necessity of strict quarantine of affected cases. In some institutions through this measure the disease has become practically eradicated. In the new-born, scrupulous cleanliness, with antiseptic treatment of the umbilical cord, especially where the mother is the victim of puerperal disease, may in many cases save the life of the infant. In the treatment of erysipelas the remedies that have been advised are innumerable. Each observer has seemed to be justified in extolling his favorite drug or remedial procedure by the favorable results obtained, forgetting that the natural uninfluenced course of the disease is towards recovery.

It will be readily understood that at different periods of the history of medicine the treatment of erysipelas has been influenced by the prevailing medical theories. The earliest treatment—that of Hippocrates—consisted merely in the continuous application of cold water. Afterwards venesection was applied, as in almost all other diseases. A reaction, however, set in later against blood-letting in erysipelas, and the method was abandoned.

During the time when erysipelas was looked upon as caused by a bilious condition, emetics, cathartics, and cholagogues were the prevalent remedial agents. And indeed it would be a lengthy task to enumerate all the various remedies that have been used in erysipelas. Every possible method of internal medication has been resorted to,—depurative, depleting, stimulating, sedative, tonic, and specific. Every possible external application has been utilized, from oil to caustics.

The local treatment has undergone various changes. At one time it was feared that the disease might be driven in by too energetic treatment, and simple applications only were employed, such as non-irritant powders and enveloping the part in cotton.

The external remedies that have been and are still used more or less extensively may be grouped into the following classes:

1. Such as protect from the air:
    - *a.* Powders.
    - *b.* Emollients.
    - *c.* White of egg.
    - *d.* Collodium.
2. Anodynes.
3. Antiphlogistics.
4. Astringents.
5. Stimulants.
6. Antiseptics.

1. Many different non-irritant powders have been utilized, such as rice

flour, lycopodium, potter's clay, oxide of zinc, etc. In ancient times emollient poultices derived from mucilaginous plants were frequently applied. Fats of various kinds have likewise been used, lard being employed either alone or as an excipient for various drugs. Lately Sir Dyce Duckworth has highly extolled a well-prepared mixture of lard and chalk. Glycerin has been looked upon with favor by many; and Frere praises a combination of linseed oil and white lead with which the erysipelatous skin is to be freely painted. White of egg is employed for a similar purpose, as is also water-glass, which was introduced by Piazza. Collodium is used for the purpose of excluding the air, and at the same time exerting a pressure by its contraction upon the erysipelatous tissue. It has long been looked upon with favor, and is still employed by many. One decided objection to the collodium is that it is liable to crack, and at the rough edges irritation is apt to occur. Lately a solution of iodoform in collodium has been painted over the affected part.

2. Anodynes have been used externally. The various narcotics have either been added to the poultices or applied independently. The ethereal solution of camphor and tannin, which was the favorite remedy of Trousseau, may be classed under this head. Trousseau recommends it especially in the case of the new-born.

3. Of antiphlogistics the only one that has been retained in favor is cold water, which was employed by Hippocrates. Ice bladders or cold compresses are placed upon the affected parts to allay the inflammatory process. Griscom advised glycerin for the purpose of withdrawing water from the inflamed tissues and thereby depleting them. As glycerin does not act through the epidermis, the method is as faulty as the theory. Dobson, Bright, Hutchinson, Lawrence, and others scarified the erysipelatous skin in many places for the purpose of depletion, and Lisfranc advised the use of as many as fifty leeches in order that the part might be thoroughly depleted. The method has been abandoned as useless.

4. As in other inflammations, so in erysipelas the employment of astringents readily suggested itself; but clinical observation has shown the results to be worthless. The astringents which have most frequently been used are sugar of lead, tannin, sulphate of zinc, alum, sulphate of iron, and vinegar, either alone or associated with anodynes and emollients.

5. Acting upon the idea that by setting up a counter-irritation either upon or near the erysipelatous portion a cessation of the peculiar inflammation might be brought about, various stimulant applications have been employed. Blisters have been applied to the erysipelatous skin; but their use has simply increased the distress without favorably influencing the condition of the patient. In 1829, Higginbottom advised the use of nitrate of silver. He thoroughly washed the part with a solution of potash, and then painted it and a portion of the healthy skin adjoining with a strong solution of lunar caustic, one to ten. This method, called the ectrotic, has been looked upon with great favor, and is still employed by many. Another

favorite mode of using the nitrate of silver was to paint a heavy line some distance from the margin, completely encircling the erysipelatous skin, in the hope that an insurmountable wall would thereby be erected that would stay the progress of the disease. However, the erysipelas passed over the black line as well as over the unpainted skin. For a similar purpose the hot iron was used by Larrey, and with the same negative results. It was found that immediately after a thorough application of the nitrate of silver the temperature fell, but only for a short time.

In like manner the tincture of iodine has been employed, with some good results. In order that the iodine should accomplish its best results it is necessary that its application be thorough. The parts should be painted until they are black-brown.

6. The employment of antiseptics is most in accord with our knowledge of the etiology of erysipelas. However, the results obtained have not yet been as striking as might have been expected. The reason thereof, probably, lies in the fact that the epidermis offers an impervious barrier to all non-volatile substances that do not cause a loosening of the cells, and that therefore most of these antiseptic solutions and unguents are practically useless.

Of all the antiseptics carbolic acid is the most frequently used, being applied either in solution or as a spray, as recommended by Verneuil, or hypodermically, as advised by Hueter. There is no doubt that the carbolic-acid solution is readily absorbed, as is proved by the olive-colored urine which is frequently secreted within a few hours after the commencement of the applications. The results obtained in many cases are excellent, although where the erysipelas has extended over large areas in young infants the possibility of carbolic-acid poisoning should be borne in mind. Verneuil uses a spray of a two-per-cent. solution of carbolic acid directed upon the erysipelatous skin during five minutes every hour. The method of Hueter has found great favor. He made hypodermic injections of three-per-cent. solutions of carbolic acid, and found that injections of a syringeful thereof sufficed for an area two inches square, one injection being made for each such portion of the affected part until all had been acted upon. He made as many as twelve injections at one time without producing any unfavorable symptoms. The carbolic-acid treatment is certainly rational, and has the support of experiment, for both Tillmann and Fehleisen find that cultures of the micrococci mixed with two per cent. to four per cent. of carbolic acid fail to produce the disease. Similarly to carbolic acid, corrosive sublimate has been lately employed, the solution used varying from one part in two hundred and fifty to one part in five thousand.

Formerly turpentine was highly esteemed as an external application. It has lately been recommended by Luecke, who finds a fall of temperature and a diminution of burning after each application and believes that the erysipelas passes off more rapidly. Kaczorowski advises a mixture of carbolic acid one part with turpentine ten parts. After this application lead-water compresses are used, and in severe cases ice. In this treatment the skin rapidly becomes intensely red, but the erysipelas is said to be aborted in from twenty-four to forty-eight hours. Nussbaum has lately advised the external application of one part of ichthyol with ten parts of vaseline, basing his favorable report upon five cases. Lorenz employs a still stronger preparation,—viz., two parts of ichthyol with one part each of glycerin and ether. Wilde advises hypodermic injections of an eight-per-cent. solution of sulphocarbolate of soda. Of this solution one or two syringefuls are injected. The temperature is said to fall immediately, the borders of the erysipelas to become diffused, and the process to disappear in three or four days. It is not followed by any unpleasant symptoms.

Alcoholic stimulants are of great service; and experience has taught us not to be sparing in their use. Of all internal medication, that with the tincture of iron, introduced by Hamilton Bell in 1851, has been received with the greatest favor. Those who are loudest in its praise advise large doses, employing with adults as much as one drachm every four hours and with children proportionately less. J. Lewis Smith gives four drops every three hours to a child of from one to two years. According to Balfour Campbell and others, it materially shortens the disease and mitigates the severity of the symptoms. Large doses of quinine have been used both as a specific and for the purpose of reducing the fever. For a similar purpose salicylic acid and its preparations have been used. Lately Haberkorn advises large doses of benzoate of soda, claiming that it reduces the temperature to the normal in forty-eight hours and that the redness rapidly disappears.

Bartholow uses small doses of atropine every hour, and Mavrikos recommends that five to six drops of tincture of aconite be given to infants in twenty-four hours. Pirogoff administers camphor followed by hot tea, and claims good results.

In the treatment of erysipelas nourishment is of the utmost importance, and this is especially the case with feeble children and small infants. The disease belongs to the asthenic form, and requires a supporting treatment. Large quantities of milk, with or without alcohol, should be insisted upon, and where much cannot be taken at a time frequent administration should be resorted to. The various forms of peptonized foods are to be highly recommended. Where the nourishment by the mouth is insufficient, or where it is impossible on account of obstinate vomiting, rectal alimentation is absolutely necessary. Strict attention should be paid to the dietary, as so much depends upon proper and systematic nourishment in this disease; and the importance of hygiene and careful nursing cannot be overestimated.

RHEUMATISM.

By W. B. CHEADLE, M.D., F.R.C.P.

Definition.—The term rheumatism has been used very loosely to indicate almost any affection accompanied by pain and tenderness of joints and muscles, and to include morbid conditions of widely different nature.

This is no doubt a partial survival from the Hippocratic age, when every disorder of the body was attributed to the flow of a subtle morbid essence or *virus* to the part affected. Rheum and catarrh both have origin in the same word ῥέω, and its stronger form καταῤῥέω. Eventually the two terms became differentiated, catarrh becoming restricted to affections of the mucous membranes, associated with actual flux therefrom, and rheum being applied to painful conditions of parts, also connected in the minds of the pathologists of the time with the shifting flow of acrid humor. Latterly there has been further narrowing of the scope of the term, and a tendency to restrict its use to the acute and genuine form of the disease in its different degrees of severity.

It is in this limited sense that the designation will be used here,—viz., as signifying acute rheumatism, with its subacute and chronic forms. Rheumatoid arthritis will be considered separately, as a thing apart, differing in essential characters from the genuine disease.

While, however, the term rheumatism must in this relation be taken strictly as indicating a special pathological state of which what is known as acute rheumatism or rheumatic fever is, in the case of adults at least, the sharpest and most distinctive expression, it must, on the other hand, be enlarged so as to include much more than inflammation of the joints and fibrous tissues.

A study of the disease as it is seen in children forces upon us a far wider conception. The delicate tissues and organization of a child exhibit a more extensive area of disturbance under the influence of the rheumatic state than the more stable structures of an adult. Some phenomena, unquestionably associated in childhood with the rheumatic state, such as chorea and the development of tendinous nodules, for example, are rare or altogether wanting in later life. Moreover, in the rheumatism of childhood the chief and most characteristic symptom of the disease as seen in adults—the articular affection—is often extremely slight, sometimes absent altogether. It is overshadowed or replaced by other manifestations set up by the same morbid influence which gives rise to the inflammation of the joints.

We are so accustomed to look upon rheumatism in the one aspect in which it has been most familiar to us, that it is difficult to conceive it in its larger connection. The old definition of rheumatism was founded upon observation of the disease as modified in adults. But it is quite inadequate as applied to the phenomena developed in the simpler conditions of early life.

In children more clearly than in grown persons clinical observation reveals a series of phenomena connected wholly or partially with the rheumatic state which are not limited to the joints or to fibrous tissues or serous membranes, but involve occasionally also mucous membrane and skin, and even the central nervous system itself. Erythema, tonsillitis, chorea, pleurisy, tendinous nodules, may be results of the rheumatic disturbance as certainly as articular inflammation or pericarditis. They are found associated with articular rheumatism, and when they occur alone are met with especially in rheumatic subjects. We must regard all these affections in certain instances as manifestations of the rheumatic state, although they may be set up in other instances by other causes, just as arthritis or pericarditis, while usually rheumatic, may be due to scarlatina or septic poisoning or pyæmia. They are not invariably rheumatic, but most commonly rheumatic. This series of rheumatic phenomena may occur in any order of sequence, in any combination. Any one of the phases may be absent, one only may be present, or two or three, or the whole series may be complete in the same patient. There may be articular affection alone, for example, or there may be in addition pericarditis or endocarditis, or these may occur without any affection of the joints, or with chorea and tendinous nodules, or there may be erythema or tonsillitis instead of any of these, or in addition to them. This is seen constantly in clinical experience. The different manifestations, again, may occur not only in any order and combination, but separated by varying intervals of time, following one another in quick succession, or some appearing months or years after the rest. Thus, an endocarditis or a pericarditis or a chorea may occur first and alone, the joint-affection long after. The various manifestations of rheumatism massed together in the case of adults tend to become isolated in the case of children, so that the whole phenomena are distributed over years instead of weeks or months, and the history of a rheumatism may be the history of a whole childhood. It has been said that affection of the joints is frequently not a marked symptom in the rheumatism of childhood, and that it is sometimes altogether wanting. It cannot, therefore, be regarded as essentially typical or characteristic in early life. If any one of the rheumatic series is to be regarded as fairly constant and representative, it is perhaps endocarditis. In childhood arthritis is at its minimum, endocarditis at its maximum.

It has been hitherto customary to speak of all manifestations of rheumatism outside and beyond the joint-affection as complications: in childhood they do not centre round the articular affection, and when the joint-affection is slight or wanting or has occurred apart, before or after, the endocarditis, or pericarditis, or pleurisy, cannot well be looked upon as secondary and dependent upon it. These inflammations are in truth just as much direct results of the rheumatic virus or disturbance, whatever its nature, as the articular inflammation, and any satisfactory definition of the disease must be broad enough to cover them. Rheumatism may be defined, then, as a general or constitutional morbid state, characterized by shifting inflammation of the fibrous structures, especially those of joints and serous membranes; this involves sometimes other tissues, such as the subcutaneous connective tissue, the skin, and the mucous membrane, and oftentimes causes also in children disorder of the central nervous system, as evidenced by the emotional and motor phenomena of chorea.

Etiology.—The common and immediate cause of rheumatism is chilling of the surface of the body. Exposure to cold is most effective when the body has previously been heated by exercise or by sitting in a hot room and the skin is perspiring and its vessels relaxed. The surface thus cools more rapidly through the evaporation of perspiration and the exposure of a larger proportion of blood in the dilated cutaneous vessels to the cooling process. Under these circumstances a draught of cold air, or remaining in damp clothes, is a frequent exciting cause of rheumatism; but it may also be induced without previous overheating, by prolonged exposure to any cooling influence, as a damp bed, or wet clothes, or an east wind. In the case of children these sources of chill are especially frequent: a child grows hot and perspiring with romping and games of play, and stands about indifferent to the dangers of wet feet and currents of cold air.

Muscular exercise has indeed been credited with the power of exciting an attack of rheumatism; and Dr. Sibson suggested that those joints were most liable to be affected which are most employed in habitual work. Rheumatism has been observed to be common among artisans who follow heavy labor, and to be liable to come on after long marches. Yet this probably means nothing more than that these conditions naturally favor heating and chilling of the body, and also by producing fatigue or exhaustion so lower the circulation as to aid the production of congestions. The formation of lactic acid in muscular action may be another factor. Although children do not commonly follow occupations which are physically laborious, their naturally active habits would tend to produce conditions of excited and then enfeebled circulation similar to those which result from the more severe muscular exertion of adults. The view that chill is the chief exciting cause of acute rheumatism is supported by evidence which goes to show that the disease is most prevalent in cold and temperate climates and in the spring and winter seasons, although by no means uncommon in warm weather or hot climates, where overheating is readily produced and subsequent chill liable to follow.

There appears to be one other direct exciting cause of the rheumatic state in the case of children,—viz., the poison of scarlatina. It is well known that affection of the joints not to be distinguished from that of ordinary rheumatism occurs occasionally in the course of scarlet fever, and that this scarlatinal rheumatism is liable to be complicated by endocarditis and pericarditis. Dr. Ashby[1] met with twelve instances of this rheumatism out of five hundred cases of scarlatina. Its occurrence may mean merely that the scarlatinal virus has a similar effect upon fibrous tissues and serous membranes to that of rheumatism, and the connection of tonsillitis with the two affections would favor this view; or it may be that the development of the true rheumatic virus is favored by scarlet fever; or, again, the inflammation may be due to septic poisoning from foul discharges of ears or throat. In some instances the endocarditis or pericarditis set up in the course of scarlet fever may be caused by the uræmic poison from nephritis. Dr. West[2] states that in fifteen cases in which he noted the supervention of cardiac inflammation in connection with scarlet fever,—viz., ten of endocarditis, three of pericarditis, and two of both combined,—the symptoms did not manifest themselves until the stage of desquamation, and were accompanied by anasarca. No mention, however, is made of the condition of the urine, or of the throat or ears. Yet endocarditis and pericarditis occur also in the early stages of scarlatina concurrently with joint-affection undistinguishable from that of acute articular rheumatism. Several such cases have come under my own observation. Henoch[3] records one in which acute arthritis appeared in the first week of scarlet fever, accompanied by chorea and mitral murmur. Dr. Barlow[4] states that he has seen well-marked arthritic symptoms on the third day. In Dr. Ashby's cases, previously alluded to,[5] the joint-symptoms came on with great regularity at the end of the first week. He holds that the arthritis is of septic or pyæmic origin, due to foul throat, or otitis, or empyema. The septic poison does undoubtedly produce arthritis on occasion, and septic poisoning not unfrequently arises from the causes mentioned in the course of scarlatina. But such septic condition does not arise until later in the course of the disease. Moreover, articular or cardiac inflammation is not found to be specially associated with foul ulceration of the throat or with otitis, and other signs of septicæmia are wanting. Those cases of rheumatism coming early in scarlet fever, then, are probably not septic, a condition which does not usually arise till much later. When arthritis occurs late, it may be in some cases of septic origin. The uræmic poison does not cause arthritis, but it may in certain cases cause an endocarditis or pericarditis coming late in the course of the specific fever. On the whole, it seems clear that either genuine acute rheumatism does arise in the course of scarlet fever, or else that the scarlatinal virus occasionally produces an inflammation of fibrous tissues and serous membranes closely corresponding with that set up by the rheumatic poison.

---

[1] Brit. Med. Jour., Sept. 15, 1883, p. 514.

[2] Diseases of Infancy and Childhood, 7th ed., p. 554.

[3] Diseases of Children, Eng. trans., p. 80.

[4] Brit. Med. Jour., Sept. 15, 1883, p. 509.

[5] Brit. Med. Jour., Sept. 15, 1883, pp. 514, 515.

Rheumatism has been attributed to the entrance of micrococci into the blood, to that of a specific vegetable organism, the Zymotosis translucens of Salisbury, and to a miasm analogous to that of malarial fevers. But these are theories which have not so far been confirmed and established. Two conditions which undoubtedly favor the occurrence of rheumatism in the adult—viz., childbirth and the existence of gonorrhœa—are not, of course, concerned in its causation during the period of childhood; and the only three active influences which can be accepted as directly determining the development of acute rheumatism are chill, perhaps excessive muscular exercise, and scarlet fever.

One of the most certain predisposing causes appears to be hereditary tendency. Rheumatism runs remarkably in families. It is passed on from parents to offspring as strongly as the tendency to gout or to tuberculosis. Statistics show that in a large proportion of cases of rheumatism there is a history of similar affection in near blood-relations. The percentage varies from twenty-five to sixty, according to the minuteness of the inquiry and the degree of joint-affection which is regarded as sufficiently distinctive of an attack of acute rheumatism. My own statistics yield forty per cent., Dr. Goodhart's about sixty per cent., in the case of children. Whatever may be the exact proportion, the fact that children who have a family history of acute rheumatism in immediate blood-relations are especially liable to acute rheumatism is indisputable.

Out of 492 cases of all kinds, both medical and surgical, among the in-patients in the Children's Hospital at Great Ormond Street, in which the history with regard to the occurrence of acute rheumatism in the family was carefully inquired into, it was found that there was a clear history of its previous occurrence in near blood-relations in 173. Of these 173 with rheumatic family taint, 38, or 20.2 per cent., developed unquestionable rheumatism. If chorea and the minor rheumatic manifestations were admitted as evidence, the proportion would be considerably higher. Taking next the remaining 319 of the gross number of cases, medical and surgical, where there is no history of rheumatism in the family, 15 only have developed rheumatism,—*i.e.*, 4.7 per cent.

It would appear, therefore, that with a family history of acute rheumatism in immediate blood-relations, especially in father and mother, the chance of any child with such hereditary predisposition contracting acute rheumatism is very nearly five times as great as that of a child whose family history is clear of such taint,—strong evidence of the influence of inherited predisposition. Sometimes several members of a family which inherits the rheumatic diathesis develop acute rheumatism.

Any number of examples might be quoted in illustration of the remarkable influence of hereditary predisposition.

A. R., a girl of fifteen, was admitted to St. Mary's Hospital, May, 1880, for extreme mitral disease of the heart. She had had rheumatic fever four times. Her father had had rheumatic fever; her mother had also had rheumatic fever.

Again: F. H., a girl of seventeen, was admitted into St. Mary's Hospital with a fourth attack of chorea. One month before this last attack she had acute rheumatism of the joints for the first time. There was no sign of heart-affection. The history of family predisposition was strong. Her mother had had rheumatic fever. One of her brothers had been a patient in St. Mary's Hospital with rheumatic fever, again with mitral disease of the heart, and after that with chorea. Another brother had chorea, but no affection of the joints.

A third example may be given, illustrating the persistence of the rheumatic taint through three generations. J. P., a girl of eleven, was admitted to hospital with a third attack of chorea. She had never had rheumatic arthritis, and there were no signs of heart-affection. Her mother had had rheumatic fever three years previously; her father had also had rheumatic fever, and died of resultant heart-disease at the age of thirty. The maternal grandfather and grandmother had both had rheumatic fever.

Dr. Goodhart[1] records a remarkable instance of heredity and constitutional predisposition. Five out of the six children of parents both of whom were rheumatic had either joint-affection or heart-disease. All the children were under fifteen, and the only one unaffected was a baby of fourteen months. The eldest, a boy of fourteen, had rheumatic fever twice, and mitral regurgitation; a second boy of ten, the same; a third child, a girl, died of mitral disease; the fourth, a girl, had rheumatic fever after scarlatina, followed by mitral disease; the fifth, a boy of four, was laid by all one winter with joint-rheumatism.

Steiner[2] relates a still more extreme instance, where of the twelve children of a rheumatic mother eleven had rheumatism before the age of twenty.

It would seem that the tendency to the disease is intensified by inheritance from both parents instead of one only; and this double inheritance also appears to favor the development of the disease in its severer and more persistent forms: this is perhaps what might reasonably be expected.

It might no doubt be contended that the occurrence of rheumatism in several members of a family is due to the influence of climate and locality rather than to inherited constitution. A careful inquiry into this point, however, has shown that this is not so. Individuals of the same rheumatic family contract the disease when widely separated in different places and under different sanitary conditions, and change of residence appears to have small influence on the result.

---

[1] Guy's Hospital Reports, vol. xxv. p. 106.

[2] Steiner, Diseases of Children, Eng. trans., p. 336.

These facts of inheritance show the importance of a minute investigation of the patient's life-history, and taint of the family on both sides, in order to obtain a complete survey of the phenomena and relations of the rheumatic state.

Temperament.—Whether the rheumatic diathesis is related to any special type of bodily conformation or temperament, as Dr. Laycock held, is extremely doubtful. Mr. Jonathan Hutchinson suggests the existence of a special arthritic diathesis common to rheumatism, gout, and arthritis deformans, but he does not connect it, as far as I know, with any special bodily form or appearance. Any one who sees rheumatism on a large scale would, I think, confess that it occurs in persons of every variety of build and complexion, both in robust and delicate, dark and light. In children it is often associated with a pale complexion, but that is due to the anæmia which is so marked a feature of the disease, particularly in early life.

Sex has a marked influence in predisposing to acute rheumatism. It is more frequent in men than in women, owing probably to the greater exposure of the former to vicissitudes of weather and to laborious physical exertion. The statistics of the Collective Investigation Committee of the British Medical Association yield in 655 cases, including all ages, but chiefly adults,[1] 57.25 per cent. of males and 43.75 per cent. of females. In children, however, the proportions are reversed: acute rheumatism is more common in girls than in boys. The statistics just referred to give 26 girls to 25 boys under twelve; but the numbers are too small to give reliable results. Dr. Goodhart found in 68 cases 42 girls and 26 boys, or nearly two to one; but here again the numbers are not large. The records of the Children's Hospital in Great Ormond Street[2] for sixteen years give 252 girls to 226 boys. My own experience shows it to be most common in girls, and this is supported by that of Drs. Meigs and Pepper.[3] The greater liability of females appears to continue up to twenty; after that age males preponderate.

Age.—Rheumatism is extremely common in early life, far more common than is generally supposed, owing to the slight development of the joint-symptoms causing it to be frequently overlooked. Yet cases of rheumatism are plentiful in the wards of the Children's Hospital, the beds are largely filled with them there, and in patients of the better class met with in private practice they are by no means rare.

Rheumatism is met with occasionally in early infancy, and it becomes more and more frequent, and the joint-affection more pronounced, as each year of life passes, up to six or seven, continuing from this period up to puberty at about the same relative frequency. Henoch[4] records a case in a child ten months old, in which there was well-marked acute articular rheumatism, accompanied by broncho-pneumonia and probably by pleurisy. Senator[1] refers to two other cases in which the patients were still younger,—one recorded by Stäger in a child four weeks old, and another recorded by Windeshofer in which the patient was an infant of twenty-three days only. I have never met with an undoubted instance of rheumatism in a child under two years, but I have seen several cases of tenderness of joints and tendons in children a few weeks or months old which were probably, although not certainly, rheumatic.

---

[1] Brit. Med. Jour., Feb. 25, 1888, p. 387.

[2] Tuckwell, St. Barth. Hosp. Rep., vol. v. p. 102.

[3] Diseases of Children, 7th ed., p. 668.

[4] Diseases of Children, p. 311.

Pathology and Pathological Anatomy.—The exact method in which chill acts in producing rheumatism is extremely obscure. Several hypotheses more or less plausible have been proposed in explanation. Of these one of the most favored is that it is due to the accumulation of lactic acid in the blood, as originally suggested by Dr. Prout and supported by Todd. The lactic acid is supposed to act as a direct irritant to the tissues, as the urate of soda in gout. In support of this theory there are certain experiments by Dr. B. W. Richardson, who claimed to have produced endocarditis in dogs and cats by injection of lactic acid into the peritoneal cavity. But the production of endocarditis without distinctive articular inflammation would not be conclusive as to the rheumatic nature of the morbid state induced, and it was afterwards shown by Reyher that in dogs the cardiac valves frequently exhibited similar appearances apart from any artificial interference of poisonous injection. Sir Walter Foster[2] related two cases in which the administration of lactic acid for diabetes in doses of from fifteen to seventy-five minims was followed by attacks which closely resembled acute articular rheumatism. I have repeated the experiment more than once, with negative results, and this has been the case, I believe, with all other observers, except in one instance recorded by Kuelz,[3] in which pains in the hip and thigh followed the administration of the drug. By some—as Senator, for example—it has been supposed that the lactic acid produces its effect by its action upon the central nervous system, and that the disordered nerve-centres react upon the joints, causing pain, fever, and trophic changes. Another theory was that held by the late Dr. Fuller,—viz., that chill disturbs the nervous system, that nutrition is thus disturbed likewise, and that lactic acid or some other acid of irritant character is retained and acts as a poison, which produces the phenomena of acute rheumatism. The tendency to thrombosis which is a notable feature of the disease may be partly due to diminished alkalinity of the blood from this cause.

In speaking of the etiology of the disease, mention was made of the theory of the dependence of acute rheumatism upon the presence of micrococci, or a specific fungoid germ, or a miasmatic poison. In further explanation of this it has been suggested that the entrance of poison in this way sets up endocarditis, and that the joint-symptoms are merely the result of multiple minute embolisms. It seems to be a sufficient answer to this that endocarditis may be entirely absent, and therefore the material for multiple embolisms wanting, and further that when endocarditis does occur it is usually sequent to the arthritis,—not antecedent. Moreover, the existence of such embolism has not, as far as I know, yet been demonstrated. Multiple thromboses might more reasonably be regarded as possible causes, since the blood has undoubtedly a special tendency to coagulate in the rheumatic state; but here again we have only pure hypothesis. As far as present knowledge goes, the most plausible theory is that the normal elimination of some metabolic product is interfered with by the action of cold, which either arrests its excretion by the skin or checks its destruction by oxidation; that poisonous matter thus accumulates and acts as an irritant to the joints, serous membranes, and other tissues. It is possible that the peccant matter may be lactic acid, which is always formed as paralactic acid by muscular action; although it is doubtful whether this is normally secreted by the skin, as Berzelius taught. It has been found in the perspiration in puerperal fever, and in the urine; yet its existence in excess in the blood, or arrest of excretion, has not yet been proved in acute rheumatism.

---

[1] Ziemssen's Handbuch, vol. xvi. p. 18.

[2] Brit. Med. Jour., Dec. 21, 1871.

[3] Quoted by Senator in Ziemssen's Handbuch, vol. xvi. p. 28.

The existence of micrococci and bacilli in the blood and serous effusions in rheumatism has been lately shown by Dr. Mantle,[1] but the specific nature of these organisms has not yet been demonstrated. The tendency to the formation of thromboses, which is a feature of acute rheumatism, and which has already been alluded to in passing, is due partly to the hyperinosis which exists, partly perhaps to the lessened alkalinity of the blood from the neutralization of some of its alkali by lactic or other acid or by the presence of bacteria; but it is also in some measure dependent upon obstruction to the circulation resulting from heart-lesion,—mitral disease or pericarditis. Thrombosis not unfrequently occurs in a large vein, and sometimes in the cavities of the heart. I have seen this happen in the axillary vein, in both iliac veins, in the femoral vein, and in the right auricle. In two cases it was followed by fatal embolic pneumonia. Possibly the hemorrhages of rheumatic purpura may be due to capillary thromboses originating in this way.

Morbid Anatomy.—The changes found in the joints post mortem are often slight; and acute rheumatism is so rarely directly fatal that the observations made have been comparatively few. The synovial membranes are hyperæmic, and there is more or less excessive effusion of fluid into the cavities of the joints and into the tissues around. The fluid in the joints may be ordinary synovia, or be diluted by serum and contain blood-disks and pus-cells. Sometimes minute hemorrhages can be seen,—especially in the vascular portion of the membrane where it joins the cartilages. There may be in rare cases suppuration, or ulceration of the cartilages. The latter show signs of inflammatory change in globular swelling and proliferation of the cells in certain portions. In the case of children, according to Henoch, the parts around may become infiltrated with inflammatory lymph, and even assume a bony hardness, and actual exostoses may be found. In children too often—more rarely in adults—the tendons and subcutaneous connective tissues are studded with fibrous nodules from the size of a hempseed to that of an almond or even larger, which were first described by Dr. Barlow and Dr. Warner, and which appear to be so closely connected with a similar condition of the cardiac valves. They occur chiefly in the neighborhood of joints or tendons, and in the aponeuroses and fascia. Microscopically they exhibit all the characters of newly-developed fibrous connective tissue. The skin may show the remains of eruption, such as erythema or peliosis rheumatica. The throat may exhibit the redness and swelling of acute tonsillitis. But in fatal cases of rheumatism the chief interest centres upon the heart, and next to this upon the pleuræ and the lungs. Endocarditis and pericarditis are of course commonly the grave lesions of acute rheumatism, and pleurisy and pneumonia are occasionally present. The morbid anatomy of these changes is fully given elsewhere in the articles on these diseases. There is, however, one condition connected with the state of the heart and its appendages which deserves mention here, as being especially prominent in the rheumatic inflammation of childhood. I mean the extension of pericarditis to the connective tissue of the anterior mediastinum. This results eventually in the matting together and enormous thickening of these parts, which become converted into a dense fibrous mass beneath the sternum. The tendency in childhood is to plastic exudation leading to adhesion and fibrous thickening rather than to effusion of serous fluid. The extreme distention of the pericardial sac met chiefly in later youth is unknown in early childhood. This is probably connected with the more subacute and recurrent character of the inflammation. Purulent effusion, again, seems to be as uncommon in children in the case of the pericardium as it is common in the pleura. Another morbid appearance met with in children as well as in adults is the existence of large fibrinous coagula in the heart and great vessels, previously mentioned, and both ante mortem and post mortem in date.

---

[1] British Medical Journal, June 25, 1887, p. 1381.

Symptoms.—In defining the meaning of the term rheumatism it was pointed out that a study of the disease in children leads to a broader conception of its nature and compels the inclusion within its scope of many morbid affections in addition to the arthritis. This, the most constant and striking feature of the complaint in adults, in childhood sinks into comparative insignificance, and is often entirely absent in an attack which is undoubtedly essentially one of acute rheumatism. Moreover, many of the phases or manifestations of rheumatism which, viewing the disease from the adult stand-point, we are accustomed to regard as complications or sequelæ of a central joint-affection, appear in childhood as initial or chief phenomena. Arthritis is at its minimum, endocarditis at its maximum. Endocarditis or pericarditis may appear first, or pleurisy, or chorea, or tonsillitis, or nodules, or an erythema, or an arthritis, and these may be grouped in any order, in any number, separated by varying intervals of time. In early childhood, as Dr. Barlow has well remarked, the tendency is to isolation and separation of the phenomena. These draw more closely together as time passes on; the disease tends to appear as a whole, instead of in disjointed parts; some features become accentuated, as the joint-affection, others grow less constant and conspicuous with advancing age, as the tendinous nodules and chorea, and these finally disappear, except in rare instances, with the advent of adult life.

It will be impossible to picture the disease in all the protean forms afforded by varying combinations of the different phenomena. The most useful plan will be to consider each manifestation and symptom apart, to point out any special features which attach to them in early life, and then to give some of the more common combinations which occur in the rheumatism of childhood.

Arthritis.—The comparative slightness of this symptom in the case of children has already been insisted upon,—in some instances a little tenderness and swelling of knees or ankles or wrists, possibly limited to a single joint, or even less than this,—mere stiffness and tenderness on movement, or even a slight feverish attack only, recognized afterwards as rheumatic by the light of developing heart-disease.

The following example illustrates this form. M. T., a girl three years old, was observed to be ailing and feverish without definite signs of any precise ailment. Two days later the great toe of one foot became red, swollen, and tender; no other joints were affected, and it was supposed at first to be merely chilblain. Two days later still, both ankles were tender and very slightly swollen. The temperature was found to be 102.5°. The condition was now judged to be rheumatic, and the heart was examined. A full blowing mitral murmur was found to exist, which persisted for many weeks. The joint-affection quickly disappeared with rest and salicin treatment, and eventually, after many weeks, the mitral murmur finally disappeared also.

In other cases, again, the rheumatic inflammation is limited to tendons or their sheaths, as in stiff neck, which is occasionally the only manifestation of genuine rheumatism. In some instances this gives rise to prolonged torticollis, as in a case recently observed, where the rheumatic nature of the affection was shown by previous arthritis of the knees, and there was likewise mitral murmur and tonsillitis. One of the most misleading manifestations of rheumatic joint- or tendon-affection is when it is limited to stiffness of the hamstring tendons at the back of the knee.

The following cases illustrate this form of rheumatic arthritis. A little girl four years old had difficulty in putting down the heel of the right foot.

The case was supposed to be a surgical one of incipient talipes varus, and the limb was steadily galvanized. No improvement following, the patient was referred to me for medical treatment. There was no deformity, but the disinclination to walk was extreme: the heel could be put to the ground, but the knee was kept bent. On examination, both knees were found to be tender, especially at the back in the hamstring tendons, and they were slightly swollen. The temperature was 100°. It was further ascertained that the child had suffered from pain and stiffness of both knees and ankles from time to time for the last six months. There was no cardiac murmur, nor other sign of rheumatism. But the mother had had rheumatic fever, and the condition was judged to be a rheumatic arthritis. Under salicin and citrate of potash the stiffness and retraction of the heel, which had lasted for weeks previously, entirely disappeared in a day or two, and the child walked perfectly.

In a parallel instance the tenderness and stiffness of the hamstring tendons came on during the night, and the child, a boy of five, on getting out of bed in the morning could walk only on the tips of his toes. The temperature was 103°. The heart was free, the only other sign of rheumatism being attacks of tonsillitis. But the history of inheritance was strong. The mother had rheumatic fever with pericarditis, and was found to have a loud mitral murmur. The child's cousin on the mother's side had had rheumatic fever and endocarditis. A cousin on the father's side had had nearly the whole rheumatic series,—viz., acute rheumatism, tonsillitis, chorea, purpuric erythema. Salicin and citrate of potash were given, and in three days all sign of stiffness had disappeared and the temperature was normal. A similar case was seen in a boy of eleven, with advanced mitral disease, but no history of rheumatic arthritis. It got well in like manner. There could be no doubt that it was rheumatic; and I have seen other examples of this rheumatic tenderness and stiffness of the hamstring tendons, causing walking on tiptoe with bent knees, which seems almost diagnostic.

Often, however, the joint-symptoms are sufficiently marked to attract attention, and both wrists, knees, and ankles, and possibly fingers, present the typical appearance of acute articular rheumatism. The older the child, the more nearly does the affection conform to the adult type in this respect. I have rarely seen a little child bound hand and foot with rheumatic inflammation of the joints, unable to move, as in a typical case of rheumatic fever in an adult. They are stiff and tender, but the patient usually moves about. Often the joint-symptoms subside in a day or two, or there may be a succession of relapses in almost continuous series. The articular affection shifts perhaps from one joint to another, and tends to change to the subacute form and to relapse.

The temperature, again, differs in its range and course from that observed in the acute rheumatism of grown persons. It seldom runs high, rarely above 102° to 103°, except in the older children,—more often 100° to 101°, and this febrile rise lasting only for a few days. This is the more notable because it is at variance with the general rise of temperatures in childhood, which tend to be more easily raised and to range higher than in later life. And, as anything like high temperature is rare, fatal hyperpyrexia is unknown, and one element of immediate danger is wanting. The pulse-rate, unless there be accompanying carditis, is but slightly raised, in accordance with the temperature.

The tongue is seldom much coated or dry, except in severe cases, or unless so-called complications are present causing increased fever and constitutional disturbance. The thickly-furred or dry brown tongue met with in severe cases of rheumatic fever is never seen in the child. The urine is hardly affected in the milder cases, but when the articular affection is severe and fever high it becomes darker, acrid, and lithatic.

Another symptom, which is so marked in the acute rheumatism of adults as to form one of the diagnostic signs, is wanting in the case of children,—viz., the profuse acid perspiration. The sweating is very slight; the patient is never seen bathed in moisture soaking night-dress and pillow; and it is not sour-smelling. The sweat-eruptions sudamina and miliaria, like the perspiration which produces them, are also wanting in young children.

Heart-Disease, Endocarditis.—In the rheumatism of childhood heart-disease plays the most prominent part. Endocarditis appears with the joint-affection in the majority of cases, and a small proportion of children only escape it: if arthritis, then almost certainly endocarditis.[1] But often this appears alone, the sole expression at the moment of the rheumatic state, or it is accompanied by the eruption of subcutaneous nodules so intimately associated with the evolution of valvulitis in early life, or by chorea or erythema. As I have pointed out, endocarditis is constantly overlooked, because the significant joint-affection is slight or wanting: the child is a little wasted and feverish, but there is nothing to call attention to the heart, and thus an insidious inflammation of the valves goes on, and is probably not discovered until long after, when hypertrophy and dilatation and loud murmur proclaim its existence.

As a rule, the endocarditis is subacute, and it is frequently protracted and relapsing; it dies down and revives again. It attacks chiefly the mitral valve, but now and again the aortic valves suffer, and in exceptional cases they are alone affected. The first sign, and sometimes the only sign, of the valvular inflammation is a soft blowing murmur, usually systolic, at the apex. This may gradually disappear after a few weeks, or more often may increase rapidly in distinctness, so as to become loud and harsh in the course of a few days. Yet sometimes the murmur, even when mitral, may be functional, due to temporary relaxation of papillary muscles and consequent imperfect closure and leakage; and this may disappear as strength and muscular tone return. Yet the fact that such murmurs appear, as a rule, in the earliest stage of the rheumatic attack, before serious debility of the cardiac muscle is likely to have occurred, points to its being due to valvular inflammation rather than to functional disturbance from paresis; and the disappearance of the murmur should be referred to resolution of the inflammatory process and restoration of the valve to its normal state. A distinct mitral murmur is usually organic, indicative of endocarditis, and commonly persistent. An aortic obstructive murmur is in like manner an almost certain indication of endocarditis. An aortic regurgitant murmur is organic without exception.

---

[1] The Collective Investigation Statistics give in males 72 per cent. of heart-affection in children, as compared with 46 per cent. in adults. In females the difference is much less.

Another cardiac sign indicative of the development of endocarditis and especially prominent in childhood is a reduplication of the second sound, audible not at the base,—as in the doubling from increased resistance in one arterial system as against the other met with in the systemic obstruction of Bright's disease on the one hand and in pulmonary obstruction on the other,—but at the apex only. This reduplication is sometimes accompanied by a diastolic murmur after the second of the two parts of the double sound. These signs may disappear; or more often they increase in intensity and gradually develop into the presystolic rumble. They are, indeed, the first sign of mitral stenosis, and certain indications of the supervention of endocarditis. In spite of the development of these signs of valvulitis going on, there may be no rise of temperature, no quickening of pulse, no distress, sometimes a pyrexia of one or two degrees, and some excitement and quickening of the heart's action. But the only certain sign of the commencement of endocarditis is that afforded by changes in the heart's sounds.

Pericarditis.—It is stated that pericarditis is less common in the rheumatism of children than in adults: it is in reality quite as frequent, its occurrence being often overlooked. This is due partly, as pointed out with regard to endocarditis, to the slightness of the associated articular symptoms and to its occasional occurrence entirely apart from them, and partly to the inflammation being generally subacute with slightly-marked symptoms which do not command attention. Pericarditis is met with in very young children even, although less frequently than in later childhood. Dr. West[1] records a case in a child of seven months, with post-mortem evidence of a previous attack at the age of four months. It is unnecessary to give a detailed account of this phase of rheumatism, since the whole subject of pericarditis is treated fully elsewhere.

There are, however, certain special features connected with pericarditis as it occurs in connection with the rheumatism of childhood which should have brief mention here.

In the first place, it is, I think, less liable to occur in the primary attack of articular affection, and also, like endocarditis, although it is at times extremely acute, this is comparatively rare; and it has a characteristic tendency to become subacute, chronic, and intermittent, to smoulder on and then become active again, with the advent, perhaps, of a fresh wave of joint-affection, or a fresh eruption of fibrous nodules, or the supervention of chorea.

---

[1] Diseases of Infancy and Childhood, 7th ed., pp. 556, 557.

Pericarditis, again, although usually associated with joint-affection, may be the first and only sign of the rheumatic state at the time of its occurrence, and be followed by arthritis or other phases of rheumatism at varying intervals; or it may be the last of the series of rheumatic events. Although not rare in the early period of this disease, it is most common, or at any rate most often observed, when the heart has already become greatly enlarged by hypertrophy and dilatation; and it is then most liable to set up fever and palpitation, with excited, turbulent, irregular action of the heart and quick pulse, sometimes excessively so,—from one hundred and twenty to one hundred and sixty even,—with cardiac pain, dyspnœa, restlessness, and distress. Very possibly, however, there may have been pericarditis before; it does not leave a record of its presence behind it, like endocarditis. This late pericarditis not infrequently is the immediate cause of death at last.

In the early attacks, however, the general symptoms are usually limited, except in the rarer acute cases, to slight fever, with a moderately accelerated pulse and respiration.

The physical signs of pericarditis are the same as in adults,—friction heard over the præcordia, followed by dulness, increased in intensity and extended according to the amount of fluid or lymph effused, and subsequent distention and thickening of the pericardium. This is sometimes considerable when the inflammation has been repeated or persistent and extended to the connective tissue of the anterior mediastinum, causing the enormous thickening previously alluded to in describing the morbid anatomy.

With this increase of dulness there is also some muffling of the heart's sounds over the central portion of the cardiac area, simulating that produced by effusion. From this it may be distinguished, however, partly by the less distinctly triangular shape of the dulness-area, but chiefly by the fact that the apex is not displaced upwards, as in serous effusion, and the heart's sounds at this point are comparatively sharp and clear. But as with general symptoms, however, so with physical signs: they are, as a rule, not pronounced in the primary attack. Instead of the marked changes described above, there is merely the double friction-sound, lasting for a limited period, and disappearing as adhesion takes place, to be renewed, perhaps, and lead eventually to the more marked changes of the later stage.

Pleurisy and Pneumonia.—These stand next to the affections of the heart in gravity and importance. They are much less frequent, however, and it is doubtful whether pneumonia can claim to be considered a certain phase of rheumatism. It occurs chiefly in three connections,—viz., in a limited form as an accompaniment of pleurisy, in more extensive degree in relation to, and probably largely dependent upon, mitral disease of the heart and pericarditis, and in the embolic form also in connection with valvular disease. In the lobar variety associated with mitral disease it is almost always on the left side.

Pleurisy, however, undoubtedly appears as a distinct expression of rheumatism: Lebert found it in ten per cent. of his cases. Like pneumonia, it is most common on the left side, and frequently associated with pericarditis. In the latter case it may be secondary, but when it occurs alone in rheumatic subjects, or as one member of a series of rheumatic phenomena, it is probably a direct expression of rheumatism. This is well illustrated by the following case. A boy of six, who had been exposed to chill while travelling in severe weather, developed pleurisy of the left side, with some local pneumonia on the fourth day. There was high fever, and it was noted that it was accompanied by profuse sweating. On the seventh day swelling, stiffness, tenderness, and pain appeared in all the joints except the fingers and toes, and he went through a well-marked attack of articular rheumatism. The stitch of pleurisy is often referred erroneously to intercostal rheumatism or pleurodynia. Pain in the side should never be passed over with a hasty diagnosis of this kind, but be the subject of careful examination with the stethoscope. The general symptoms of pneumonia occurring in the course of rheumatism are usually only rise of temperature, to 103° or 104° perhaps, and somewhat accelerated respiration. There is little or no cough, no characteristic sputum, even in the case of adults,—nothing to call attention specially to the state of the lungs. So that pneumonia is frequently only discovered accidentally on routine examination; and, as auscultation of the posterior portion of the chest is often omitted in rheumatism on account of the pain which it inflicts, the existence of the inflammation of the lung is very liable to escape recognition. The physical signs differ somewhat from those of ordinary pneumonia. There is bronchial or tubular breathing, but fine crepitation is not commonly found. This, however, is usually present in the limited embolic form.

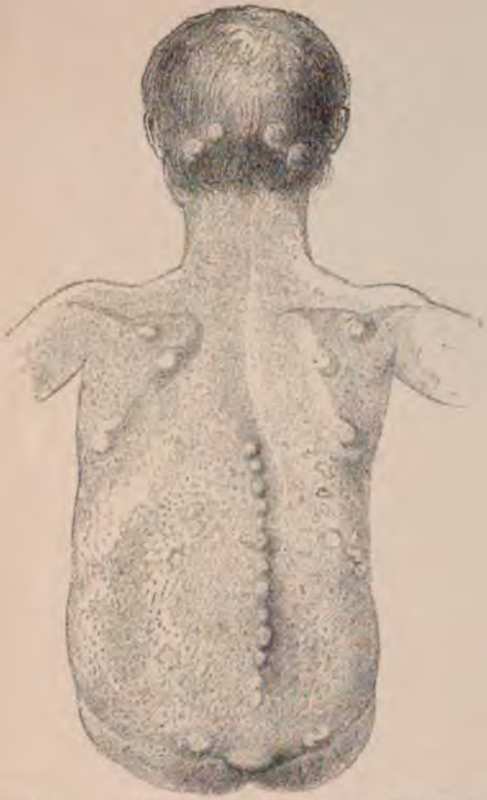
Pleurisy and pneumonia occurring as simple inflammations excited by the rheumatic virus usually resolve readily, and fluid effused as a result of the former is reabsorbed, unless, as in some cases, it becomes purulent. But when dependent upon heart-disease it is different: the pneumonic consolidation and pleuritic effusion are liable to remain, or disappear only after a lengthened period.

Bronchitis is a less frequent symptom, but it occurs, according to Lebert, in nine per cent. of cases.

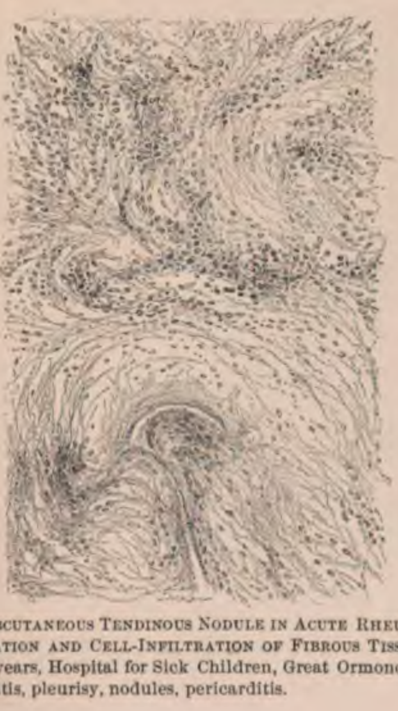
Tonsillitis.—There can be no doubt, I think, that children who are prone to articular rheumatism are prone also to tonsillitis,—that it often ushers in an attack of articular rheumatism, or occurs during its course. Trousseau recognized a rheumatic sore throat. The statistics of the Collective Investigation Committee[1] show that tonsillitis occurred as an antecedent to acute

---

[1] Coll. Inv. Record, vol. iv., 1888, p. 71.



RHEUMATIC NODULES, ERYTHEMA, CHOREA, DOUBLE MITRAL MURMUR, ARTHRITIS.—W. S., æt. four years and three months, Hospital for Sick Children, Great Ormond Street, under the care of Dr. Cheadle, December 10, 1887.



SECTION OF SUBCUTANEOUS TENDINOUS NODULE IN ACUTE RHEUMATISM, SHOWING ACTIVE PROLIFERATION AND CELL-INFILTRATION OF FIBROUS TISSUE.—John T., æt. seven and a half years, Hospital for Sick Children, Great Ormond Street. Chorea, arthritis, endocarditis, pleurisy, nodules, pericarditis.

articular rheumatism in 24.12 per cent. of cases, with 10 per cent. of sore throat of uncertain nature. This only gives instances in which tonsillitis came first in the rheumatic series; and its full significance is only realized when we consider that the throat-affection occurs also as a later as well as an initial affection, although not so frequently, and that it occurs apart from articular symptoms in rheumatic subjects. I have within the last six months seen two cases in children in which tonsillitis followed immediately after articular rheumatism, endocarditis, and chorea, and one in which it followed articular rheumatism and endocarditis; coming in each instance last in the series. In another case repeated attacks of tonsillitis extending over several years followed an attack of acute articular rheumatism which never recurred, but which was succeeded by chorea and purpuric erythema. So that there can be no hesitation in accepting tonsillitis as a genuine member of the rheumatic series. The tonsillitis presents no special features: it is accompanied by sharp fever, with a temperature of 102° to 103°. The inflammation extends to the pharynx and soft palate not unfrequently, but rarely results in either suppuration or ulceration.

Fibrous Nodules.—Dr. Barlow and Dr. Warner[1] have drawn attention to the development of fibrous nodules in the subcutaneous tissue in connection with rheumatism. The existence of these nodules had been previously noted by Dr. Hillier and certain German and French observers, and I had also seen them, but entirely failed to appreciate their frequency and their great pathological importance. They are extremely common in children, but are rare in adults, although cases of their occurrence in grown persons have been noted by Dr. Stephen Mackenzie and Sir Dyce Duckworth.[2] I have observed them in adults twice only: in one of these cases they were extremely numerous. These bodies vary in size from that of a pin's head to that of an almond, or even larger. They are not tender. They are found chiefly in the neighborhood of joints, especially at the back of the elbow, about the margin of the patella, and the malleoli. They occur also about the vertebral spines, the spine of the crista ilii, along the clavicle, the extensor tendons of the hand and of the foot, the pinna of the ear, the temporal ridge, the superior curved line of the occiput, and the forehead. I have seen them in one instance on the flexor tendons of the palms, as large as almonds, and quite preventing the proper use of the hands for the time. The largest crop I have ever observed was some thirty or forty, chiefly confined to the front of the chest, in relation to the tendons and fascia of the intercostal muscles. They sometimes appear in successive crops, sometimes singly, sometimes multiple. In one case in which this point was noted they developed in the course of ten days; but they usually take many weeks to subside. There can be no question of their relation to rheumatism: out of a large number of cases I have not met with one in which they did not occur in connection with some rheumatic manifestation. In Drs. Barlow and Warner's cases there was distinct evidence of rheumatism in twenty-five out of twenty-seven. In several cases also there was chorea, and frequently erythema marginatum. Their chief association, however, is with endocarditis and pericarditis; and this gives them an especial clinical significance and value. They seem to appear concurrently with endocardial inflammation, and, when the eruption is plentiful and recurrent, are signs of great import.[1]

---

[1] Trans. International Medical Congress, 1881, vol. iv. p. 116.

[2] Clinical Society's Proceedings, vol. xv., 1883.

Erythema.—Exudative erythema appears as one of the phases of rheumatism in several of its various forms. Of these, erythema marginatum and urticaria are the most common. The former is a frequent accompaniment of articular rheumatism in children, being far more often observed in them than in adults, appearing on the body as well as on the limbs. Out of eight cases of acute articular rheumatism, with nodules and heart-disease, under my care in the wards at Great Ormond Street at one time, three have had erythema marginatum, and one urticaria. Dr. Barlow[2] gives a series of striking cases in which the marginate or urticarious form appeared simultaneously with pericarditis, or immediately preceded it, the joint-affection following later.[3] Out of twenty-seven cases of fibrous nodules, erythema papulatum or marginatum appeared in eight.[4] The claim of erythema nodosum to be included in the rheumatic series is more doubtful. It is, no doubt, usually accompanied with pain and tenderness of joints, closely resembling a rheumatic arthritis, and, according to my observation, in joints removed from the site of eruption, so that the pain cannot always be due to the presence of swelling on unyielding parts, as Dr. Barlow has suggested. Yet, as he justly affirms, until this form of erythema is found associated with heart-disease or other attacks of undoubted rheumatism, we must hesitate to accept it definitely as a rheumatic expression. I have, however, now under my care a case of a boy of two and a half years with erythema nodosum on the shins only. He has had pain in all his limbs, and has a well-marked mitral murmur. He has no arthritis; but his father has rheumatism, and his father's sister has had rheumatic fever twice.

About purpuric erythema—the peliosis rheumatica of Schönlein—there can be less doubt. It is said to occur almost exclusively in young adults; but I have seen several cases in young children. It is to be distinguished from the purpuric eruptions which occur in nephritis, pyæmia, and other forms of blood-poisoning, in morbus cordis, and in wasting of organic diseases. The special characteristics of this form of erythema are excellently portrayed by Dr. Kennicott,[5] who shows that it is quite distinct from a simple purpura; and with this view I fully agree. The subcutaneous hemorrhages which form a conspicuous feature of the eruption are probably due to thrombosis of small vessels, as in blood-poisonings; and this is entirely consistent with the rheumatic connection, for in rheumatism the blood is hyperfibrinous, and thromboses in even large veins occur during life, and abnormal coagula after death.

---

[1] See Endocarditis, vol. ii.

[2] Brit. Med. Jour., Sept. 15, 1883, p. 313.

[3] It is possible that in some instances the erythema may be due to the toxic action of salicylate of soda, quinine, or arsenic.

[4] Barlow and Warner, Trans. Int. Med. Cong., 1881, vol. iv. p. 118.

[5] American Archives of Dermatology, 1875.

Purpuric erythema may develop as an isolated phenomenon quite apart from other symptoms, as in the case of a boy of eight under my observation who had had acute articular rheumatism. A year after this attack, having in the mean time been entirely free, he got a severe chill from standing in wet grass after becoming overheated playing cricket. He was seized next day with stiffness and tenderness of both ankles, and the legs were covered with slightly raised purple patches interspersed with purpuric blotches. These were chiefly on the sides of the ankles and lower calf,—not along the line of the shin-bones,—and the raised portions were smaller and bluer and much less raised than those of erythema nodosum, and more distinctly hemorrhagic. There was never any rise of temperature. Under salicin the swelling and tenderness disappeared in the course of a few days, and the eruption gradually faded away. Later the boy had chorea and repeated attacks of tonsillitis. The heart remained unaffected, and no nodules were developed at any time.

Allied to this condition, perhaps, is hemorrhage from the bladder, which I have met with in one or two instances associated with rheumatic cystitis, but never in children.

Chorea.—That there is some connection between chorea and rheumatism is generally admitted. The question of such connection is narrowed to one of degree. We cannot go so far as M. Roger, who regarded all chorea as of rheumatic origin. All cases of chorea cannot be traced to rheumatism: some are probably entirely apart from it. Other factors are concerned in the production of chorea. But in a very large proportion there is a remarkable association with the rheumatic state.

The neurotic factor is no doubt an important one. The mobile temperament of children, the wider expression of movement insisted on by Dr. Sturges, the large preponderance of cases in girls as compared with boys,—nearly three to one,[1]—the occurrence of the affection chiefly in quick, emotional children and in emotional races, the agency of fright or mental excitement as an immediate exciting cause, all point to a nervous factor. The statistics of the Collective Investigation Committee[2] yield a neurotic family history in forty-six per cent.; but, as this includes fourteen per cent. of chorea itself, and disorders such as sunstroke, injury to spine, sciatica, herpes zoster, tubercular meningitis, drunkenness, paralysis, etc., many of which are purely accidental, and others have obviously no connection with an unstable nervous system, they are of little practical value. A careful inquiry into all my own cases shows distinct inherited neurosis in a small proportion only.

---

[1] Coll. Inv. Record, vol. iii., 1887, p. 45.

[2] Ibid., p. 54.

The existence of the neurotic or emotional factor does not exclude the existence of a rheumatic factor. This point is constantly discussed as if the two were mutually antagonistic or destructive and it were a question of either one or the other. I believe there is a close association of the two, and that they are constantly at work together.

The evidence of the intimate relation of chorea to rheumatism is conclusive. In the first place, it is constantly seen as a sequel or an accompaniment of acute articular rheumatism, and of no other acute affection except scarlatina, with which also rheumatism has a curious connection. Again, it is followed in certain cases by acute articular rheumatism, at varying intervals.

It occurs in connection with simple joint-pains, which are probably rheumatic, since exactly similar joint-pains without swelling are often associated with endocarditis and pericarditis. It occurs also in conjunction with endocarditis and pericarditis, together with certain other affections of rheumatic nature, such as subcutaneous nodules and erythema, or with these alone. When it occurs with heart-disease without any other rheumatic manifestation, the morbid changes found in the heart are exactly those met with in rheumatic inflammation,—viz., mitral valvulitis and pericarditis.

Looking at the proved connection of chorea with rheumatism on the one hand and with heart-disease on the other, the association of the two affords presumptive evidence that both are rheumatic.

The association of emotional excitability with both chorea and rheumatism affords further presumption in the same direction. The greater frequency of chorea in girls corresponds with the greater frequency of acute rheumatism in girls and the prevalence of mitral stenosis in young women.

Statistics as to the connection of chorea with rheumatism are numerous. We may, however, at once place on one side those which deal solely with rheumatic arthritis which is either antecedent to the chorea or its immediate accompaniment. For any calculations based on this one point of contact alone must obviously be inadequate, since they take no account of the cases in which chorea comes after the arthritis. These statistics, moreover, as a rule, deal only with well-marked attacks of articular rheumatism; they omit also to weigh the presumptive evidence afforded by the coexistence of endocarditis and pericarditis, of subcutaneous nodules, of erythema, and of inheritance. The statistics of the Collective Investigation Committee, recently published, upon which I have already drawn, are of higher value. They are based upon 439 cases, and give 24.4 per cent. of antecedent rheumatic arthritis and 12.6 per cent. of concurrent or immediately subsequent arthritis, making a total of 37 per cent. unquestionably rheumatic. If 5.6 per cent. of cases with vague pains probably rheumatic were included, the proportion would be 42.6 per cent. Yet this must, in the nature of things, be still below the mark, since the cases in which arthritis occurs some time later are necessarily excluded; and the evidence afforded by mitral disease, by pericarditis, by fibrous nodules, by erythema, by tonsillitis, apart from arthritis, is not estimated. When these occur in combination, their cumulative weight as evidence of rheumatic connection is considerable. Dr. Barlow found satisfactory evidence of rheumatism, exclusive of family history, in 57.3 per cent. My own statistics, taken with minute care with especial regard to this point, and including in several instances many years of the after-history, give 73 per cent. From this, however, some reduction must be made, probably of 20 to 25 per cent. of those cases in which family history is the only evidence, on account of the normal incidence. This would bring the estimate into close equality with that of Dr. Barlow. Whether a given case of chorea is rheumatic or not can be determined only by a comprehensive survey of the patient's life-history.

Chorea may occur at any point in the series of rheumatic symptoms: when it is extreme and combined with severe endocarditis or pericarditis it is of great gravity. Nearly all fatal cases of chorea appear to be thus associated, and to be rheumatic.

Thrombosis and embolism are conditions which have to be reckoned with in the course of rheumatism. The existence of hyperinosis and the tendency to the formation of clots has been mentioned in speaking of the pathology of the disease; and this is sometimes a serious source of danger. I do not think that plugging of any large vessel occurs except when the circulation is interfered with and slowed by the concurrent mitral disease or pericarditis. I have twice seen fatal consequences follow. In one case, a girl of seven, with acute arthritis and endo-pericarditis, had thrombosis of both iliac veins, followed by extreme œdema of both lower extremities. This subsided; but six weeks later, on jumping up to look out of the window at a passing band, she was suddenly seized with faintness and dyspnœa; disseminated pneumonia appeared in both lungs, of which she died in a few days.

In the other case—one of rheumatic pericarditis—thrombi formed in the right auricle, accompanied by extremely irregular and turbulent action of the heart, and then fatal embolic pneumonia.

There are other symptoms associated with rheumatism of which brief mention must be made. One of these is emotional excitability. It is a question whether this is a direct consequence of rheumatism, or only a part of the chronic phase. I am inclined to think that the emotional, neurotic disposition goes generally with the tendency to rheumatism. Rheumatic children are, apart from the chorea, abnormally excitable: they are restless, fidgety, nervous.

Meningitis is stated to occur in the course of rheumatism, and cases are quoted by Senator.[1] It is possible that the serous membrane of the brain may be stirred up to inflammation by the rheumatic poison, like other serous membranes, but I have not met with such a case. The cerebral symptoms observed in some cases, which Trousseau put down to cerebral rheumatism, appear to be effects of pyrexia or pericarditis, and sometimes nowadays of salicylate of soda. Night fevers, headaches, and incontinence of urine are stated by Dr. Goodhart to be especially common in rheumatic children. I have not observed any special relation of these conditions to rheumatism, but the association with nervous excitability and anæmia makes the connection a probable one. Anæmia is indeed a prominent symptom in the rheumatism of childhood. It is perhaps present in some degree in ordinary cases, but it is remarkable especially when there is serious heart-disease, either valvular or pericardial. It sometimes progresses to an extreme degree, and is then a sign of grave import. Children with serious heart-damage from endocarditis or pericarditis suffer in nutrition in a most striking way; they not only grow extremely pallid, but also lose flesh and strength, and die at last of progressive debility and heart-failure.

---

[1] Ziemssen's Handbuch, vol. xvi. p. 50.

It has been shown that the different manifestations of rheumatism may occur in various combinations and in various order of succession. Examples of some of the slighter forms have been given in illustration of different points previously. More complete examples of the chief of the different series of rheumatic events are briefly shown in the following clinical examples.

1. Rheumatic arthritis first, then mitral disease followed by chorea and pericarditis. Family history of rheumatism.

A. I., a girl of thirteen, admitted to St. Mary's Hospital, January 26, 1885, suffering from chorea and morbus cordis. She had had three attacks of rheumatic fever, the last six months before. At that time heart-disease was first discovered. Chorea commenced a fortnight before admission. On examination, a loud mitral regurgitant murmur was found, with thrill, and signs of considerable hypertrophy and dilatation. Nineteen days later severe pericarditis set in without joint-symptoms. With this the chorea ceased, and she eventually recovered. Her father had had "rheumatism," and two of her sisters rheumatic fever.

In this case there can be no doubt that the chorea and the pericarditis were rheumatic, although they followed six months after the arthritis.

2. Rheumatic arthritis, endocarditis three years later, then arthritis again.

E. R., a girl of twelve, admitted November, 1877, to St. Mary's Hospital with pyrexia, —temperature 103°,—palpitation, and dyspnœa. There was no murmur, no pericardial friction. She had had rheumatic fever three years before. Her father had had rheumatic fever. From this history endocarditis or pericarditis was suspected. Four days later a loud aortic murmur developed. A fortnight later, or when convalescent, slight swelling and tenderness of one ankle appeared. Two years later still, general rheumatic arthritis occurred.

In this case, again, there can be no doubt that the endocarditis was rheumatic, although unaccompanied at the time by arthritis.

3. Chorea, mitral disease, no arthritis. Family history of rheumatism.

H. C., a boy of ten, admitted into the Hospital for Sick Children, June, 1878, with mitral stenosis, regurgitation, and great hypertrophy. He had not been known to have any joint-affection, but he had had chorea three years before. His mother had had rheumatic fever, and was suffering from mitral disease.

The chorea and mitral disease were almost certainly of rheumatic origin, the character of his heart-disease and the fact of the mother having had rheumatic fever and endocarditis affording strong presumptive evidence, although the patient had never developed articular rheumatism.

4. Chorea, mitral disease. No history of articular affection in patient or family.

S. B., a girl of nine, admitted with a third attack of chorea. No cause could be assigned. Physical examination disclosed a loud mitral murmur with hypertrophy. She had never had any stiffness or tenderness of joints. No history of rheumatism in any form in the family.

Here there was no evidence of rheumatic taint; yet viewed in the light of the constant association of such heart-affection and chorea with the rheumatic state, the most probable supposition is that they were thus connected. The articular affection might follow later.

The next example—where the joint-affection appeared last in the series—throws light upon such cases as the two preceding.

5. Chorea, mitral disease, then arthritis four years later. Rheumatic family history.

A. W., a girl of sixteen, admitted to St. Mary's Hospital with stiffness, swelling, and tenderness of wrists, knees, and ankles. She had never had any joint-affection before, but four years previously chorea. On physical examination, marked signs of mitral disease were found,—a loud regurgitant murmur, heaving impulse, extensive præcordial dulness, showing the heart-disease to be of old standing and not due to the present attack. Her mother and her mother's sister had both had rheumatic fever.

Now, this case was almost exactly similar to the two preceding up to the time of the occurrence of the joint-affection four years after the chorea. Previous to this the case would probably have been classed as non-rheumatic.

6. Mitral disease first, followed by arthritis after a long interval, then chorea.

K. D., a girl of eight, admitted to St. Mary's with advanced mitral disease of old standing, causing palpitation and dyspnœa, the result of endocarditis years before. Yet she had never had any articular affection until five weeks ago, when she was confined to bed with stiff and tender joints for ten days. Fourteen days after admission she developed marked general chorea.

In this case there was nothing to connect the endocarditis in any way with rheumatism at the time of its first occurrence. The nature of the case was not proved until the arthritis and chorea betrayed it long afterwards.

These cases were recorded before the significance of nodules and erythema and tonsillitis as signs of the rheumatic state was appreciated. The following recent examples show a much fuller series, and the four last exhibit the disease in its gravest and most fatal form in childhood.

7. Chorea, arthritis, endocarditis, second chorea, nodules, tonsillitis, erythema.

W. L. D., a boy of eight, admitted to the Children's Hospital in Great Ormond Street, November 11, 1887. A year ago he had chorea, brought on by fright, followed six months later by arthritis, accompanied by endocarditis. Six months later still he was admitted for a fresh attack of chorea and tonsillitis. There was a fibrous nodule over the right elbow, and a loud double mitral murmur. While in hospital he had an extensive eruption of urticarious erythema.

8. Arthritis, chorea, nodules, endocarditis, slight arthritis, erythema.

F. M., a boy of five, was admitted on March 8, 1888, with chorea. He had had two previous attacks of rheumatic fever. There was a mitral regurgitant murmur, with accentuation and reduplication of the second sound at the base. Large nodules were found on both malleoli, others of different sizes on the condyle of the femur, the patella, clavicle, scapula, extensors of fingers, and posterior scalp. Slight tenderness and swelling of the left wrist-joint appeared next day, but subsided quickly. A week later a copious eruption of erythema marginatum appeared on the back.

9. Chorea, arthritis, second chorea, second arthritis, copious evolution of nodules, endocarditis, erythema, third arthritis, second nodules, fresh endocarditis, tonsillitis, progressive anæmia, death.

W. S., a boy of four, admitted to Great Ormond Street, December 3, 1887, with a second attack of chorea, and pains behind the knees. The first ailment was chorea, a year ago. No heart-affection observed then. Now a full double mitral murmur. A plentiful crop of nodules, some as large as the end of a little finger, on the knees, elbows, spine, and scalp. While in hospital he had two eruptions of erythema marginatum. In seven weeks he was discharged, convalescent, but was readmitted three weeks later, with fresh copious eruption of large nodules, fresh arthritis, and fresh endocarditis, and subsequently tonsillitis. He became extremely anæmic, the heart's action became excited, with increased heat and slight rise of temperature, and he shortly died of exhaustion and heart-failure. No post-mortem examination could be obtained.

10. Arthritis, endocarditis, fresh arthritis, abundant nodules, erythema, pericarditis, death.

A. C. B., a boy of eleven, admitted for articular rheumatism. A loud systolic mitral murmur, also a diastolic over sixth space, and increased area of cardiac dulness. A numerous crop of subcutaneous nodules was found on the extensors of the fingers, elbows, knees, malleoli, and spine. Marginate erythema followed, then fresh nodules began to be developed; anæmia increased remarkably, and the temperature rose; pericarditis set in, accompanied by marked anæmia and distress, with vomiting, and the child died four days afterwards. Post-mortem examination showed the pericardium adherent throughout, greatly thickened, the mitral valve rigid and thickened, the aortic valves covered with a fringe of recent vegetations.

11. Chorea, second chorea, then arthritis, endocarditis, unusually large eruption of nodules, emotional attacks, fresh chorea, fresh crop of nodules, progressive anæmia, pleurisy, pericarditis, and death.

J. T., a boy of seven, brought in for articular rheumatism. He had had two attacks of chorea, the last three months before. A large number of nodules, from the size of a pea to that of a nut, formed a most striking feature of the case. They were found abundantly on the occiput, the spinous processes, the wrists, flexors of hands, the ankles, and the feet. Twenty-five were counted in all. Endocarditis followed, culminating in double mitral disease. Occasional emotional attacks of crying and laughter. Fresh evolution of nodules, and progressive anæmia, followed, then pleurisy, persistent pericarditis, lasting two months, and death. The whole course after admission lasted eight months. Post-mortem revealed adherent pericardium with enormous thickening, especially in front, granulations on aortic, mitral, and tricuspid, with thickening of the two former.

12. Articular rheumatism, chorea, pleurisy, pericarditis, death.

M. B., a girl of fifteen, admitted to St. Mary's with extreme chorea, following articular rheumatism. The chorea was so violent that she could be fed only with great difficulty, and she was quite unable to speak. In order to take the temperature, two nurses had to restrain her: it was 103.4°. It was almost impossible to examine the heart, owing to the extreme jactitation, but great dulness was made out over the cardiac area, with friction at one point, and muffling of the sounds. The diagnosis was acute rheumatic chorea with pericarditis. The girl died a few hours later. Post-mortem examination revealed extreme distention of the pericardium with purulent fluid.

Diagnosis.—There are few diseases in which diagnosis is more easy and certain than in a well-marked attack of acute articular rheumatism in an adult. The swollen, tender joints, the profuse sour sweating, the mere attitude of the patient, lying still and motionless, afraid to move hand or foot, are in themselves almost sufficient to distinguish it. In children, however, we rarely see this typical extreme arthritic form: it is not common in older children even before the age of puberty; in very young children it is unknown. The diagnosis of rheumatism in early life, when arthritis is at its minimum and fever and sweating are not pronounced, is often difficult enough, and in many cases it is only by a complete and careful survey of the whole history of the patient that a correct conclusion as to the nature of the attack can be attained.

If the articular affection is distinctly present, the tender, painful, swollen joints, with a faint blush of redness on them perhaps, the tendency to sweating, the rise of temperature to from 100° to 103°, the behavior of the inflammation in shifting from one joint to another, declining in a few days and then reappearing, are characteristic. When the tenderness and swelling are extremely slight, confined to a single joint, or in a tendon or fascia, or if there is merely a little stiffness, it is occasionally difficult to decide whether the affection is really rheumatic or not, although the mere existence of anything of the kind is in itself in the case of a child very suggestive of rheumatism. Such conditions are usually rheumatic, and are, it is to be remembered, genuine rheumatism, bearing with it all the possibilities of cardiac inflammation. How constantly these cases must be overlooked is shown by the fact that out of six hundred and fifty-five cases of all ages given in the statistics of the Collective Investigation Committee[1] thirty-two only were in children under ten; yet, as I said before, it is one of the commonest diseases in the wards of a children's hospital, and not infrequent in private practice. The discovery of a fibrous nodule, or the rash of erythema, or a mitral murmur, or pericardial or pleuritic friction, or that the patient has had a previous attack of rheumatism in any form, or inherits a family taint of it, will help to solve the question. All these points should be minutely inquired into, and in every case the heart carefully examined day by day. There are one or two affections which might be mistaken, and indeed now and again are mistaken, for acute rheumatism. One of these is infantile paralysis. In certain cases of this disease there is at first extreme tenderness of the affected limbs, and sometimes also a little swelling of the foot and ankle, if the legs are affected. It may be distinguished, however, by the flexed muscles, the helpless rolling of the limb on movement, the pitiful inability of the child to move it except by lifting it with the hands, and the absence of sign of heart-affection or other rheumatic symptom.

Another condition which is frequently mistaken for rheumatism in adults is pyæmia. It is not common in children, but it does occur, and in them, as in adults, is distinguished by hectic sweats and temperature, by the existence of some local suppuration, and by the course of the disease. An acute suppuration of joints, of obscure origin, but possibly septic, not infrequent in young infants, has some resemblance at first to a rheumatic arthritis. The greater pain and swelling, and usually greater fever and constitutional disturbance, with the eventual presence of pus, serve to distinguish it from true rheumatism.

Scrofulous affections of the joints are liable to be mistaken at the outset, but they may be recognized usually by their steady, unshifting character. Rheumatism in a child may be persistent, but it is shifting and changeable.

The tender swellings of limbs and œdema of ankles met with in certain cases in connection with rickets, and which I have shown to be essentially scorbutic,[2] are often put down as rheumatic. They sometimes affect the tissues around joints, as well as the periosteum of the long bones and the muscles, but they may generally be distinguished without difficulty by the condition of the gums, the various hemorrhages usually present, the normal or subnormal temperatures, and the fact of the child being fed upon scurvy diet. In simple rickets, when extreme, there is bone- and muscle-tenderness; but the existence of the obvious signs of rachitis is sufficient to show its nature. There is one condition connected with rickets, however, which is sometimes taken as rheumatic, and indeed has been styled so by some authors, although it is not in any way connected therewith,—viz., the swelling of the dorsum of the foot and back of the hands which occurs in the tetany or carpo-pedal contractions of young rickety children. The tumefaction appears to be due to the pressure of muscles in tonic contraction, exactly analogous to that produced by a tight bandage.

---

[1] Coll. Inv. Record, vol. iv., 1888, p. 67.

[2] Lancet, Nov. 16, 1878, vol. ii. p. 685.

There is a disease of the bones developed in congenital syphilis which in some degree simulates rheumatism,—viz., inflammation of the ends of the long bones where the shaft joins the epiphysis. There is tenderness and occasionally swelling of a joint, with effusion, and possibly suppuration. The affection is one attending the first explosion of the congenital disease, —within the first three months; and the age is almost in itself diagnostic. The existence or history of other signs of syphilis, such as characteristic eruption, snuffles, wasting, cranial bosses, or tabes, renders its recognition easy. A certain amount of arthritis with extravasation of blood into the joints and serous effusion around them, with a rise of temperature, is said to occur occasionally in hæmophilia and purpura hæmorrhagica. The diagnosis from a rheumatism accompanied by a purpuric erythema would appear to be difficult, but the history of bleeding, with the graver symptoms and longer course, serves to separate them from the light and transient disturbance of rheumatic purpura. If the joint-affection has passed, or is altogether absent at first, the determination of the rheumatic or non-rheumatic nature of other manifestations is a matter of more nicety.

When endocarditis arises in a child, there is always a strong *prima facie* presumption that it is of rheumatic nature: from fifty to eighty per cent. can be traced to rheumatism, according to the investigations of Roger, Goodhart, and myself. If with the cardiac inflammation we have a group of symptoms known to be associated with rheumatism,—chorea, fibrous nodules, erythema, or tonsillitis,—whether any or all of these have occurred recently or cropped up from time to time at intervals through months or years, the cardiac inflammation is almost certainly rheumatic. The presence of one of these fibrous nodules alone is almost absolutely diagnostic. The coexistence of chorea would have great weight, although not absolutely conclusive; the concurrence of two conditions each so closely connected with rheumatism is necessarily of much significance. The same holds good with regard to pericarditis: it is almost always rheumatic in children, and the coexistence of nodules or chorea or a series of other rheumatic phenomena would fix the diagnosis. As to the rest, the tonsillitis and erythemas (all except perhaps purpura rheumatica) are less strongly and constantly identified with the disease, and the value of their corroborative testimony would depend much upon their combination and other circumstances.

I do not know that fibrous nodules are ever found apart from arthritis or chorea or heart-disease; but they are associated with no other morbid condition except rheumatism.

The claims of erythema and tonsillitis to be considered as rheumatic phenomena have been stated in speaking of the symptoms. In case of their appearance alone, collateral evidence would have to be sought as to the existence of other more accepted signs, either at the moment or at some previous time.

The existence or absence of a strong family predisposition to acute rheumatism in its different phases would be evidence of much weight and would have to be duly considered.

In all these cases one phase or symptom alone must not be relied upon: a comprehensive survey of the whole available evidence from all sources must be examined and appraised.

But always in the case of children, whether unmistakable arthritis be present or there be merely a stiff and painful tendon, or an unexplained febrile attack, or chorea, or tonsillitis, or erythema, it is most essential to bear in mind the possibility of having to deal with rheumatism, and to examine the heart carefully day by day. It has happened over and over again in such circumstances that a cardiac murmur or a friction-sound has been discovered, betraying an endocarditis or pericarditis never even suspected, its presence not having been suggested by any marked fever or ordinary sign of rheumatism. It is impossible to urge too strongly the necessity for the most watchful care on this vital point.

Prognosis.—The prognosis of acute rheumatism in a first attack is unquestionably favorable, at any rate as to its immediate issue. The general direct mortality is only about three and a half per cent.,[1] and it is probably less in children than in adults. The latter are exposed to the danger of hyperpyrexia, a risk that barely seems to exist for the former, possibly because it occurs chiefly in those given to alcoholic excess.

Now and again, in rare cases, children do die in the primary attack from acute heart-disease, or from thrombosis and embolism. As a rule, however, in a first attack the joint-symptoms quickly get well, the endocarditis or pericarditis subsides, and if there be tonsillitis or erythema they seldom persist many days. Should chorea be present, it may linger on and relapse, but usually it passes off in the course of a few weeks.

If the first attack clears up quickly, if at most only a soft cardiac murmur at the base of hæmic character has been developed, or even if a mitral murmur has appeared and subsided, it may be hoped that little permanent damage has been done. But even then it is necessary to give a cautious forecast of the future. It is only by careful examination for long afterwards that the extent of cardiac mischief can be safely estimated. If a mitral or aortic murmur does not die away, or reappears and grows distinct, and signs of dilatation and hypertrophy follow, there is certainly serious valvular mischief. Owing to the greater liability to endocarditis and pericarditis in early life, the risk of permanent damage to the heart-structures is greater also. Compensation is no doubt more perfect if the lesion is small, and more quickly attained with children than with adults; but, on the other hand, if the valvular injury is great, the subsequent changes of hypertrophy and perhaps of dilatation also progress more rapidly, owing to the more ready growth of tissues in the young and their more yielding character. The hypertrophy, however, outstrips the dilatation.

---

[1] Senator, in Ziemssen's Handbuch, vol. xvi. p. 56.

And in prognosis it must be borne in mind that the danger is not over with the subsidence of the primary attack; it lies partly in the after-effects of the heart-affection, and less in the severity of the primary attack than in the frequent immediate relapses or the return of the disease at longer intervals. I have already stated that in children the cardiac inflammation has a tendency to go on smouldering in subacute form, and thus by chronic changes or frequent repetition to produce a grave cumulative result. If there have been previous attacks of rheumatism, and especially if the heart-valves have been already injured, the prognosis is much less favorable than in a first attack. The cardiac inflammation is apt to revive, and the mischief to valves or pericardium to increase. The worst cases are those in which the heart already shows signs of great enlargement, evidenced by heaving, diffused impulse, and increased area of cardiac dulness. Fresh endocarditis and pericarditis are then especially apt to supervene, often accompanied by pleurisy; dyspnœa and rapid action of the heart follow, there is progressive anæmia, and the patient sinks from failure of his embarrassed and weakened heart. Probably in such cases there is myocarditis. Pleurisy and pneumonia are apt to occur also; they are chiefly dependent upon the heart-lesion and consequent impediment to the circulation, and this, of course, adds to the gravity of the prognosis. Vomiting is an unfavorable sign when it occurs persistently in the course of pericarditis. Marked anæmia is a sign of evil omen: it is one of the most striking results of cardiac rheumatism in children. It is as characteristic of mitral disease and pericarditis of children as it is of aortic regurgitation in adults.

The congested face so frequently seen in patients with mitral disease is rarely met with in children: they are always pallid and bloodless, and emaciated instead of being full-fleshed.

In these cases in children, too, dropsy is only occasionally met with: it is comparatively rare to see a child hopelessly swollen, waterlogged from dilatation of the right heart sequent to mitral disease.

Another symptom which I have come to regard as of very grave import is the copious and repeated evolution of fibrous nodules. They go especially with chronic valvulitis and pericarditis; and so long as the nodules continue to appear there is reason to fear progressive heart-mischief from a similar sub-inflammatory process in the fibrous tissue of valves and pericardium. Such cases do badly, as a rule: they have constant and repeated relapses, progressive heart-mischief, and progressive anæmia. Nearly all I have seen have proved fatal in the course of a few months. Three such examples have been described in the clinical cases previously given. Another danger is copious effusion into the pericardium: this can be estimated by the increasing dulness, muffling of the heart-sounds, and upward displacement of the apex-beat. Yet this is far less common than in adults: an adhesive pericarditis with subsequent fibrosis is the rule with children, rather than one with copious effusion; although I have twice seen this fatal in young girls about the age of puberty. A less common sign of the greatest gravity is the supervention of dropsy, indicating extreme dilatation of the right heart. In one instance under my observation this followed a fresh attack,—simple acute dilatation without valve-lesion; this is, however, more common after scarlatinal nephritis than after acute rheumatism.

Another symptom which always adds to the gravity of the prognosis is the supervention of extreme chorea,—*i.e.*, chorea so severe that the patient can hardly be kept in bed or obtain sleep on account of the violence of the jactitations,—especially if the child is a female approaching the age of puberty.

The statistics available are not quite satisfactory, since they make antecedent arthritis the sole test of rheumatism; yet these appear to show a special relation to exist between fatal chorea, rheumatism, and the age of puberty, in girls.[1] The coexistence of endocarditis or of old valvular disease, and still more of pericarditis, especially if accompanied by signs of effusion, with embarrassed, quick, irregular action of the heart, and dyspnœa, or of very active plastic inflammation, renders the condition more dangerous still. In such cases the prognosis must always be serious and doubtful; and the expectation of favorable issue will depend chiefly upon the decline of the cardiac symptoms, the power of taking food, and of rest in sleep.

The occurrence of thrombosis adds a fresh element of danger. As previously stated, I have twice seen death take place in children from pulmonary embolisms and pneumonia due to thrombosis,—in one instance of both iliac veins, in the other of the right auricle.

Lastly, there must be considered the possibility, if attacks of arthritis are repeated, of permanent alterations in one or more joints, such as thickening and ankylosis. These events are, according to my experience, rare in early life. In cases where suppuration follows a so-called rheumatic attack it is probable that the condition was really pyæmic.

Treatment.—The principles upon which a case of acute rheumatism with arthritis should be treated at the outset are—first, to prevent fresh chill to the surface; secondly, to keep the affected parts at rest, so as to lessen the flow of blood there and friction of parts, and thus to lighten inflammation and relieve pain. This applies not only to the joints, but more still to the heart. Dr. Sibson's observations show how important a means this is of modifying the after-effects of endocarditis. Thirdly, to modify, if possible, by specific remedies, the fever and neutralize the irritant effects of the rheumatic poison on the fibrous tissues of joints and tendons; fourthly, to prevent, if possible, the inflammation of the endocardium and the pericardium, or, if this has set in, to minimize and arrest it; and lastly, to relieve pain directly by anodynes if necessary. It may be thought that many of these ends cannot be attained with certainty; and indeed the statements made as to the effects of remedies in rheumatism are in some respects conflicting and unsatisfactory. Yet there is evidence that most if not all of the objects laid down can be some of them effected with certainty and others greatly aided by the use of remedies. In the last twenty years great progress has been made in this respect.

---

[1] Sturges, Chorea, pp. 77–80.

If the tongue is coated or the bowels are constipated, a dose of one to three grains of calomel should be given at the outset,—an excellent preliminary to other treatment. To insure against chill and give rest, the patient should be kept in bed, either between blankets or in a flannel night-dress, until some time after the temperature has become normal and all symptoms have ceased. The tender joints should be placed in easy positions, propped by soft pillows, and wrapped in a little soft cotton-wool, but not covered with oiled silk or gutta-percha, as is usually done: this overheats the parts, and also, by keeping in acrid perspiration, increases the local irritation. If necessary, the weight of the bedclothes may be supported by a cage. If the pain and tenderness are great, a little liquor opii sedativus may be sprinkled on the cotton-wool, or, better still, the joints wrapped in light bandages kept wet with a lotion containing ten grains of bicarbonate of soda and ten drops of liquor opii sedativus to the ounce of water. This is far more effectual than any application of belladonna.

The application of cold in the form of water-dressings is advocated by Senator,[1] or even of ice, by Esmarch;[2] but of this practice I have no experience. It is stated to be safe, to relieve pain, and to lessen the duration of the joint-symptoms.

Another plan which has been highly commended for the relief of pain is the hypodermic injection of a one-per-cent. solution of carbolic acid under the skin over the affected joints, and the application of carbolized oil (one in fifteen).

The application of blisters, which certainly are of great service in relieving pain by reducing effusion, in adults, is not suited to children: the remedy is generally worse than the disease.

In early life the arthritis is seldom severe enough to require such applications. It will be frequently found sufficient to apply the alkaline opiate lotion and keep the part at rest. In the more chronic cases it may be worth while to secure this by the application of a splint.

The fever and pain of the arthritis of rheumatism can be subdued most quickly by the use of salicin or salicylate of soda. There is a very general agreement on this point, although opinions are still divergent as to their ultimate effect on the duration of the disease and the prevention of cardiac inflammations.

---

[1] Ziemssen's Handbuch, Eng. trans., vol. xvi. p. 68.

[2] Quoted by Senator, op. cit.

The statistics recently published by the Collective Investigation Committee,[1] based on six hundred and fifty-five cases, give an average duration of—fever, 8.65 days; pain, 10.18 days; whole attack, 19.03 days.

Salicylate of soda freely administered hardly ever fails to bring down temperature and relieve the pain of rheumatism in the course of twenty-four to forty-eight hours;[2] but there are several drawbacks to its use. In the first place, it sometimes sets up nausea and vomiting to a distressing degree. It has also a depressant effect upon the heart; the pulse loses strength, and the first sound of the heart becomes faint, so that in ten cases observed by Dr. Greenhow it was almost inaudible. It also produces in certain cases deafness, buzzing in the ears, vertigo, delirium. In extreme instances the symptoms have been alarming, such as great prostration, violent delirium, albuminuria, collapse. These results occur much less frequently in children than in adults; yet, in view of the proneness to heart-affection in the young, it is well to use a depressant drug with caution. But in truth it is only occasionally in children that the pain and fever are sufficiently important to require its administration. If the joint-symptoms are severe, however, and temperature much raised, salicylate of soda will reduce them more quickly than any other remedy. It should be given for the first twenty-four or forty-eight hours, and after that salicin be substituted for it; for salicin has little if any of the evil properties of salicylate of soda, although it appears to act through its conversion into salicylic acid in the blood, through the action of ptyalin or other ferment.[3] Possibly it acts less violently because more gradually passed into the circulation; it certainly produces its effect more slowly. In all but the most severe cases salicin is, then, preferable to salicylate of soda, and may be given for a day or two in doses of five to eight grains every three to four hours to a child of five, mixed with water and syrup of orange.

The salicin should be continued in less frequent doses for some days after all symptoms have ceased, or a relapse is liable to occur. These remedies will serve the purpose of reducing the temperature and the arthritis, but, unfortunately, they seem neither to prevent the occurrence of carditis nor to arrest or modify it when developed. The statistics adduced at the debate on this subject at the Medical Society of London in 1881 showed no distinct power of salicylates or salicin to prevent heart-affection. It would have appeared probable that a drug which cut short the arthritic manifestations of the rheumatic poison would also prevent or arrest what are believed to be similar changes in the cardiac valves and pericardium; but this has not proved so, and I have observed that the evolution of nodules, which may be taken, perhaps, as the external index of what is going on in the fibrous tissues of the heart, continues to develop in spite of the steady administration of salicin. The only treatment which can claim to show good evidence of power in this respect is that by alkalies. Statistics are not always reliable, but the cumulative weight of several independent results is considerable.

---

[1] Report of Collective Investigation Committee, vol. iv., 1888, p. 75.

[2] The Report previously quoted records twenty-two cases out of two hundred and ninety-six treated by salicylates and salicin in which they were ineffectual. The doses given were probably too small and at too long intervals.

[3] Senator, in Ziemssen's Handbuch, Eng. trans., vol. xvi. p. 1040; postscript by Dr. Buchanan Baxter.

Fuller had nine cases of heart-disease only out of four hundred and seventeen cases treated by alkalies, Chambers nine out of one hundred and seventy-four, Dickinson one out of forty-eight. On the other hand, the two latter observers had five out of twenty-six and thirty-four out of one hundred and thirteen in patients treated in other ways.[1] Moreover, the influence of alkalies in mitigating the joint-symptoms and shortening their course appears distinctly favorable, although much less decided and rapid than that of salicin and the salicylates. The recent statistics of the Collective Investigation Report[2] give an average duration of fever 13.23 days, pain 19 days, whole attack 36.3 days,—much more prolonged than cases under salicylates and salicin given above. Alkalies should, then, be given in combination, salts of soda in preference to those of potash, as being less depressant. With the salicylate of soda, six to ten grains of bicarbonate of soda; the same with the salicin. The amount of alkali must be regulated by the state of the urine; enough should be given to keep it neutral or slightly alkaline. If, however, endocarditis or pericarditis come on, the salicylates or salicin should be at once stopped, and the alkali given in freer doses,—ten to fifteen grains every four hours, with half a drachm of syrup in half an ounce of water. In severe and obstinate cases of endocarditis or pericarditis, when there is high temperature, palpitation, cardiac dyspnœa, and distress, quinine should be given in addition, in doses of two to three grains every four hours for a child five years old. This may be done by giving ten-grain doses of citrate of soda, two grains of quinine, and five grains of citric acid; or the acid hydrobromate of quinine may be given every four hours alternately with the alkali. This salt is extremely soluble (ten grains to one drachm), so that the dose can be administered in a single teaspoonful of water; and it has also the advantage of being less liable to cause sickness than the sulphate.

Many other drugs have been advocated in the treatment of rheumatism, some of which are useless, others harmful, or even dangerous. Among the latter must be mentioned antimony, aconite, and veratria. Their action on the joint-condition is uncertain, and, although the two latter have distinct antipyretic properties, the fact that they are, like antimony, heart-depressants, and of even more dangerous power, negatives their use in this disease, especially in children.

---

[1] The statistics of the Collective Investigation Committee, unfortunately, give no information on this point.

[2] Coll. Inv. Rep., vol. iv., 1888, p. 75.

Colchicum has not the same specific influence in rheumatism as in gout. As with so many other drugs, cases have been recorded in which it has seemingly helped to shorten the attack; but it is depressant, and the relief of arthritis can be effected better by other means.

Iodide of potassium is quite inefficient, or rather it would seem to have a retarding effect, the cases treated with salicylates and iodide together showing, in the Collective Statistics referred to, the highest average duration.

Nitre and lemon-juice, each highly extolled at one time, are distinctly inferior to salicin, the salicylates, and alkalies.

In cases of high temperature favorable results follow the use of antipyrin, which is advocated by Fränkel, who reports on thirty-four cases; and I have seen equally good results follow the use of antifebrin; but I have never had occasion to use these drugs in the case of children. Under both I have seen serious syncopal attacks occur in adults, and I should hesitate to use them at all freely with little children.

In my hands the results with salol have been unfavorable. It appears to combine the objectionable qualities of salicylic and carbolic acids, of which it is a compound, without marked effect upon the arthritis.

The following table, obtained from the very valuable Report of the Collective Investigation Committee so often referred to, drawn up by Dr. Whipham, gives the relative results of different forms of treatment:

	AVERAGE DURATION (IN DAYS) OF		
TREATMENT.	FEVER.	PAIN.	WHOLE ATTACK.
Salicylates (sodium or potassium)	(173 cases.) 8.65 days.	(171 cases.) 10.18 days.	(167 cases.) 19.03 days.
Salicylic acid	(9 cases.) 13.8 days.	(9 cases.) 10.7 days.	(9 cases.) 10.7 days.
Salicin	(14 cases.) 9.28 days.	(14 cases.) 15.07 days.	(14 cases.) 23.92 days.
Alkalies	(26 cases.) 13.23 days.	(26 cases.) 19.0 days.	(26 cases.) 36.30 days.
Alkalies and then salicylates	(22 cases.) 11.54 days.	(22 cases.) 13.90 days.	(21 cases.) 22.22 days.
Salicylates and alkalies (combined)	(12 cases.) 10.83 days.	(11 cases.) 15.54 days.	(13 cases.) 34.92 days.
Salicylates and then alkalies	(19 cases.) 10.78 days.	(18 cases). 13.16 days.	(17 cases.) 30.64 days.
Salicin and alkalies	(3 cases.) 11.6 days.	(3 cases.) 19.3 days.	(3 cases.) 24.0 days.
Salicylates and potassium iodide	(7 cases.) 17.14 days.	(6 cases.) 24.16 days.	(7 cases.) 46 days.
Salicylates and iron	(18 cases.) 11.77 days.	(19 cases.) 10.89 days.	(18 cases.) 27.7 days.
Salicylates and tonics	(16 cases.) 8 days.	(16 cases.) 10.18 days.	(16 cases.) 18.68 days.
Alkalies and opium	(8 cases.) 10.75 days.	(8 cases.) 12.60 days.	(8 cases.) 18.75 days.
Salicylates, then iron and quinine	(5 cases.) 10 days.	(5 cases.) 13.8 days.	(6 cases.) 20.33 days.

	AVERAGE DURATION (IN DAYS) OF		
TREATMENT.	FEVER.	PAIN.	WHOLE ATTACK.
Salicylates and opium	(10 cases.)	(11 cases.)	(9 cases.)
	9.9 days.	8.45 days.	30.3 days.
Salicylates and blisters	(7 cases.)	(7 cases.)	(6 cases.)
	6.14 days.	12 days.	15.83 days.
Alkalies and then quinine	(6 cases.)	(6 cases.)	(5 cases.)
	13.5 days.	21.6 days.	35 days.
Salicylates and quinine	(6 cases.)	(6 cases.)	(6 cases.)
	10.5 days.	17 days.	31.6 days.

It has been said that when endocarditis or pericarditis comes on, the alkaline treatment should be adopted, as giving the best results, salicylates or salicin being discontinued, and quinine substituted if the temperature runs high. Warm poultices to the præcordia may be useful in endocarditis, but I can see no advantage to be gained by the application of blisters or leeches, as advocated by some. There is no connection between the circulation in the skin and that of the endocardium. With pericarditis it is different: there the superficial vessels have free communication with those of the parietal pericardium, and local depletion of the surface must directly relieve the hyperæmia of the serous membrane below. At the outset of pericarditis, one to three leeches, according to the age of the child, are often of distinct service. Care must be taken that the bleeding from the leech-bites does not go on too freely after their removal. I have twice seen serious enfeeblement of the heart from too great loss of blood through carelessness in this respect. Blisters, on the other hand, seem of chief value when there is effusion; not in the early stage of active inflammation. They cause children annoyance and distress, and should not be resorted to without good reason.

If in endocarditis or pericarditis the action of the heart is rapid and turbulent, three to five drops of tincture of digitalis may be given every four hours to a child of five years for twelve or twenty-four hours, after which it should be given less frequently. Yet it must be administered cautiously: it is a dangerous remedy when there is much pericardial effusion, or if the heart is greatly embarrassed by thickened adherent pericardium. When the palpitation is due to feebleness or dilatation, digitalis has great power to steady and give tone and force to the cardiac contractions. Although stimulants should be avoided if possible, they are sometimes necessary when signs of heart-failure appear. In such cases they may be given freely to the amount of three ounces of wine and one and a half ounces of brandy in the twenty-four hours. Alcohol is wonderfully well borne by children; and it is to be noted that it produces little excitement, but rather acts as a sedative. For young children it is one of the best of sedatives, often as effectual as, and more safe than, opium.

A remedy of immense value in most stages and forms of rheumatic fever is opium. It may be required to ease pain and restlessness and produce sleep. It may be given freely in doses of one to three minims every four hours for a child of five years, if there is no concurrent pneumonia or bronchitis. The vomiting which sometimes sets in at the close of pericarditis should be combated by ice, by small doses of hydrocyanic acid and soda, and by the substitution of nutrient enemata for twelve hours for food by the mouth. If chorea is severe, and the patient cannot get sleep on account of the constant movement, chloral and bromide may be given in doses of five grains of each in syrup of orange every four hours until drowsiness comes on.

Once in my experience obstinate sleeplessness was found to depend upon pain caused by the incessant jactitation of an arm of which the wrist was swollen and tender from acute arthritis: bandaging the arm to the side in a comfortable position relieved the difficulty for the time, and the administration of salicin quickly removed the joint-tenderness, and sleep followed without narcotic.

When the temperature has come down and remained normal for a week, iron should be given for the anæmia which, as has been stated already, is so marked in the rheumatism of childhood. The citrate of iron in doses of five to six grains, with ten to fifteen grains of citrate of soda and syrup of ginger in half an ounce of water, should be given as a precaution against relapse, especially if there has been any cardiac inflammation; or citrate of iron and quinine five grains with citrate of soda or potash in the same way, with two drachms each of water and chloroform-water; but in this case five grains of citric acid or a teaspoonful of lemon-juice must be added, to prevent the precipitation of the quinine by the excess of alkali which is practically always present in the soda or potash salt. If the anæmia is extreme, or the chronic symptoms persist, arsenic should be given with iron in the form of two drops of liquor potassii arsenitis in two drachms of wine of iron twice a day after food. This is the most efficient of all drugs in the restoration of red blood-corpuscles: it should not, however, be prescribed until all symptoms of active inflammation are over, for it stirs up hyperæmia in skin and mucous membranes, as evidenced by the reddened conjunctiva and tongue and flushed skin produced by full doses of the drug, and may presumably affect fibrous structures and serous membranes in like manner.

The erythema goes with the subsidence of the other symptoms, and requires, as a rule, no special treatment. Tonsillitis yields to salicylates and salicin with great readiness.

The diet in cases of rheumatism does not call for the close limitation which is usually enforced. When the temperature is raised, and acute symptoms are present, it should consist entirely of beef tea or broth with milk. In cases of great anæmia or prostration, Valentine's meat juice, or even raw meat pulp itself if it can be taken, should be given as blood-restorers; and self-digesting foods, or peptonized milk, are most useful. As the fever declines, light pudding, eggs, bread-and-butter, and tea may be permitted; and thus the patient may pass on to fish or meat.

Large quantities of sugar are theoretically objectionable, as tending to favor lactic fermentation. The patient should rest in bed for at least ten days after all acute symptoms have finally disappeared, so as to insure against chill and preserve extreme quietness of cardiac action.

Prevention.—A child who has once had acute rheumatism is prone to have it again, the tendency gradually becoming less as age increases. A child born of rheumatic stock has also a special liability to the disease. In both cases precautions should be taken to protect those who are thus predisposed from overheating, chill, and over-fatigue, the great sources of rheumatism. To this end, the child should not be kept too tenderly, but should be kept out of hot, close rooms, should live in a cool and even temperature, should be clothed in woollen next the skin, while the body is hardened by tepid salt-water baths and vigorous friction. In case of accidental exposure to cold or wet, brisk exercise should be taken until a full glow of warmth is experienced, and damp clothes changed at the earliest moment. When heated or overtired by severe exertion, standing about in cool air should be carefully avoided, and the body protected against chill by an extra covering until it cools down again. Damp air and cold soil and variable climate should be avoided. When circumstances permit, the rheumatic child should be removed to a dry, warm climate, with sandy soil, in a situation not overcrowded with trees, exposed to sunlight, and with a free circulation of air.

CHRONIC RHEUMATISM.

Chronic Rheumatism is rare in children,—much more rare than in adults. It is to be distinguished from the relapsing form of acute rheumatism, where fresh exacerbations of a mild kind—sometimes nothing more than stiffness and vague pains without swelling—recur from time to time. But in certain cases affections of the joints, such as effusions or ankylosis, do remain in chronic form after an acute attack. Henoch speaks of exostoses and bony formations in the muscles and tendons as liable to be developed in rheumatic children, occasionally to such a degree as to convert a large part of the muscular and tendinous system into a bony mass, rendering almost every movement of the body impossible.

For inveterately chronic cases, medicinal baths of sulphuretted waters, or brine baths, or hot springs of other kinds, afford the most hopeful treatment, the more stimulating waters or the simpler warm baths being used, according as the promotion of absorption or the relief of pain is the chief object. Wrapping the joints in wool impregnated with pine oil, which keeps up a constant mild stimulation, is an excellent plan in slighter cases. For the more confirmed and severe, small blisters repeated from time to time, or the application of oleate of mercury in mild form (from two to five per cent.), are more useful. Other counter-irritants, such as the tincture of iodine painted over the joint, or a stimulating and anodyne liniment of chloroform and belladonna, have their place in treatment. Internally, iodide of potassium affords perhaps some help in reducing the chronic congestion of the parts and the resultant exudation.

RHEUMATOID ARTHRITIS.

Definition.—A chronic disease of the joints, characterized by slow inflammatory and degenerative changes of the articular structures, and leading to distortion and other deformity.

Etiology.—Rheumatoid arthritis (arthritis deformans) can hardly be called a child's disease, for it is as rare in childhood as acute rheumatism is common, yet it must not be passed over without brief mention. Sir A. Garrod affirms that he has seen it in severest form in children of ten or twelve years; Dr. West records a few cases, and speaks of it as most rare. I have seen three cases in little children which presented all the symptoms of rheumatoid arthritis. Seeing that rheumatoid arthritis sometimes remains as a chronic sequel of genuine rheumatism, it might be expected to be more common in children than it is; yet I have never seen it in this relation. In each instance which has come under my observation it resulted from the direct and sustained operation of certain potent causes, cold, damp, feeble health, and other depressing conditions.

Pathology and Morbid Anatomy.—This must be presumed to be the same as that met with in similar conditions in adults, although I know of no instance in which this has been actually verified by post-mortem examination. The condition is, no doubt, one of chronic inflammatory changes affecting all the joint-structures, cartilages, bones, synovial membrane, ligaments, and extending even to tendons and muscles. There is more or less complete absorption of the cartilages, wearing away of the denuded bones, formation of new bony growths along the free margins of the cartilages, and the ossification may extend to the tendons and capsule. With these changes are associated wasting and fatty degeneration of the muscles and tendons related to the joint.

Symptoms.—The disease exhibits no special features in the case of children. It is associated with the smaller joints of the extremities, the form found in younger adult patients, as contrasted with that affecting the larger joints, which is, I think, solely a senile change. It begins probably in the same way with fugitive articular pains, then stiffness, especially after the joints have not been moved for some time, as first thing in the morning. Then comes a little swelling of the articular ends of the bones, with perhaps slight tenderness. Next follows some displacement, owing to altered shape, the formation of nodules, excrescences, and erosion of cartilage, and the parts grate on movement.

Perhaps the most striking part of the affection is the singular wasting of the muscles which are in relation to the joint. This is best seen in the hands, which are most often the first parts affected, when the atrophy of the extensors, together with the deflection of the phalanges to the ulnar or less commonly to the radial side, and the flexing of the fingers, gives the hand the peculiar claw-like appearance which is absolutely characteristic. With this are thinning and glossiness of skin over the affected parts.

All these changes are occasionally met with in their typical form in children, and, inasmuch as they differ in no respect from the symptoms met with in adults, require no detailed description here.

There is now at the Hospital for Sick Children, under the care of my colleague Dr. Sturges, a little boy three years old who presents the characteristic signs of rheumatoid arthritis. The child has had swellings of the joints with tenderness since the age of eighteen months. The fingers, wrists, elbows, shoulders, knees, and ankles are all affected. In the larger joints there is slight effusion, and in the elbow and shoulder crepitus on movement. The ends of the phalanges are enlarged. The muscles related to the affected parts are very greatly wasted. The lymphatic glands in the arms are greatly enlarged. The child has signs of rickets; and the question has been raised as to whether the condition might not be connected with this affection or with congenital syphilis. I examined the child several times, and came to the conclusion that the case was one of genuine rheumatoid arthritis.

Diagnosis.—It is extremely difficult to distinguish rheumatoid arthritis from the more chronic or subacute forms of genuine rheumatism until the characteristic deformities have been developed. Indeed, in many cases arthritis deformans would appear to be a later development or sequela of the acute form. But when the enlargement of the joints, the crepitus on movement, the wasting of muscles, the thinned glossy skin, and the distortion of the fingers arise, there is no further difficulty in determining the exact nature of the disease.

Prognosis.—Cases of this kind are so rare in children that but scanty means of forming a judgment as to its course and issue are available. The prognosis in the case of a child might fairly be expected to be more favorable than with an adult; and one or two cases where the attacks have been slight have apparently ended in recovery more or less complete.

But in the more severe cases rheumatoid arthritis is as persistent as with adults; although it is capable of being modified by treatment and is attended with no immediate danger to life. The feeble health with which it is associated is a cause rather than a consequence of the disease.

Treatment.—The affection being especially associated with enfeebled general health, all lowering or drastic treatment is not only useless, but positively injurious. I have often observed serious aggravation of all the symptoms to follow severe treatment by purges, alkalies, and colchicum, with low diet, prescribed under the mistaken impression that the disease to be dealt with was true gout, or genuine rheumatism, and on the other hand have seen remarkable improvement result from the change to more tonic treatment and more generous diet. Everything which tends to improve general health and nutrition is beneficial in rheumatoid arthritis. A warm, dry, sunny climate, simple nutritious food, and warm clothing are of the first importance. And locally as well as generally all severe measures are hurtful. The joints will not bear blisters or strong stimulating applications or counter-irritants. These tend to increase the morbid changes going on in and around the joints. A weak solution of iodine with glycerin—fifteen drops of the former and a drachm of the latter to the ounce of water—may be applied under oiled silk or the pine-oil wool, but nothing more. Gentle rubbing and exercise of the joint help to keep the movement free. Much benefit sometimes follows the administration of iodide of potassium and arsenic. Three grains of the latter, with two drops of liquor potassii arsenitis and two drachms of wine of iron in six drachms of water, may be given twice a day. In children cod-liver oil may be added with advantage.

But the means which do most good are hot baths, especially those of a stimulating kind, such as the sulphur waters and brine and mud baths. If circumstances preclude the patient from visiting the natural warm springs, sulphur baths made by adding four ounces of sulphur or of sulphate of potassium to thirty gallons of water will often answer the purpose sufficiently well.

MALARIA.

BY F. FORCHHEIMER, M.D.

MALARIA is becoming of great importance to us, as physicians and scientists, from year to year. Those who follow the theoretical aspect of the question find themselves more interested as they get more deeply involved in trying to solve its mysteries. To the practitioner, there is so much that is necessary even in his routine of daily work, the disease itself, its various manifestations, its relations to other diseases, etc., that the chapter on malaria is of the utmost importance. This has always been the case, but at the present day the subject has additional value, for no practitioner can go through his work without seeing a greater or less number of cases of malaria. If he lives in a malarial district, he becomes familiar with the disease; if he does not, the cases are brought to him for cure, in this day of rapid transit, on account of the fact that he lives in a non-malarial district. In this way, at times, he sees even those most violent forms which are seen only where the poison is most intense and active. It is said that malaria and civilization are antagonistic; yet in this country this can hardly be said to be true; but medical geography does prove that malarial districts are constantly shifting, so that a region that has been looked upon as perfectly safe suddenly becomes malarial (the valley of the Connecticut River, for instance, or parts of the Atlantic coast), and in the course of a practitioner's lifetime it may become highly necessary for him to be able to distinguish the various forms of malaria. At present the term "malaria" is frequently used as a charitable mantle to cover sins in diagnosis. Indeed, it has become one of the fashionable diseases: as our forefathers took their after-dinner pills or their blue mass for biliousness, so, to-day, the household is incomplete without sugar- or gelatin-coated pills of quinine. These are taken indiscriminately by old and young, by strong and weak, and for everything: it is all malaria,—the dyspepsia of the worn-out merchant, the hysterics of the æsthetic damsel, or the colic of the baby. After all, we, as physicians, are to be blamed for this, as it is so easy to tell a patient, and so satisfactory to him, "You are suffering with a slight attack of malaria, which a few doses of quinine will relieve." But with this comes an amount of mental indifference as to diagnosis, which has allowed an abomination like "typho-malaria" to obtain full swing, and which has cost many a patient his life in that the physician was mentally incapable or unwilling to draw his lines strict enough.

When it comes to children, we find the laxity still greater; for it is only comparatively recently that the whole of the study of children's diseases has been elevated above anything that would deal with more than teething, growing pains, or worms. As a result, the literature referring to children will be found very meagre; yet sufficient has been done to show that excellent observers have been on the alert and have watched many of the interesting manifestations of this poison in children, some of which seem to be peculiar to them.

Definition.—By malaria is meant a series of clinical pictures due to a specific poison. These clinical pictures have been divided into various groups which have been characterized by the type of fever that accompanies them: in this way we have the intermittent, the remittent, and the continuous forms. It has been found, however, that well-marked attacks of malaria exist without the production of fever, so that forms occur in which the symptoms may be of any one of these types—*i.e.*, intermittent, remittent, or continuous—without being characterized by an elevation of temperature. As will be seen when we come to the discussion of intermittents, these divisions are again subdivided; so that there is room for sufficient classification, which, however, is of comparatively little importance.

Etiology and Pathology.—Scientists, all over the world, are now trying to specify what the poison is, its habitat and its properties. It is but just to say that up to the present time we are justified in the conception that it is some lower form of life, which has eluded scientific proof fully to establish its acceptance. As early as 1717, Lancisi[1] stated that malaria was due to parasites which entered the blood; and from that time to this many things have been found, accused, and then not found guilty. It is within the memory of all of us (1866) how positively our own countryman Salisbury had discovered the essence of malaria, and what a pitiful end his wonderful and fearful experiences took. It would lead us too far to make note of all the researches into what always proved an *ignis-fatuus*, so that we start in the historical development of the subject with a something that seems at least tangible. In 1879, Klebs and Tommasi-Crudeli published their researches[2] in which they had investigated the air, the soil, and the water of the Pontine Marshes. They found a great number of lower forms of life,—cocci, bacteria, algæ, variously-shaped bodies. Of all of these there were left bacilli, only, after culture. These bacilli were from four to six micro-millimetres in length, they were mobile, and some contained spores and aërobies. When a pure culture of these was injected, in large quantity, into the veins of a rabbit, the animal got a fever. Objections were raised to these experiments as follows: animals that live in these malarial regions do not suffer with malarial affections; so many materials, when injected into the blood-vessels, produce fever; it was not proved that these animals experimented upon had anything like intermittent fever; and, lastly, the methods used were not above suspicion as to their scientific exactness. Crudeli[1] proves that fever can be given to rabbits by inoculation, that they have a chill, and that during the chill the animal contains the bacillus malariæ; that pure cultures of the bacillus can be made by taking blood from the spleen of the animal inoculated, and that these cultures, in their turn, will produce fever. This is a complete chain of evidence; but, unfortunately, other experimenters have been unable to verify these results completely. Some find nothing in the blood, others have not even been able to verify the possibility of causing malarial attacks in animals by the injection of affected blood. These investigations have, however, led to the discovery of a something in the blood which seems to be practically of great importance. Laveran was the first to describe bodies in connection with the red corpuscles of the blood (1881), and of these he claims that there are various kinds, two cystic and two mobile, representing different stages of the same parasite. Body No. 1 is semilunar, containing pigment, often attached to the red corpuscle, and somewhat larger than it. Body No. 2 is spherical, and varies in size, being sometimes as large as a red corpuscle, at other times very much smaller, and also containing pigment. Both bodies possess amœboid movement, and bodies Nos. 3 and 4 are formed from them. No. 3 is simply No. 1 or 2 with cilia, and No. 4 are the cilia or filaments themselves detached from their cysts, representing the fully-developed form of the parasite. In 1883, Marchiafava and Celli published their researches which eventually led them to accept as the cause for malaria the so-called Plasmodium malariæ. We make an abstract of their latest communications (1886). There are found in the red corpuscles of the blood, in subjects suffering with malaria, micrococci and masses of protoplasm, with all the characteristic properties of protoplasm. These bodies take up certain aniline coloring-matter,—fuchsin, methyl- or gentian-violet,—and should be examined fresh. Within them there is found reddish or black pigment, due to their action upon hæmoglobin, and not a part of their natural structure. When blood is taken from patients suffering from malaria and injected into the veins of healthy subjects, the result is infection with malaria; and these same bodies will be found in the infected subject. Marchiafava and Celli claim to have seen the micro-organism in division, and in this way, they think, reproduction takes place. They admit that Laveran has seen the right thing, but claim that his interpretation is incorrect, and look upon the various figures which he has given as representing different states or stages of their plasmodium. It was impossible to cultivate the plasmodium, and examination of soil resulted negatively, although the micrococcus was found, but not pathogenic as to animals. If we look back upon the investigations made, it will be seen, then, that we have two series,—the one referring to a bacillus, the second referring to a protoplasmic body which is now accepted as the plasmodium. The former series seems to have been dropped by investigators. Whether or not a bacillus or micrococcus will be found as forerunner of the plasmodium, as is hinted at in the publications of Marchiafava and Celli and more definitely expressed in an article by Von Sehlen, remains to be seen. The latter series has been favorably commented upon, and been taken up, so far as diagnostic value is concerned, by many authors (Welch, Councilman, Osler, Von Sehlen). In the most recent communications made by Crudeli[1] he states positively that the plasmodium, or pseudo-plasmodium, as he calls it, is the result of the Klebs and Crudeli bacillus. This "plasmodium" can be produced by a variety of poisons acting upon the red corpuscle, typhus and scarlatina, and in progressive anæmia. He had come to the conclusion that the blood-changes combined with the great quantity of pigment were characteristic of malaria; but the paper of Rosenstein has convinced him of the contrary. These researches still require verification, but they show that even the diagnostic value of the plasmodium malariæ is denied by excellent observers. Although we are not justified, in the present condition of the question, in saying that the plasmodium produces malaria, we can state that it is found in one form or another in greater or less numbers in every case of malaria. From this it will be seen how very important the subject is from a practical stand-point. Cases have been reported (Councilman) in which the absence of the plasmodium in the blood was sufficient to disestablish a diagnosis, and in my own experience two cases have occurred which have conclusively proved to me the importance of examining the blood in doubtful malarial cases. The one case was an atypical typhoid fever, in which, as there was a strong tendency to an intermittent type of fever, the diagnosis of intermittent fever had been made, corrected by the repeated failure to find the plasmodium, and in which the patient finally died of a perforation of the intestines. The typhoid lesions and perforations were all demonstrated by the autopsy. The other case was one in which the diagnosis of typhoid had been made, and in which the plasmodium was found during a chill and the case promptly relieved by a large dose of quinine. Not only in grave cases like these, but also in comparatively mild ones, neuralgias, intestinal troubles, etc., the plasmodium can be demonstrated, and, with patient search, a positive diagnosis can always be arrived at. On account of their practical importance, we have had copied the excellent figures given by Councilman in his article published in vol. i. of the "Transactions of the Association of American Physicians and Pathologists," and also an illustration from Osler's paper.

---

[1] See Laveran, Traité des Fièvres palustres, Paris, 1884.

[2] Arch. f. Experiment. Path., July, 1879.

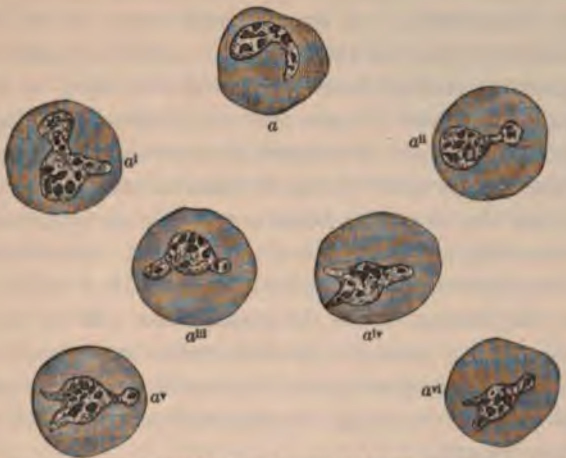
---

[1] Practitioner, 1880.

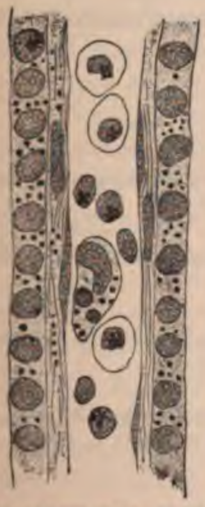
There are several things to be remembered in searching for this lower form of life. The nearer the chill the blood is taken, the surer we are of finding the organism, as it finds its maximum development about the time of the chill. In order to see the plasmodium, the blood must be spread out in a very thin layer and then looked at with a power not below 500–600 diameters. Staining is not necessary, but brings out the contours better, and fuchsin or methyl-violet can be used. The blood must first be dried

---

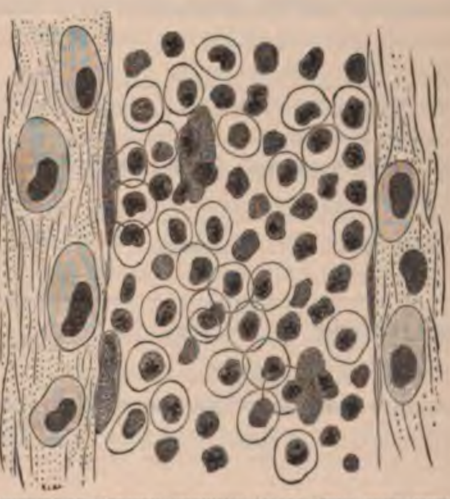
[1] Deutsche Med. Woch., 1887, p. 993, and Berlin. Klin. Woch., 1887, p. 695.



PIGMENTED BODY IN RED BLOOD-CORPUSCLE: outlined with camera ($\frac{1}{12}$ Zeiss, C eye-piece) by Dr. J. P. C. Griffith; illustrating some of the changes during an hour and a half's observation. a, at 11.45, slow alterations in outline, and the pigment-granules are in active dancing motion. a^{i}, 12.15. a^{ii}, 12.25, body has rotated as well as altered its shape. a^{iii}, 12.30. a^{iv}, 12.40. a^{v}, 1 o'clock. a^{vi}, 1.02. (From "Hæmatozoa of Malaria," by Wm. Osler, M.D., British Medical Journal, March 12, 1887.)



SMALL VEIN OF THE PIA MATER FILLED WITH PIGMENTED HYALINE BODIES. From a case of comatose malarial fever. Zeiss, obj. $\frac{1}{18}$, oc. 2. (Councilman.)



SECTION OF KIDNEY, SHOWING SMALL VEIN CONTAINING PIGMENTED HYALINE BODIES AND PIGMENTED LEUCOCYTE. Zeiss, obj. $\frac{1}{18}$, oc. 2. (Councilman.)

while passing the thin cover or slide over the flame of a spirit-lamp several times. The organisms cannot be kept for a very long time unless they are mounted in balsam. Morphologically, only the organisms within the red corpuscle are characteristic. I have several times found the crescentic bodies described by Osler in the blood of perfectly healthy individuals. Whether a great number of these present in the blood or in some other fluid, as the urine, in which I have also found them, would alone be sufficient to establish a diagnosis of malaria, it is too early to say. I have seen the plasmodium in very mild forms of malarial trouble, as well as in the very severe; but the more the blood-corpuscles are examined with high powers the more fully am I convinced that a great many bodies are found there which have been only incompletely described, and which to the superficial observer might seem to be the plasmodium. It is, therefore, necessary before beginning to look for the abnormal to make one's self perfectly familiar with the normal appearance: this should be done by using the same methods employed for detecting the plasmodium, and with the strongest lenses at one's command.

If we are to seek for the poison in the soil,—and all observers agree that this is its natural habitus,—it will readily be understood that certain conditions favor, and others, on the other hand, retard, its growth. Thus, temperature and moisture seem to be the quantities increasing or diminishing malarial attacks. It is not surprising, therefore, to see how various authors differ as to the time when most cases are met with, inasmuch as they live in different climates and environments. A casual glance at reports made from a few sources will be instructive as to when the greatest number of cases occur.

MONTHS.	COUNTRY.	AUTHOR.
July, August, September	Southern Russia.	P. Werner, Beobachtungen über Malaria, Berlin, 1887.
April and May	Sweden.	Geissler, Schmidt's Jahrbücher, 1880, Bd. clv. p. 78.
August to October . . .	Italy.	Geissler, Schmidt's Jahrbücher, 1886, Bd. ccxi. p. 72.
February to September .	Northern Germany.	Bohn, Handbuch der Kinderkrankheiten, Gerhardt, vol. ii. p. 448.

With us, it seems, there are two maxima, one in the spring and one in the early fall, although there is no month of the year in which we do not see some cases, either domestic or imported. A glance at this short table will show that in all of these authors the maxima come at a time when the temperature is not at the lowest.

So far as moisture is concerned, this seems to be absolutely necessary;

reports are common where malaria has appeared after a flood, and, *per contra*, where it has disappeared after the complete drainage of swamps. No better exemplification of the latter fact could be asked for than the table published by Asche[1] concerning the number of cases of malaria in the Algerian garrison at Boria. This little place, surrounded by swamps, caused a great number of deaths among the soldiers garrisoned there: as soon as drainage and cultivation were resorted to, the mortality and morbidity diminished, to be increased immediately the swamps were allowed to fill up again. In 1865, nine hundred and fifty-eight cases, with five deaths, were observed; while in 1880 there were only two hundred and forty-seven cases, with two deaths. The statement is also made that those who attended to the manual labor of draining suffered most. There is no doubt that civilization and cultivation are antagonistic to the intense forms of malarial intoxication, but cause an increase in the milder forms. There is not a sewer or a gas-pipe put down in any of our cities built upon malarial ground which does not cause an epidemic, more or less localized. The same can be said for the digging of foundations in soil which has been heretofore covered with grass and is upturned, exposing to the air the malarial poison which has lain dormant for years. Speaking from a local stand-point, those people suffer most from malaria who, from their position and wealth, should suffer least. This, of course, is true only of cities; in the country every one is alike, those living in villages perhaps suffering less than those in the open.

It is an accepted idea by both people and practitioners living in malarial districts that infection is more liable to take place between the hours of sunset and sunrise; and, although it seems impossible to settle this scientifically, there are many observations which seem to show the truth of this view. So far as children are concerned, this would seem a reasonable explanation for the comparative immunity infants enjoy, as they are not liable to be exposed to the poison at the above-mentioned time. There is no doubt of the fact that malaria sweeps along, causing epidemics, to places which have been non-malarial;[2] and, so far as the geographical distribution of the disease is concerned, it can be safely said that, at present, few countries are entirely free from it. Indeed, the picture is so variable that statements made ten years ago regarding the medical geography of malaria could not be accepted to-day. The tropics and subtropics furnish us with the most violent forms; the temperate zones produce a great number of cases, but not so intense; and even those countries lying far to the north do not seem to be entirely free.

Morbidity.—Malarial poisoning spares neither age nor sex. The effect of malaria upon pregnant women is frequently that of producing miscarriage,—whether from general intoxication or from infection and death of the fœtus it is difficult to say. A great many cases are on record in which children have been born showing the evidences of intra-uterine affection (cachexia, dropsy, enlargement of the spleen, etc.),[1] or have been taken sick with chills and fever shortly after birth. The period of infancy seems to be comparatively exempt, but far more cases occur during this period than is supposed, as, for a great many reasons, they are overlooked. Werner[2] says, "Mortality among children is enormously great, especially among infants." More statistic data in this direction would be very desirable, but with the proviso that infants be examined carefully. All authors agree that in children the period of from two to seven or eight years furnishes the greatest number of cases, and that in the beginning of an epidemic children are the first to be affected. After the seventh or eighth year of life their chances of infection are about the same as those of adults.

---

[1] Schmidt's Jahrbücher, 1885.

[2] See Stricker, Die Infectionskrankheiten.

Pathological Anatomy.—The prime and principal lesion of malaria is that of the blood. The latter is the carrier of infection, as has been repeatedly demonstrated by injections made upon man, causing typical attacks in the subject receiving malarial blood.[3]

This poison may act in two ways: first, generally; second, locally. Its general effect may be summed up as that of the introduction of almost any foreign substance into the circulation,—the production of chills, fever, etc. This series of symptoms is preceded by a period of incubation, which according to different authorities may vary from a few hours to a few years, but in the average number of cases is represented by two weeks. The local effects are due to an especial development of the virus at given places,—the spleen, the liver, the brain, the blood-vessels, etc. In these localities the great quantity of poison is demonstrated by its results, pigment, and, from the fact that symptoms of a local nature are produced, we are justified in the conclusion that wherever the latter occur, there a localization of the malarial poison has taken place (neuralgias, for instance). The effect of the poison upon the blood is a destruction of red corpuscles, an increase in pigment (directly depending upon it), and a diminution in albumen. The effect upon organs or tissues in which the poison or its results are lodged is the production of irritative changes leading to hyperplasia or hypertrophy. With these data in view, the lesions are readily understood. Of all the organs in the body, the spleen suffers earliest and most. During an attack the spleen is enlarged; this enlargement disappears, as a rule, when the fever disappears, to reappear with the next attack, and finally to become more or less permanent. This enlargement is due to hypertrophy and the deposit of pigment. Frequently there is inflammation in the capsule, sometimes peri-splenitis; rarely the enlargement becomes large enough to cause rupture. The liver also becomes enlarged, but later than the spleen: this enlargement is due to a similar process, but is characterized by an enormous deposit of pigment, especially around the radicles of the hepatic vein.

---

[1] See Duchek, Prag. Vierteljahresschrift, 1858, and Bazin, Gazette des Hôpitaux, 1871.

[2] Loc. cit.

[3] Gerhardt and others.

The disintegration of the red corpuscles gives rise to an almost endless number of changes. In very many places the lymphatic spaces around the vessels are filled with pigment, so that the contours of the vessels are emphasized,—as in the brain; at other times there is more or less diffuse deposit of this coloring-matter in the kidneys and the skin. On account of nutritive changes in the blood, the vessel-walls frequently become weakened, and then hemorrhages follow, under the skin, into the cavities of the body, or with the secretions. For the same reason, possibly, we have disturbances in the digestive apparatus, although they may set in so early as to be explained by a localization of the poison or lack of digestive power on account of fever. As a final result of the change in constitution of the blood, we have hydræmia or anæmia, with all its manifestations, local and general. This, with or without the deposit of pigment, gives rise to what is known as the malarial cachexia.

Local disturbances, of great importance to the pediatrician, are quite common in the bronchial tubes, so that it may happen that the patient survives his attack of malaria but succumbs to catarrhal pneumonia, the sequel to the malarial bronchitis. In the pernicious forms the lesions are characteristic, enlargement of the spleen, changes in the brain, hemorrhagic infarctions, etc., usually being found. The kidneys are more or less affected; even in mild forms this is often the case, and very few patients with chronic malarial poisoning will be observed without some more or less appreciable change present in the urine. Those who have seen most of the pernicious forms claim that there is a positive change in the heart; the myocardium is rendered softer, and its color is changed. Recovery from this is possible, although the case is very much complicated by its presence. Endocarditis occurs in some very mild forms also, but is not very common.

From this brief recital of pathological changes it will be seen that there is hardly a tissue or an organ in the body which may not be attacked by this poison; indeed, there are but few poisons that man is infected with which may produce such general changes. It will also be seen that diagnosis must frequently present difficulties, and how very important the plasmodium will become when its absolute value as a diagnostic feature has been positively settled.

SYMPTOMATOLOGY.

I. **Intermittens.**—This is by far the most common form of malarial poisoning we have to deal with. The attack presents itself in three divisions: first, the chill; second, the fever; third, the sweat. When the attack presents itself every day, it is called quotidian; every other day, tertian; every fourth day, quartan. There are also double forms,—double quartan, for instance, in which there is an attack on two successive days and one day without an attack, or double quotidian or tertian. In double quotidian we have two chills daily,—one in the morning, one in the evening;

in double tertian one chill daily, but the time of chill alternating every other day. In children the quotidian form is most common: Bohn gives the comparative frequency of the three forms as 3 : 2 : 1, although this must, of necessity, vary with the epidemic. The attack usually comes on between ten o'clock in the morning and one in the afternoon. It will be understood that this is the case in the great majority of instances: there is no time in the twenty-four hours when a chill may not come on, but for practical purposes it is always best to assume that an attack will follow the rule and not the exception. Authors differ very much as to this rule,[1] and possibly the time when infection has taken place or the method of infection may have something to do with the different observations that have been made. If anything has been established, it is the liability to relapses. Here, again, the time of relapse has been subject to very much discussion. Children are more liable to relapses than adults, and, depending upon the type of attack, these are most common on the seventh, the fourteenth, and the twenty-first day: this is true especially of the quotidian and tertian forms. The quartan form has a tendency to relapse on the eighth day, although changes of type from quartan to tertian or quotidian are by no means uncommon.

There are two forms of the intermittent type,—the pernicious and the mild form. In the pernicious form, which is not rare in infancy and childhood, the patient is taken sick suddenly. The child has been perfectly well, and suddenly may go into convulsions. Before this the parents may have observed that the child is restless, that it has assumed a bluish-pale color, perhaps that it has vomited or has had one or two loose passages. The physician who comes in will see a patient well nourished, in convulsions, with a very high temperature (104°–108° F. in the rectum); he will examine the patient carefully and find nothing, except perhaps an enlarged spleen, and this by no means constant. The convulsions may continue, the patient is soporose or comatose, the pupils are contracted or one is dilated and the other is contracted, lividity occurs over the whole body, the extremities get cold, and this first attack may end fatally. Or the convulsions gradually diminish in intensity and number, the extremities grow warmer, the bluish color disappears, the temperature begins to fall, consciousness returns between the convulsions, and towards evening the child seems comparatively well. The same attack may come again the next day, either weaker or stronger,—usually the latter, and may then end the patient's life. Or the attack comes on simply as an attack of coma in an otherwise healthy child, from which condition the patient never rallies, lying for from one to three or four days, and then dying from asthenia, œdema of the brain, or other complications. The convulsive form may leave the child in this comatose condition and the termination be in the same way as in the case where coma sets in immediately. Rarely do these pernicious forms terminate in the development of the benign intermittent, and the prognosis is almost invariably a fatal one. The absence of any positive symptoms renders it impossible to make a diagnosis with any great amount of certainty. There is no doubt that a great many children die of this form in whom the diagnosis of convulsions, congestion of the brain, etc., satisfies the practitioner. The importance of searching for the plasmodium malariæ in these forms cannot be too strongly dwelt upon.

---

[1] See Griesinger, Die Infectionskrankheiten, Virchow and Bohn, loc. cit.

In the benign forms of chills and fever we must discriminate two almost entirely separate clinical pictures,—the one occurring in infants and the one occurring in older children. In infants we rarely have a complete attack,—*i.e.*, one made up of chill, fever, and sweat. It is either one of these alone, or, most commonly, two together. The one link which is most commonly missing is the chill, the one which is always present is the fever. It is stated that very young infants do have chills (Bohn and others), and I have repeatedly been assured by physicians practising in very malarial districts that they have seen infants shake like grown people. I have never seen in an infant a true chill produced by malaria: it has always been represented by something showing symptoms of cold on the part of the child, but always fragmentary, and not like a well-developed chill. This fragment of a chill manifests itself as follows. The child begins to yawn, or stretches itself as if it were very tired; with this there is a change of expression and color about the face. The nose becomes pinched, cold; the eyes sink in and have bluish lines about them; the lips are blue, and the little one looks very tired. With this, the finger- and toe-nails become cyanotic, and if this occurs after a meal the patient vomits or feels nauseated. All this belongs to the mildest manifestations of a chill. The next step is the involvement of the nervous system. Here there is twitching of the eyelids or of the extremities, associated with what has been described above, which causes the physician to fear the next step in development, convulsions. A great many infants have convulsions at the onset of any acute affection, and we frequently find that in intermittens the chill is represented by a convulsion, which is then followed by the next stage. These convulsions, naturally, cause very much anxiety, because in and of themselves they are dangerous, and for the reason that at first the physician does not know whether he may not be dealing with something very much more serious than an ordinary attack of chills and fever. After the convulsions have ceased, —and they do this, as a rule, after a short time, not exceeding a few hours, the first one being usually the severest,—there comes the period of fever. During the chill period the temperature has gone rapidly up to 103°–108° F. (rectal), and remains there throughout the whole period of fever, sinking very gradually towards the end of this period, and after from three to five hours reaching normal or subnormal. With this fever there is more or less restlessness, the patient is very much flushed, feels very dry, is fretful and cries very much, and, as in the previous stage, may have gastro-intestinal disturbances. The sweat that follows is profuse when it does occur, and, although the little patient seems exhausted, the appetite returns and the patient seems perfectly well. Yet after one or two attacks—and this is true of older children as well—the cachexia begins to manifest itself. The patients lose their natural color, they are pale, sometimes jaundiced, listless, languid, have lost their appetite, and do not take as much interest in their surroundings as has been their custom. With this there is developed the enlargement of the spleen. It is rare, according to my experience, not to find the spleen enlarged in the malaria of children; yet excellent observers have made statements to the contrary.[1] It will be remembered, in this connection, that negative proof of the enlargement of the spleen—by palpation, for example—must not be taken as final; for there are numbers of cases on record in which the spleen was examined *intra vitam* and pronounced not enlarged, and yet post-mortem examination revealed a very large but very soft spleen.[2] Besides, we must bear in mind that the spleen in malaria, especially in the beginning of the disease, is enlarged at certain times only, to return to its normal condition after four or five hours. It will be seen, then, that, with the exception of the fever, not one of the features of a characteristic attack is always present in infants. But we have, in addition, other forms in which the poison seems to localize itself in a special spot. These will be described in connection with intermittens in older children.

The intermittens of older children does not differ materially from that of adults. They are able to describe their sensations, and they react like adults. There might be added to this that the cachexia develops much earlier with them than in adults, and that the gastro-intestinal tract is more liable to become affected, constipation, however, being more common than diarrhœa. They are subject to all the forms that are found in adults, and with them it is sometimes exceedingly difficult to arrive at a diagnosis. It is in them especially that we have intermittent, non-febrile conditions producing the most fantastic combinations of symptoms. All these combinations can be classified so as to give a summary of the various symptoms, but it is almost impossible to describe what collections of symptoms can be present in an individual case. The Germans call the form without fever intermittens larvata,—a very good name. We might call all these forms bastard, although some are very much more dangerous than frank, well-developed cases.

Manifestations on the Part of the Nervous System.—There is not a nerve in the body that seems exempt from affection by malaria. If we look at the brain and cord and its membranes, we see meningitis, œdema of the brain, peculiar forms of insanity. The form of meningitis most commonly seen is the cerebro-spinal. I have described some cases in connection with and developing from torticollis intermittens.[3] Since that time I have seen several other cases in which I was able to demonstrate the presence of the plasmodium, so that, to my mind, there was no doubt as to the malarial nature of the affection. Ziemssen[1] says there should be no difficulty in differentiating the two diseases; yet there can be no doubt that some, if not a great many, cases of sporadic cerebro-spinal meningitis are due to malaria. Sometimes, however, the cerebral meninges alone are affected, and we have the clinical picture of an acute lepto-meningitis. It is very rare for all these cases to develop as such in the beginning; they come from well-marked cases of intermittens, as a rule, with decided tendency to the production of symptoms on the part of the nervous system. The prognosis, although bad, is by no means so unfavorable as in other forms of meningitis.

---

[1] L. Emmett Holt, Amer. Jour. of Obstetrics, and Diseases of Children, 1883, xvi., for instance.

[2] Schneidler, Jahrbuch für Kinderheilkunde, N. F., xiv., 1879.

[3] Archives of Pediatrics.

Œdema of the brain is not uncommon in the violent forms, or as terminating a long-continued cachectic case. A slight accession of temperature or a complication arising will be the predisposing cause and be followed by death.

The mental disturbances of malaria in children are not so uncommon as we were accustomed to believe from the description of Griesinger and Bohn. Walliser[2] found eighty-eight cases out of eleven hundred of all ages, and of these over one-half were children under ten years of age. Of these, seven died of convulsions during the acme of the disease. Various mental disturbances have been observed in children. Somnolence is especially well marked in infants; after the second or third attack they go to sleep,—a sleep from which there is no waking,—and die in this condition. Kraepelin[3] divides the psychoses of malaria into three groups: 1, those forms that take the place of regular attacks of intermittens or alternate with them; 2, those that take upon themselves a remitting or continuous character after fever or mental attacks; 3, non-intermitting attacks after intermittens. Ten per cent. of all his cases were children. Post-mortems revealed œdema and hyperæmia of the central organs, sometimes deposit of pigment. In half of the cases active melancholia, with fear, a tendency to destruction, and hallucinations were the principal symptoms. In about one-third of the cases the paroxyms were preceded by an aura, sometimes tenderness over the cervical vertebræ. In two cases there were epileptic seizures followed by coma. In one-fourth of the cases there was mania accompanied by ideas of grandeur. The paroxysm lasted for several hours, and the whole disease persisted four weeks in thirty-two per cent., less than a week in twenty per cent. It was rare to find cases lasting longer than months or for one year. All the adults recovered entirely, and of the children only the seven cases, convulsive, mentioned before, died.

In the cerebro-spinal system of nerves, the symptoms that are produced usually manifest themselves as neuralgias. Frequently the attacks of pain are not accompanied by fever, but very often carefully-repeated measurements of temperature will show that there is a slight elevation. The fifth pair of nerves is the one most commonly affected. We have supra- or infra-orbital neuralgia, frontal or occipital headache, pain in the teeth and, sometimes, along the side of the nose. Neuralgias of the sciatic nerve, the intercostal nerves, and the nerves of the stomach are by no means uncommon. Then there is that peculiar form of nervous affection which has been called torticollis intermittens. There are three states of this condition, the first being purely that of torticollis, the second absolutely intermittent with high fever or continuous, and the third with brain- or cord-complications presenting the picture of a cerebro-spinal meningitis. The patient is attacked at a certain time in the day with pain in the back of the head and along the upper part of the spinal column. With this there is torticollis. The attack lasts for from two to five or six hours, and then the patient feels perfectly well. This is the mildest form. The next day the attack repeats itself, and behaves in every respect like an ordinary intermittens. The forms described above may run into one another, and, although at first very amenable to treatment, may develop so as to be beyond control. I have seen sixteen cases of this description in children, and possibly many more before my attention was attracted by one very well marked intermittent case. I should say, therefore, that a great many cases will run a favorable course even without treatment of any sort. These cases have been described by numerous authors (Henoch, Bierbaum, Bohn, L. Emmett Holt, and others), and must be looked upon as well-recognized forms of intermittens. The relation which they bear to meningeal troubles, to which I would call especial attention, makes them of especial importance.

---

[1] Handbuch d. Speciellen Path. u. Therap., article "Cerebro-spinal Meningitis."

[2] Schmidt's Jahrbücher, 1871, Bd. clxxx. p. 58.

[3] Ibid., 1882.

So far as concerns the nerves of special sense, there are no observations on record which go to show that they are especially affected. It is not uncommon for patients to complain of peculiar sensations of taste, or that they hear noises, etc. All this, however, can be explained by other means than by taking into consideration special lesions of the nerves.

There are also disturbances of the vaso-motor nerves which cause peculiar symptoms. Among these, special reference is made to intermittent swellings, especially well marked about the joints, and sometimes within them. They give rise at times to great pain, at other times they are painless. On account of their ill-defined character, they are sometimes confounded with hysterical joint-lesions or tubercular affections. Where enlargement of the spleen exists, the diagnosis is easy; where it is absent, anti-malarial treatment will make clear the nature of the malady.

Affections of the Respiratory Apparatus.—The whole of the respiratory tract, from the mucous membrane of the nose to the alveoli of the lung, may suffer from malarial poison. Sometimes we observe true intermittent attacks of coryza, or, combined with this, pharyngitis or enlargement of the tonsils. One alone, or all combined, may exist. The most common form of catarrhal trouble due to malaria is a subacute or chronic condition extending over the whole mucous membrane of the pharynx, nose, and eyes. We also have attacks of epistaxis: these may become dangerous to life on account of great loss of blood during the attack, or on account of repeated attacks. It is advisable to examine the nose in these cases; for frequently there will be found that peculiar ulceration upon the septum which is readily treated. In order to prevent relapses, however, the general condition must be taken into consideration. In the larynx there are produced a series of symptoms resembling croup. Briand[1] has described this affection as attacks of high fever coming on at more or less regular intervals, combined with intense redness of the pharynx, hoarseness, stridor, and dyspnœa. These paroxysms may last for hours; and the whole affection resembles croup. Although he calls attention to the difficulty in diagnosis, there ought to be no such difficulty if the time of attack, enlargement of the spleen, periodicity, and intervals of almost perfect health be taken into consideration. Briand says that the prognosis is very much better than in croup, because the disease can be readily treated. Attacks of bronchitis more or less diffuse, as the only symptoms of malarial infection, also occur. If they are in the capillary bronchi, they may become very dangerous. As it is, they must always be carefully watched, and the patient be given the full benefit of liberal treatment.

So far as the relations of pneumonia to malaria are concerned, there may be several positions taken. There are those who assert that malarial poisoning may produce pneumonia; then, again, those who maintain that pneumonia develops in patients having malaria, and therefore takes upon itself a malarial type. While there can be no doubt that pneumonia (catarrhal) is produced by a multitude of causes, diphtheria, measles, typhoid fever, etc.,—*i.e.*, by the special poisons of these diseases,—it has yet to be proved that malaria is one of these. For fear of being misunderstood, let us say that nothing which has been attributed to malaria (bacilli, micrococci, plasmodium) has ever been found in patients who have died of malarial pneumonia, although everything points to the fact that the poison of malaria could act in the same way as that of any other disease. However this may be, there is a distinct clinical picture in which patients have pneumonia the symptoms of which take upon themselves quotidian, tertian, or quartan exacerbations. These exacerbations are as to fever and the concomitant subjective and objective signs of pneumonia. This form is not more dangerous than any ordinary form; the exacerbations, as a rule, become weaker from day to day, and finally disappear, or are readily prevented by the use of quinine. Almost the same clinical picture is presented when a croupous pneumonia develops in a patient already malarial.

Manifestations in the Alimentary Tract.—Very few cases of intermittens occur without some symptoms being produced in the alimentary canal; but these complications have been treated of before, and we now refer to those forms in which the symptoms on the part of the alimentary canal are the principal manifestations. The stomach, the small intestine, and the large intestine, either alone or together, may be the seat of disturbance which alone goes to make up an attack. On the part of the stomach we have the symptoms of dyspepsia, either constant or intermittent. These attacks are entirely independent of any food that is taken. The little patient may be fed in the most approved and physiologically correct manner, and yet the attacks continue. That form which manifests itself in attacks of vomiting is very peculiar; the child may be in perfect health, playing about, happy and jolly, when it is suddenly taken with the ordinary symptoms of nausea. Then vomiting comes on. After four or five hours, the child, although looking dragged out, seems perfectly well, its appetite returns, and it remains well until the next attack comes on. With this there may be a slight elevation of temperature (101°–102° F.), the spleen is usually enlarged, as it is in every form of malaria in children, and when these attacks continue the little patient suffers very much so far as general health is concerned. It may be well to state that all the symptoms coming from the alimentary tract in malaria are most easily controlled by quinine.

---

[1] Gazette des Hôpitaux, 1883, No. 40.

On the part of the intestine we have diarrhœas. These are of two kinds, the large and watery stools and the small, slimy, bloody ones. The attacks consist simply in having these stools. There is no pain, as a rule, except in the large intestinal variety; the patient does not suffer inconvenience, and after the attack is over he is perfectly well. The diagnosis of these gastro-intestinal forms is readily made; the fact, alone, that all the remedies which usually control diarrhœa, combined with proper diet, fail, is sufficient to cause the practitioner to suspect that he is dealing with malaria. The prognosis is favorable in all these forms. I have seen little patients who have suffered for months from the large intestinal variety apparently in good health. It must not be forgotten, however, that there may be deeper lesions present in the intestines which may lead to very unpleasant complications,—tuberculosis, peritonitis, etc. In the violent forms as they are found in the tropics, all grades and characters of intestinal lesions have been observed, from the simple loss of epithelium to the most perfectly pronounced diphtheritic process. Fortunately, these hopeless forms are very rare in civilized countries.

Manifestations on the Part of the Organs of Circulation.—It seems strange that attacks on the part of the heart are so rarely noticed. They are certainly very rare; otherwise, more cases would be found recorded in the literature of malaria. An irregular distribution of blood is noticed in that form in which vertigo, with congestion of the face, is the only symptom. This dizziness is the only thing the patient complains of, but it returns with the same regularity that characterizes all these forms. Another form consists in regularly-recurring attacks of palpitation of the heart, easily controlled by quinine.

A. S. Smith[1] has put upon record a case of "malarial affection simulating Basedow's disease." A boy of eleven years was suddenly taken sick in the evening with enlargement of the thyroid gland, which disappeared the next morning. The next evening this returned, and with it palpitation. Over the thyroid gland there was heard a loud systolic bruit. The pulse was 110, temperature 101.5° F. The spleen was very much enlarged. After ten days of energetic treatment with quinine, all these symptoms were removed permanently.

---

[1] New York Medical Record, xxx., Nov. 20, 1886.

Manifestations on the Part of the Urinary Organs.—The two principal manifestations of attacks in the urine are hæmaturia or albuminuria and glycosuria. In regard to the first, it consists of the appearance of blood in the otherwise normal urine. This blood is discharged with or without pain, depending upon the fact that it coagulates in small or large masses, is usually of a bright-red color, and its loss does not affect the patient very much. In the urine of patients suffering from hæmaturia intermittens is found the plasmodium malariæ, but especially are found in great numbers those crescentic bodies referred to under the heading of etiology. In a few cases I have seen intermittent albuminuria without blood. Contrary to the experience of some authors, I have found kidney-complications comparatively common in children, the most common form being a subacute or even chronic albuminuria, in which the urine is almost characteristic of the disease. When it is passed it is of a brownish color; and when it stands for a little while, a brownish-black deposit settles, over which the urine is perfectly clear. If this deposit is examined under the microscope, it will be found to be made up of small masses of pigment, concerning whose nature or origin nothing is known. Besides this we find casts, amorphous urates, or crystalline forms. The urine always contains albumen in variable quantity, from a trace to two per cent. or over. These cases, as a rule, are of slow recovery, but the prognosis is not bad. They are not affected by quinine, but removal to a non-malarial climate gives relief in a very short time.

No cases are on record in which glycosuria represents the principal symptom of the attack. But the importance of examining the urine for sugar is shown in the following table of Bardel, quoted by Blau:[1]

In 134 quotidian cases glycosuria was found 29 times, = 22 per cent.
" 122 tertian cases " " " 17 " = 14 "
" 76 quartan cases " " " 11 " = 14 "
" 11 pernicious cases " " " 32 " = 80 "
" 40 well-marked cachectic individuals glycosuria was found 32 times = 80 per cent.

Jaccoud and Vallin assert that this temporary diabetes may become permanent. This must certainly occur very rarely. In the first place, diabetes is not common in childhood, and, secondly, malaria is very common; so that the relation of one to the other, in childhood, must be extremely remote.

---

[1] Schmidt's Jahrbücher, Bd. cciv., 1884.

Manifestations on the Part of the Skin.—There is hardly any skin-disease whose origin has not been ascribed to malaria. But no attempt at proof has been offered that there exists any relation between these affections and malaria. In the chronic forms there is no doubt that malaria does produce certain forms of skin-disease, which will be referred to hereafter. The only form of skin-disease which may take the place of an attack is urticaria. This is comparatively rare. Hebra had seen five hundred cases of urticaria in twenty-five years, and not one of them malarial. Zeissl in two hundred cases has seen but one case of intermittent urticaria. However, this is not to be looked upon as an absolute criterion, as Vienna is a non-malarial place. They are very much more common than is represented by these figures, as any one who lives in a malarial region can testify. There is nothing characteristic about the attack, except that it occurs like any attack of intermittens and that the spleen is enlarged. As a rule, they are very amenable to treatment.

Organs of Special Sense.—In the ear, attacks of neuralgic pain (Voltolini), furunculosis of the external meatus (Weber-Liel), injection of the tympanum (Voltolini and Hotz), and periodic attacks of middle-ear trouble (Hotz) have been observed. In a great many of these cases the only evidence given of the malarial nature of the disease is the fact that quinine removed the trouble. It certainly must be admitted that this evidence is not conclusive, especially when the quinine has been given in very small doses (Voltolini and Hotz). Some of the cases, however, are beyond doubt, and prove that the poison may localize itself in the ear as well as anywhere else. Concerning the effects of malaria upon the eye there are many observations which have been published, but none which show that any manifestations taking the place of a chill occur in that organ.

Remittent Fever (including continuous forms and the so-called Typho-Malaria).—We here come to manifestations of malaria which are exceedingly difficult of diagnosis, and for whose entity, as malarial, little if any proof has been offered. If we analyze the symptoms, we shall see that not one of those which we have seen to be characteristic of the affection is present in great constancy. Even the effect of quinine is recognized as having lost its specificity in these cases. So that, practically, the only reasons we have for calling these forms malarial are that they occur in malarial regions, that they occur at those times of the year when malaria is most common, that they occur in epidemics for which there seems to be a common cause, and that the lesions resemble those of the intermittent forms. To illustrate this latter statement: In 1884, a street in Cincinnati which had been torn up late in the preceding winter was again dug up for sewage purposes. Along that street a great number of cases of remittent and continuous fever developed which could not be called typhoid, and for whose existence no cause could be found except the disturbance in the soil of the street. These cases corresponded more or less with the descriptions given of malarial fever, and were therefore called such. Since the discovery of the plasmodium and its application to practice, nothing has been published to warrant us in taking advanced ground on the position taken above. It is possible that the same poison in different states of development may in the one instance produce an attack of intermittent, and in the other a continuous form. Until these questions are settled, however, it is the duty of the physician to give his patients any benefits that may arise from inability to make a positive diagnosis. We refer here especially to the difficulty of differentiating between typhoid fever and some of the forms of malaria. Whenever such difficulty arises, the patient must be treated for typhoid fever, and not for malaria, for reasons that are manifest. In the extensive epidemic of typhoid fever that existed in this country in 1887–88 many physicians persisted in the opinion that they were dealing with malaria, and would not be convinced of the contrary until they were shown the characteristic typhoid lesions. To such an extent has the malarial idea gained foothold, especially in malarial regions, that typhoid fever is not recognized when it does occur. But the weight of professional opinion is so much in favor of the acceptance of at least two forms, the remittent and the continuous, that a description follows with the reservations mentioned before,—that these forms may be due to a different stage of the malarial virus, or that we are dealing with an entirely different poison, as no direct evidence exists as to any of the three propositions.

Remittent fever usually comes on suddenly, without prodromata of any sort. The patient, who has been feeling very well, is suddenly taken with headache, and pain in the back or limbs; he has malaise, anorexia, perhaps feels slightly nauseated or vomits. Combined with these there is chilliness, or repeated chills, slight in degree, especially in coming in contact with cold air, or, if he is in bed, when uncovered. He may have a slight dry cough, so that the diagnosis of a cold has been made. The physician upon examining the patient will find him with flushed face, unusually bright eyes, more or less restless, and complaining that he feels very badly. Upon examination there are found a rapid pulse and a comparatively high temperature (102°–104° F.). The tongue is usually coated, yellow at the back, the breath slightly offensive. Examination of the chest reveals the signs of slight bronchial catarrh. The spleen is usually enlarged. The bowels are constipated, but nothing abnormal is found about the abdomen. On the second day the temperature is lower, and the patient perhaps better in every respect. This condition lasts for from three to five days, and recovery takes place. Relapses are common after the lapse of five to seven days. The relapses are of the same character as the first attack, as a rule. It is rare to find this form of fever lasting longer than from five to ten days. Such is the clinical picture as we see it in our zone. In the tropics or subtropics this form of fever may be pernicious. Werner[1] makes the following divisions: 1, the ordinary form (which has just been described, and from which, as he says, there are no deaths); 2, the adynamic form, a low fever, with typhoid symptoms, attacking especially those that have had previous attacks of malaria, prisoners, badly-developed persons, etc.; in this form the mortality is about six per cent.; 3, the comatose form, high temperature, 104° F., continued coma, death not later than the fourth day; mortality, among adults, fifty per cent.; 4, the hemorrhagic form; bleeding from the nose, under the skin, from the bowels, etc.; death between the sixth and sixteenth day; mortality, seventy-one per cent. All these forms are characterized by enlargement of the spleen, sometimes very great, and frequently by the deposit of pigment.

---

[1] Loc. cit.

For the description of the continuous form we cannot do better than quote from an excellent article by Dr. Maury, of Memphis, Tennessee:[1] "Its invasion, instead of being abrupt, as is the case with remittent, is sometimes marked by prodromes. In many cases the patient has been ailing for a week before going to bed. In other instances he will have had a repetition of the chills for two or more weeks, at irregular intervals, when finally the fever which follows the chill assumes a continued form, and goes on rising gradually, day by day, until on the sixth or seventh day, when the temperature has reached 103½° or 104°. In other cases there are no premonitory symptoms. The patient is attacked in the midst of apparent health, without appreciable cause, having had no previous manifestation of malaria. The fever presents a stadium of increase of about one week, a stadium of height of five or six days, and a stadium of decrease which terminates completely on the twenty-first day.

"Its thermometric range is decidedly lower than that of typhoid. It seldom goes above 103½°. Vomiting of bile is quite a common symptom during the first days of the attack. Bronchial catarrh is generally present. Constipation and a concave abdomen are marked features. Appreciable splenic tenderness or enlargement has been so rare in my observation that from memory I can recall but two cases in fifteen years. . . . All the essential features of typhoid or enteric fever are absent. There is no diarrhœa, no ileo-cæcal tenderness or gurgling, no meteorism, no eruption of rose-colored spots. As a rule, there is entire absence of abdominal symptoms. In some instances, where the patient's previous condition was bad, perhaps the system undermined by malaria, and where he was unfavorably situated for treatment or had no treatment, I have seen diarrhœa, dry, red and shining tongue, sordes, and low delirium, with picking at the bedclothes, and a condition closely resembling typhoid.

"In quite a large proportion of cases of this fever a prominent feature is neuralgia. It will come about mid-day and continue until after midnight, —in some instances so severely as to demand the hypodermic use of morphia. In one case the pain will be supraorbital and extend to the side of the face, neck, and shoulder; in another the neuralgia attacks the intercostal nerves of one or both sides so severely that there is dyspnœa from inability to expand the chest. In a third I have seen this pain localized in the right side, over the region of the ascending colon, extending from there upward into the chest and downward to the hip. In a fourth it is in the abdomen. These neuralgias are periodical, coming on in the forenoon."

---

[1] Amer. Jour. of Med. Sci., vol. cl. p. 395, 1881.

It will be seen from this graphic description how absolutely impossible it would be to differentiate this form from typhoid fever. The points of differentiation that are given are certainly not sufficient to warrant any one in excluding typhoid fever absolutely. Every clinician has seen cases of typhoid fever to which the description given by Dr. Maury would apply perfectly. Now, if, in addition, cases present themselves *with* more or less abdominal tenderness, *with* diarrhœa, *with* enlargement of the spleen, *with* an eruption, it seems almost impossible to realize that it is not typhoid but malarial manifestations we are dealing with. And yet every one practising in a malarial region has seen these cases called typho-malaria. It is not the individual case which ought to influence us, but the general picture of such an epidemic. From this we shall be able to judge whether it is typhoid fever or continued malarial fever we are dealing with. One thing ought to be added to Dr. Maury's description,—that relapses do occur. I have seen three relapses in a child eleven years of age; and this could be used as an argument against the typhoid nature of the fever. Not that relapses do not occur in typhoid fever, but they are more common in those mild attacks which have been called malarial. The whole subject must be left for decision, however, to the microscopist and the bacteriologist: it seems impossible on clinical grounds to come to a positive conclusion.

So far as the subject of typho-malaria is concerned, this is, fortunately, a thing of the past. A disease is either typhoid fever or malarial fever, or a combination of both. Such a thing as typho-malaria *per se*, as an entity or the result of an individual poison, does not exist. While the term may be a convenient one to use, as expressing something more serious than malaria and less dangerous than typhoid fever, it is one that has done and is still doing serious harm to the profession as well as to the layman. The only *raison d'être* it possesses is for the latter; and very little consolation it is to him to have the patient die from an intestinal perforation or hemorrhage as a result of typho-malaria. We repeat that whenever a case is doubtful, for the good of the patient as well as of the physician it must be called typhoid fever and treated as such.

Chronic Malaria and the Malarial Cachexia.—Malarial cachexia is the usual concomitant of chronic malaria in children. This is the case in the majority of instances; although there are mild forms of chronic malaria in which the general health does not seem to suffer at all. On the other hand, we have the cachexia developed in patients who are constantly exposed to the effects of the poison, without the production of active symptoms. Children having the cachexia present well-marked external evidences. They are emaciated, extremely anæmic, and the pigment deposited in their skins gives them a peculiar color. I have seen all the colors from a yellowish tint to a brown, almost the same as in Addison's disease, well marked. The children are dull, listless, have lost their appetites, usually suffer from some gastro-intestinal derangement, dyspepsia, constipation, or diarrhœa; they have a little elevation of temperature every evening, and they are very weak. The spleen is enormously enlarged, always painful upon pressure; if the child is old enough it will complain of pain in the region of the spleen. The enlargement of the spleen arrives at its greatest development in this form of malaria; it reaches to the umbilicus, towards the right, and sometimes as low as the pelvis; its contours may be sharply defined, its edges well rounded, and its resistance great, so that the idea of amyloid degeneration suggests itself, which is frequently proved upon post-mortem examination. At other times the spleen also enlarges upward, impinging upon the diaphragm and pushing aside the thoracic organs. The liver is also enlarged, and frequently becomes amyloid. Frequently the kidneys also become affected. At times there is set up an hydræmic condition, giving rise to dropsies,—anasarca, ascites, or serous effusions into the various cavities of the body. Embolic or thrombotic processes are not uncommon,—in the skin, producing petechiæ, gangrene; in the sinuses of the brain; in the lungs; in the kidneys. It is remarkable how, in some cases, death may be almost imminent and yet the patient taken to a non-malarial climate may still recover. On the other hand, a trifling illness, a follicular angina, a bronchial catarrh of comparative unimportance to a healthy child, may close the chapter in a malarial cachectic.

It would be idle repetition to go over the various forms under which chronic malaria can manifest itself. In general terms, the manifestations are the same as those described under intermittens. The symptoms become continuous or prolonged over a great period of time, and the patient suffers until relief comes, until the malarial poison has been eliminated, or until death terminates the disease. To these there must be added some affections not mentioned before. In the eyes, Kipp[1] has described keratitis, Brill[2] intraocular hemorrhages, Landsberg[3] hemorrhages in the region of the macula lutea, opacity of the vitreous, and iritis, and Prucet[4] choked disk and pigment-deposits. Troubles in the mouth are common in chronic malaria, from the simple stomatitis to cancrum oris,—the latter sometimes ending the life of a cachectic subject. Stomatitis ulcerosa is very common, and unless the cause be removed the disease itself will go from bad to worse. Patients with malaria frequently have mouths which resemble those of scorbutic patients. Indeed, the differential diagnosis is impossible from the appearance of the mouth alone. Oppenheim, in Heidelberg, has tried to establish the existence of a relation between rachitis and chronic malaria.

---

[1] Trans. Amer. Opth. Soc., 1880.

[2] New York Medical Record, xxx., 1886.

[3] Archiv f. Kinderheilk., xiv. 84.

[4] Ann. d'Ocul., iv. 39, 1878.

While his arguments are very ingenious, facts do not seem to carry out his views. We cannot leave this subject without calling attention to the great difficulty that exists in recognizing the nature of chronic malaria. Only he will succeed who tries to find the cause for symptoms: he who is satisfied with treating symptoms alone will never recognize this form of trouble, and, therefore, will never have results.

Prognosis.—The prognosis must be pitched for each individual case. The elements to be taken into consideration are the age of the patient, the form of the affection, and the nature of the surroundings. *Pari passu*, the younger the child the worse the prognosis, the older the better. Pernicious forms always mean a bad prognosis; and the more rapid and violent the onset the more serious the case. Nervous complications on the part of the central organs always increase the danger. The surroundings must be taken into consideration. A highly malarial district will produce worse forms than one in which malaria is beginning to disappear. The physician himself comes in as a prognostic factor. The sooner the diagnosis is made, the more energetic the treatment, the better the results. Sometimes the giving or the withholding of a large dose of quinine means life or death to a patient. For the latter's sake, it is far better to see malaria too often than too rarely, provided the symptoms are of a *foudroyant* nature. The children of wealthy parents, as in almost every other form of children's disease, will do better than those of poor ones: the former can be removed to a non-malarial region, while the latter have to remain, taking in new poison and going from bad to worse.

Treatment.—I. *Prophylaxis.*—Much can be done to prevent the introduction of malarial poison into the system. In a malarial region children should be kept in the house from sundown to sunrise. They must be required to live more strictly than otherwise according to the physiological rules for diet, clothing, exercise, etc. A great many observations have been made which tend to show that a person may live for a long time in a malarial region and escape infection, then be infected by malarial poisoning after excess in eating or drinking, or after some nervous effect, such as fright, sorrow, etc. The reports concerning the beneficial prophylactic effects of the continued use of small doses of quinine vary very much, some praising highly, others detracting just as positively. Some articles of diet are looked upon as liable to produce malaria; in the South of the United States the nutmeg melon has been looked upon as a bugbear, notably by the colored population. It is not at all probable that any article of diet will carry malaria; but indigestible food should be avoided. When any digging of earth is to be done, if at all feasible, it should be done when the temperature averages below 50° F.,—lower, if possible. Places in which the earth has been freshly dug up are to be especially avoided. There is no protection against malaria like sodding: the sod forms an almost impermeable covering for the earth and prevents the malaria from "rising." It is said that the cultivation of the eucalyptus-tree will render a malarial region non-malarial. The books are not closed on this subject yet, and it is too early to judge conclusively thereupon. In the prevention of malaria it would be very fortunate if the overflow of rivers and creeks could be prevented. This, with advanced civilization, will undoubtedly be accomplished. As yet no other means, such as chemicals, have been found to destroy the malarial poison; but sanitary science has done more to change the geography of malarial distribution than that of almost any other disease. Yet still more should certainly be done, especially in cities, in which the turning up of the earth ought to be controlled by public authority. The physical discomfort, to say nothing of loss of life and money, occasioned to individuals by the putting down of a series of pipes is often but inadequately counterbalanced by the gain to the community in having these pipes. It should be the duty of every sanitary officer to superintend all changes that are made which imply turning up of the earth, laying foundations, etc., so as to reduce to its minimum the chance of infection by malaria. The individual has a right to demand this of the communal government, and the community will reap an enormous profit by a little care in this direction. Any number of examples could be mentioned, in different cities of this country, in which a local epidemic of malaria was started by digging up a street or by laying the foundation for a large building.

II. *Medicinal and Otherwise.*—Quinine is the sovereign remedy against the manifestations of malaria. A host of remedies have been recommended to take the place of this one, but we have always come back to it. The administration of this drug to children is no easy matter. Quinine, to break up some forms of malaria, must be given in large doses; it is excessively bitter; a great many children cannot bear it upon their stomachs. In order to overcome these difficulties, the physician must be thoroughly well acquainted with all the various modes in which quinine can be given. First, as to dose; in order to prevent the recurrence of an intermittent attack, the quinine must be given at one dose, or, what practically amounts to one dose, the whole quantity administered within half an hour. On account of the fact that the drug manifests its maximum effects about four hours after administration, it will have to be given in the majority of cases between six and half-past six A.M. Many a time have I had the following experience. An intermittent, quotidian attack has been coming on at five o'clock in the evening. The next day the patient is advised to take his quinine at one o'clock P.M. The attack does not present itself with such violence, but the patient complains of having felt badly, say, at noon; so that he is ordered for the day after to take his medicine at eight o'clock in the morning. That day he complains of feeling badly, but much less so than before. The next day, perhaps an increased dose is administered at the normal time, and the attack is broken up. There are exceptions to this rule, however: they represent those mixed-type cases, or some, very few, cases in which the attacks come on in the evening or night. The explanation for the apparent unreliability of the patient's statements is to be found in the fact that the maximum but not the beginning of the attack presents itself at the time when the patient states it. Now, I make it the rule, after having tried one large dose to accord with the patient's observations, to give the quinine at the normal time, and usually I succeed in breaking up the attack. Sometimes the patient's observations are correct, and the application of a sufficiently large dose of quinine will either prove or disprove his statements. Another important matter is to prevent relapses. In the simple forms, with the exception of the quartan, the relapse, if it comes, will appear on the seventh, the fourteenth, and the twenty-first day after the first chill. For the quartan form, which frequently changes its type, it is the eighth, sixteenth, and twenty-fourth days. On these days, at the normal time, the full doses of quinine must be given. The percentage of relapses differs at different times of the year and in different places, but I never allow a patient to go without his quinine, after an attack of intermittens, on the day of his relapses, unless several examinations reveal the spleen reduced to its normal size. Even then, on account of the uncertainty of diagnosis of enlarged spleen, relapses will occur: so that the safest plan is to give the patient his full dose of quinine four times. In some cases, of mixed type, it is almost impossible to calculate when the day of relapse will be; and in these cases it is advisable to give the full dose on two or three consecutive days, in this way positively precluding a relapse. The dose should be sufficiently large: it is better to give a little too much than too little.

Much has been said about the permanent ill effects of large doses of quinine. Where specialists are dealing with a condition which, according to their own observations, may be produced by malaria, it is rather risky for them to assert that the remedy produces the malady, without proving the statement beyond a doubt. The normal dose for intermittens that I use is as follows: below six months of age, one to two grains; from six months to one year, two to two and a half grains; from one year to two years, two and a half to three grains; from two years to five years, three to five grains; and from five to twelve years, five to eight or ten grains, depending upon the size, the strength of the patient, and the return of the affection. Quinine can be administered by one or all of the methods used in giving drugs,—by the mouth, the rectum, the skin. There is no method known by which the bitter taste of quinine can be effectually disguised to make it applicable to the administration of sufficiently large doses. If the sulphate or muriate be used, liquorice is the best vehicle, the Syrupus Liquiritiæ (Glycyrrhizæ) Aromaticus of the German Pharmacopœia being the best preparation. The quinine is here given as a mixture (never add free acid, as it produces a precipitate), but one drachm will not cover over the taste of more than one grain of quininæ sulphas. For the latter reason, the applicability of this method is very limited: just where we need it most, in children from three to five years, it cannot be used, on account of the large quantity of vehicle required. When the patient can swallow pills, this mode of giving the remedy is to be preferred. To the practitioner using very much quinine it is not necessary to state that he should be careful whose pills and what kind he prescribes. One is tempted to make remarks about how the dose of quinine has been increased within the last ten or fifteen years; but the question arises, how much the administration of prepared pills has to do with it. The tannate of quinine is a preparation which is very serviceable in about double the dose of the sulphate. This, given with pulv. glycyrrhizæ or in the form of the chocolate tablets, can be used in older children where no other preparation will be taken. The objection that applies to the liquorice mixture does not apply to the tablets, for when children take them at all they are not particular as to number. The results are not so satisfactory as those obtained with the sulphate, but the tannate is very valuable, especially in malarial intestinal catarrhs. There are children who, although good and willing, cannot take quinine, as their stomachs will not retain it. These children swallow quinine without resistance, but as soon as they have it down it is vomited. This is not due to any general effect, as Binz has stated, but simply to local irritation of the gastric mucous membrane. Two methods are open in such children,—the administration of the drug by other channels than per os, or the exhibition of one or other of the alkaloids of Peruvian bark. In one or two cases the patients have been able to bear cinchonidia where quinine was always regurgitated: otherwise there is no advantage in giving substitutes for quinine, especially when we take into consideration that these substitutes have the same bitter taste and must be given in larger doses. The question of difference in cost is unimportant.

In giving quinine by the rectum, in which way it works just as promptly as per os, double the dose must be used. Two ways are open to us,—by injection and by suppository. It must be confessed that the latter method is much more satisfactory than the first. The principal objection to it is that there is great difficulty in causing the patient to retain the remedy; more or less irritation of the rectum is bound to follow, and upon this depends the capability of the patient to retain the quinine. For injection I suspend the quinine in sweet cream: any bland fluid, such as an emulsion of sweet oil, would undoubtedly do as well. The cream, if good, is not coagulated by the quinine, and is retained as well as any other fluid I have used. Suppositories cannot be made larger than to contain five grains each: although this is sufficient for infants, for larger children two or more are necessary. Great care must be exercised in making these suppositories, as the quinine has the property of crystallizing on their outer surface, and in this way causes them to irritate the intestinal mucous membrane. The rectal method, when it can be used, is the most pleasant to the patient, especially to the older who cannot take pills. The use of quinine by hypodermic injection is necessary in cases in which all the other methods fail or in which it is necessary to get quinine into the system as quickly as possible, as in the pernicious forms. The objection to its use in this way, that it produces abscesses, is sentimental. It is better for the patient to have an abscess than to lose his life; in addition, if proper antiseptic precautions are taken, the production of abscesses after the hypodermic use of quinine is by no means so certain as most authors assert. In malarial districts practitioners are in the habit of using quinine by the endermic method. Although I have never been able to break an attack in this way, I have been repeatedly assured by practitioners that this can be done. The use of the remedy as a tonic according to this method is certainly indicated.

No substitute has been found for quinine; but the remedy next in importance is arsenic. This is most applicable to the chronic forms, and is to be administered between various doses of quinine which are given to prevent relapses. It must also be given in full doses, although it is entirely unnecessary to produce its toxic effects. It can be given for months at a time, and ought to be given until we have reasonable assurance that the spleen has returned to its natural size.

In the remittent and continuous forms quinine does not have any other effect than it would have in any other fever,—*i.e.*, that of an antipyretic. The treatment of these forms is purely symptomatic. Those who have dealt most with them prefer to begin the treatment with a mercurial, following this up with quinine in small but frequently-repeated doses. I am not prepared to say that this or any other method will have any effect upon the duration of the disease. Especially in the continuous form, the patient should be kept in bed, put upon diet, and watched very closely until the physician is positive that he is not dealing with a mild type of typhoid.

For the treatment of the chronic forms and the cachexia, nothing succeeds so well as removal to a non-malarial region. This, naturally, is also true of the acute forms; but there it is rarely necessary, as attacks can be controlled with quinine. If the removal is only from one district of a city to another, provided there be no malaria in the quarter to which the patient is taken, the result will still be good.

Physicians should be very careful where they advise their patients to move to. In my own experience I have seen several instances in which the patients were brought home from places advertised as non-malarial with worse forms than they had been sent away with. Everything else being equal, mountain resorts are to be preferred to the sea-coast.

Besides quinine and arsenic, all the tonics have been used,—especially iron. In anæmic or hydræmic cases iron with quinine frequently produces very good results. For the affections of the nervous system accompanying malaria, strychnine in very small doses is of value.

The enlargement of the spleen will be found frequently to require special treatment. When this organ is very much enlarged, the iodides are sometimes of value. When the enlargement is only moderate, faradization has seemed to me frequently to give good results, although recent reports do not appear to warrant us in using the method, because of its being absolutely unserviceable.

In all the various neuralgic forms antipyrin frequently acts like a charm,—not curative, but palliative. The remedy can be given per os or by hypodermic injection. Acetanilide seems to have special control over the neuralgia of the fifth pair of nerves due to malaria.

It is not necessary to add that the treatment has to be frequently modified to suit the individual. There are some patients who cannot take quinine at all; in some it produces urticaria, in others it causes a series of nervous symptoms which, at times, are worse than the original malady. These patients must be treated with substitutes or removed to a non-malarial climate.

Among the laity there are many remedies which are used for chills and fever. Of these the only one which has given me any results—and I have been faithful in trying any that are within the boundary of common sense—is lemon-juice. This given in doses of two drachms to a half-ounce twice or three times daily has certainly, in some cases, produced good results. In others, on the other hand, the results have been negative. Further observations will be necessary to establish its comparative value.

YELLOW FEVER.

By JOHN GUITÉRAS, M.D.

Synonymes.—French, Fièvre jaune, Typhus amaril, Fièvre matelote; Spanish, Vómito negro, El vómito, Fiebre amarilla; Portuguese, Fiebre amarela; German, Gelbfieber; Italian, Febbre gialla.

The yellow fever of children is described in the tropics under the names of mauvaise fièvre, fièvre inflammatoire, fièvre d'acclimatement, fièvre grave des enfants, by the French; fiebres malas, vómito de los criollos, tifo, fiebre de borras, in Spanish; pernicious fever, ardent fever, etc., by the English. These names indicate that the disease in children is not recognized as yellow fever.

Definition.—Yellow fever is a contagious miasmatic disease, traceable to populous centres in several sections of the shores of the tropical Atlantic, whence it is transmitted by commercial and individual intercourse. The specific symptoms of the disease are (1) a fever of from two to seven days' duration, characterized by a sudden invasion, and a fastigium of from one to four days' duration, terminating by lysis, the latter being interrupted, in severe cases, by a secondary exacerbation; (2) a steady fall of the pulse, commencing during the fastigium and leading to a remarkable slowing of the heart-beat; (3) jaundice; (4) vomiting; (5) albuminuria; (6) a tendency to stasis of the circulation, and (7) to hemorrhages. The specific lesions are parenchymatous inflammations of the liver, kidneys, and stomach.

History.—This is the first time, I believe, that a chapter in a treatise on the diseases of children has been devoted to the yellow fever. And yet no disease is more deserving to be so classed.

The history of yellow fever in children reduces itself to occasional statements in the descriptions of epidemics occurring outside of the yellow-fever zone. Most of these observations are directed to show that there is a comparative exemption from the disease in early life. It is a curious fact that the existence of the disease in children of yellow-fever countries has been persistently denied by the native physicians. These views, which were founded on the idea that the disease was an acclimating fever, still dominate general opinion, especially in those countries. The only concession made in this direction has been to accept the occurrence of *exceptional* cases;

and pitched battles have been fought over these, as to whether they were or were not cases of yellow fever.

Blair,[1] however, in his report on the yellow fever of Guiana, recognized the susceptibility of children. Rufz de Lavison[2] suspected that the immunity of the creoles from yellow fever was the result of a previous attack in early life. Lota,[3] in a most interesting paper, upholds the same opinion, supporting it with clinical observations. Reyes,[4] of Cuba, and Bordas,[5] of Key West, reported several cases, but without making any generalizations. Chaillé,[6] of New Orleans, has given the most convincing arguments in this discussion in his analysis of the death-rate of that city.

My own tables of the death-rate of Key West will throw, I think, some light on this subject. They not only prove the susceptibility of white children in the tropics, but show also the errors of diagnosis that have led to the opposite opinion.

The facts that I have collected warrant the conclusion that the foci of endemicity of yellow fever are essentially maintained by the creole infant population,—the very population whose supposed immunity has been the subject of innumerable theories.

During the epidemic of 1887 in Key West I found that the disease spread very extensively among the children. I discovered, however, a remarkable immunity of the children seven and eight years old. These were the children aged respectively one and two years at the time of the last preceding epidemics of 1880 and 1881. The following table is constructed from data kindly furnished by the president of the local Board of Health, Dr. J. Y. Porter, U.S.A.:

TABLE I.—*Cases of yellow fever among the natives of Key West during the epidemic of* 1887, *classified by ages.*

Less than 1 year	2	10 years	7
1 year	4	11 years	1
2 years	10	12 years	4
3 years	13	13 years	0
4 years	13	14 years	1
5 years	15	15–20 years	2
6 years	3	20–25 years	1
7 years	0		
8 years	0	Total	82
9 years	6		

Many more cases undoubtedly occurred, but were not reported, because most of the Cuban physicians practising in the city did not believe that the native children could have yellow fever.

---

[1] Yellow Fever in British Guiana, London, 1852 and 1856.

[2] Chronologie des Maladies de la Ville de Saint-Pierre, 1869.

[3] Archives de Médecine navale, 1870, p. 315.

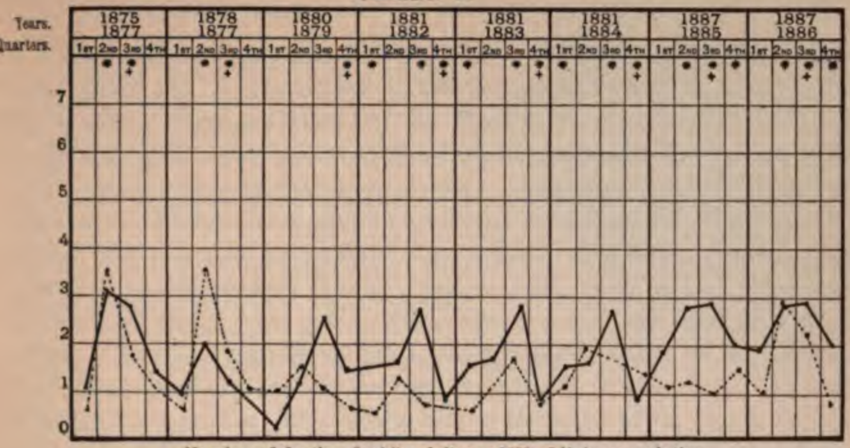
[4] Fiebre de Borras de los Niños, W. Reyes.

[5] Fiebre amarilla de los Niños, Crónica Med.-Quir. de la Habana, 1881.

[6] Annual Report of the National Board of Health, 1880.

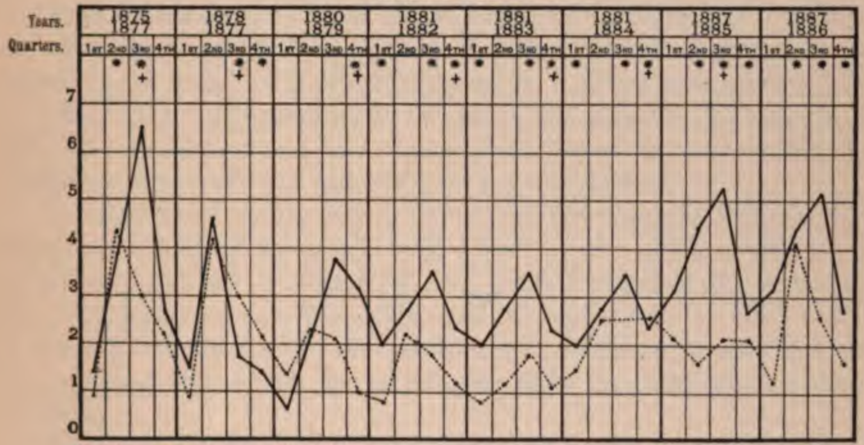
YELLOW-FEVER YEARS ——— NO-FEVER YEARS ....

CHART I.



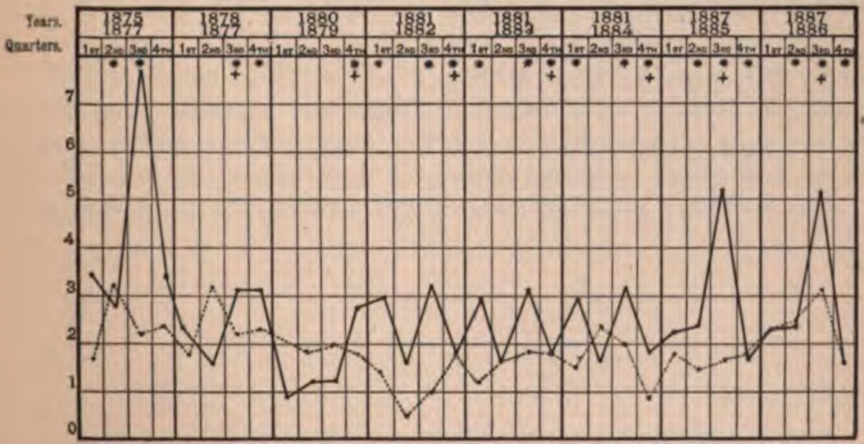
Number of deaths of *white adults* per 1000 of living population.

CHART II.



Number of deaths of *white* children, *five years of age and under*, per 1000 of living population.

CHART III.



Number of deaths of *white* children, *one year of age and under*, per 1000 of living population.

John Guitéras, M.D.

I tabulated the death-records I found in Key West, covering the period from 1875 to 1887. The details of this investigation will be found in the Report of the Surgeon-General of the Marine Hospital Service for 1888. The following charts—from I. to VI.—show graphically the results. The vertical spaces represent two series of years, contrasting a yellow-fever year with one of absolute or perhaps only relative immunity. The years are divided into quarters, as follows: first quarter, December, January, and February; second quarter, March, April, and May, and so on. The quarters in which deaths from yellow fever were reported are marked with an asterisk. The one quarter in which the greatest number of deaths from that disease were reported is further indicated by a cross. In the horizontal lines are marked the death-rates per thousand of the living population for the different quarters. The rates are represented by the continued line in yellow-fever years, by the dotted line in no-fever years.

These charts leave no doubt of the increase of the death-rate of children in yellow-fever years. The exception of the year 1878 is explained by the prevalence of an epidemic of whooping-cough. This disease appeared also in 1884. The correspondence of the rise of the infantile death-rate with the yellow-fever quarters, and the accentuation of the rise during the greater epidemics of 1875 and 1887, are remarkable facts.

In order more conclusively to prove that this excess of infantile mortality was caused by the yellow fever, I have contrasted the death-records of whites and blacks. Charts IV., V., and VI. represent the curves for the colored population. The mildness of yellow fever in the black race is universally admitted. We are not surprised, therefore, to find that the prevalence of yellow fever has no influence upon the death-rate of colored children.

Inasmuch as this increase of the number of deaths of white children is not accounted for in the mortuary records under the diagnosis of yellow fever, it becomes of great importance to discover under what heads they are to be found.

This is shown in Table II. I selected certain diseases with which yellow fever might be confounded, and these were classified in several groups,—Group A, Group B, etc. The figures represent the death-rate for the quarter of the year in which the greatest number of deaths from yellow fever were reported, contrasted with the same quarter of a no-fever year.

TABLE II.

Number of deaths in the city of Key West per 1000 of living population from certain specified causes that might be confounded with yellow fever. The rate given is that of the quarter of the year having the greatest mortality from yellow fever, contrasted with the same quarter in a no-fever year.

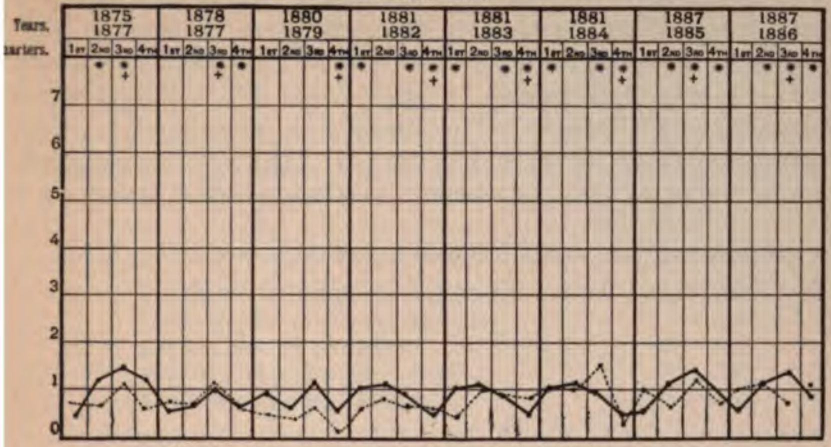
Yellow-fever years.	A. Meningitis, cerebritis, cerebro-spinal fever, etc., in children.		No-fever years.	Yellow-fever years.	B. Tuber-cular menin-gitis.		No-fever years.	Yellow-fever years.	C. Malarial fevers of natives, mostly children.		No-fever years.	Yellow-fever years.	D. Other fevers, excluding yellow fever.		No-fever years.	Yellow-fever years.	E. Convulsions, dentition, worms.		No-fever years.	Yellow-fever years.	F. Diarrhœal diseases, acute.		No-fever years.
1875	0.28	0.37	1877	1875	0	0	1877	1875	1.40	0.12	1877	1875	1.56	0.37	1877	1875	0.28	0.12	1877	1875	2.12	1.11	1877
1878	0.46	0.37	1877	1878	0	0	1877	1878	0.34	0.12	1877	1878	0.34	0.37	1877	1878	0.57	0.12	1877	1878	0.46	1.11	1877
1880	0.40	0	1879	1880	0	0	1879	1880	1.31	0	1879	1880	0.10	0	1879	1880	0.50	0.21	1879	1880	0	0.10	1879
1881	0.37	0.26	1882	1881	0.09	0	1882	1881	0.18	0	1882	1881	0.09	0	1882	1881	0.18	0.08	1882	1881	0.47	0.08	1882
1881	0.37	0.16	1883	1881	0.09	0	1883	1881	0.18	0.08	1883	1881	0.09	0.08	1883	1881	0.18	0.41	1883	1881	0.47	0.24	1883
1881	0.37	0.15	1884	1881	0.09	0	1884	1881	0.18	0.07	1884	1881	0.09	0	1884	1881	0.18	0.84	1884	1881	0.47	0.38	1884
1887	0.56	0.21	1885	1887	0.25	0	1885	1887	1.25	0.35	1885	1887	0.12	0.21	1885	1887	0.37	0.14	1885	1887	0.50	0.57	1885
1887	0.56	0.40	1886	1887	0.25	0	1886	1887	1.25	0.13	1886	1887	0.12	0.40	1886	1887	0.37	0.26	1886	1887	0.50	0.26	1886
Total	3.37	1.92			0.77	0			6.09	0.87			2.51	1.43			2.68	2.18			4.99	3.85	

The average annual death-rate corresponding to these figures is:

	A.		B.		C.		D.		E.		F.	
	1.68	0.96	0.38	0	3.04	0.43	1.25	0.71	1.81	1.09	2.49	1.92

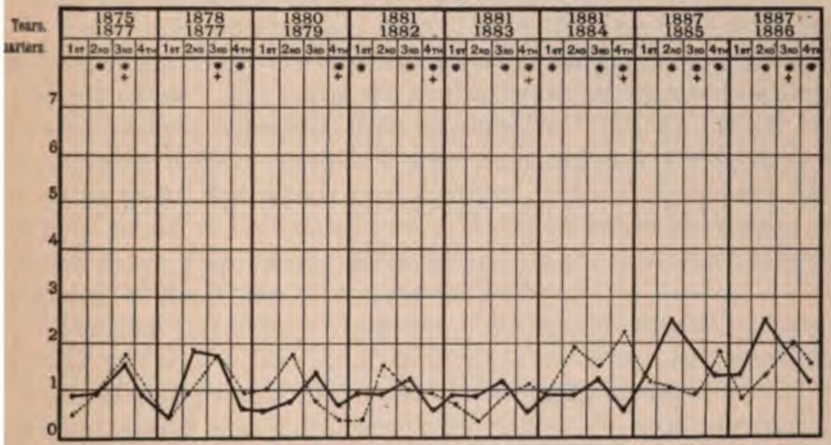
YELLOW-FEVER YEARS ———— NO-FEVER YEARS ··········

CHART IV.



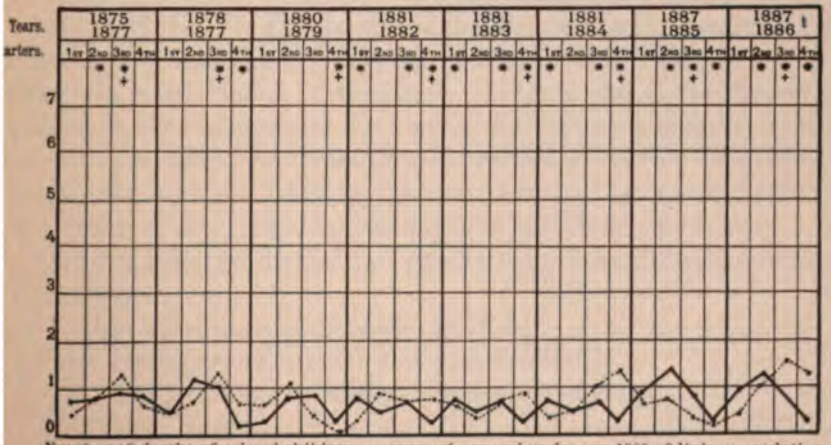
Number of deaths of *colored* adults per 1000 of living population.

CHART V.



Number of deaths of *colored* children, *five years of age and under*, per 1000 of living population.

CHART VI.



Number of deaths of *colored* children, *one year of age and under*, per 1000 of living population.

John Guitéras, M.D.

A glance at this table shows that the excess of deaths is to be found under the head of acute cerebral and meningeal affections and the malarial fevers. This result I had anticipated in consideration of the astonishing frequency with which the diagnosis of meningitis or pernicious fever is made for the diseases of childhood in the tropics. The death-rate from meningitis (so reported) in the city of Havana is extraordinarily high, as shown by the following terms of comparison:

Annual Death-Rate per 1000 of Living Population from Meningitis, etc.

For the United States, census of 1880	0.20
Louisville, Ky. (highest in the Union)	0.50
Havana, mean of seven years	1.27
Key West in yellow-fever years	1.40
Key West in no-fever years	1.27

In regard to the malarial fevers, I may say that I have elsewhere[1] furnished proof that Key West is not a malarious locality.

We have reason, then, to believe that yellow fever in its native haunts is essentially a disease of childhood, the adult natives being protected by a previous attack. The prevalence of the disease among adults—foreigners—is an accident consequent upon immigration. The same would happen with measles in Northern cities if these were subject to immigrations from countries where that disease was unknown. And if this foreign element were to belong to the colored race, it is very probable that measles would assume a very grave form, and would throw in the shade the milder manifestations of the disease in native white children.

Etiology.—The early historians of the Spanish invasion of America furnish grounds for the belief that yellow fever was endemic with the American aborigines of the littoral of the tropical Atlantic, and that the disease appeared with them sporadically, and in the shape of periodic epidemics, in the same manner that cholera exists in portions of Asia. Certain it is that yellow fever manifests itself in this shape among the present indigenes of the yellow-fever zone. But the increase of commercial intercourse and travel has established two modalities of the disease: on the one hand, the yearly influx of foreigners has given rise to the formation of centres, in the yellow-fever zone, where the disease is annually epidemic; on the other hand, the communications with the exterior have caused the transportation of the infecting agent to more or less distant lands, where the vitality of the germ does not appear to hold from year to year.

I have therefore divided the regions affected by the yellow fever into three classes:

I. The regions of annual epidemics (focal).
II. The regions of periodic epidemics (perifocal).
III. The regions of accidental epidemics (occasional).

---

[1] Annual Report of the Marine Hospital Service for 1888.

The focal zone (I.) includes the ports of Havana, Vera Cruz, Matanzas, Rio Janeiro, and other important seaports of intertropical America, perhaps also a portion of the African coast on the Atlantic. In these ports the disease, never entirely absent, regularly spreads every year with the advent of warm weather, and invades the inhabitants who are not protected by a previous attack, with few exceptions. In Havana, for instance, we find that the lowest number of deaths from yellow fever in one out of seven consecutive years is five hundred and fifteen, and the highest sixteen hundred and nineteen, in a population of about two hundred thousand. And this does not include the deaths of native children.

In the perifocal zône (II.), or regions of periodic epidemics, belong the majority of the ports of the tropical Atlantic in America and Africa, and a smaller number on the Pacific coast of the American continent. New Orleans, Charleston, and probably other American cities belonged once in this category.

The zone of accidental epidemics (III.) includes those places of the temperate zone or of high altitude where the disease is occasionally imported and will prevail for one season only. It would be rash to limit absolutely the boundaries of this area; that is, the limits within which, once imported, the yellow fever may assume an epidemic form. No place can claim an absolute immunity, in the countries bordering on the Atlantic Ocean between the parallels of 45° north latitude and 35° south latitude, and below an altitude of fifteen thousand feet above sea-level. It is true, however, that as we approach the limits above specified the probabilities of infection become almost *nil*.

As examples of this third group we may mention the epidemics of New York, Philadelphia, the Mississippi Valley, Florida, Spain, Montevideo, Cuzco, etc. The increasing facilities of railroad communications in southern countries are fast divesting the watercourses of their importance as necessary means of transportation of the yellow fever.

In the regions of Classes I. and II. we say that the disease is endemic because it occurs in succeeding or periodic seasons without the necessity of importation. The theory of spontaneous origin of infectious diseases has not received any support in bacteriological researches. We say, therefore, that in these localities the germs of the disease are not destroyed during the healthy season. This endemicity is by no means permanent, as is shown by the fact that New Orleans and Charleston have been transferred from Class II. to Class III. It is very probable that by efficient sanitation the centres of Classes I. and III. could be eliminated,—those of Class I. by being transferred to Class II., and those of Class III. by securing an absolute exemption.

The centres of Class II., which represent the natural modality of the disease, deserve a more extended consideration. We remark here the periods of more or less prolonged exemption. This absence of the disease, in my opinion, is not real. The belief in it is founded on the opinion that the disease does not occur among native children. But the facts advanced in the historical part of this article, proving the susceptibility of all children, warrant the opinion that in these localities the foci of endemicity are maintained by the native children. This is an important factor in the epidemiology of yellow fever. We must remember that these periods of latency follow, as a rule, after extensive epidemics, during which the great majority of children have had the disease and have acquired immunity. From experience we know that after these epidemics the number of houses or rooms where exceptional conditions, that we know very little about, will keep up the vitality of the germs are very few, and few also will be, in the following year, the number of unprotected children. In the fewer instances still in which these two conditions are brought into mutual operation, a case of yellow fever will present itself. These cases are not recognized, because the foreigner is looked upon as the only touchstone for the detection of the disease. Now, in yellow-fever countries the foreigner is not a frequenter of the homes of the creoles. Thus the disease, like cholera in some of the cities of India, may be confined to the natives. Only occasionally we hear, during these periods of supposed exemption, of a sporadic case of yellow fever in a foreigner. It can be readily seen how, in this manner, epidemics have occurred where it was impossible to trace the origin to importation, thus giving rise to the opinion that the germs of the disease developed spontaneously from local causes.

In some epidemics we may be able to trace the outbreak to both sources: we will have, after some years of apparent exemption, the disease assuming an epidemic form among the native children who have grown up without being protected by a previous attack; and at the same time the disease may break out around some focus traceable to importation. This latter eruption generally occurs in the business quarters of the city, and among strangers. It constitutes, as a rule, the first accepted evidence of the presence of the disease. These facts account for the statement frequently made, to the effect that epidemics of yellow fever are preceded or announced by the prevalence of bilious fevers, etc., especially among children.

This double origin of an epidemic I discovered in Key West in 1887. Charts II. and III. of the present article show an increased mortality of white children for the first quarter of the year, before the first recognized cases of yellow fever occurred in the business quarters of the city around a focus evidently of imported infection. Even the preceding summer I notice an increase of the infantile mortality.

This tendency to spread of the domestic infection, together with the facility with which the imported infection takes hold during the same season, may be simply due to the greater vulnerability of the population resulting from the increase by immigration of unprotected strangers, and by birth of natives. The fact, however, that this susceptibility seems to affect large extents of territory points towards the existence of some atmospheric, telluric, or other conditions, entirely unknown, that prevail in certain years and favor the spread of the disease. In this sense we may speak of an epidemic wave or territorial receptivity.

In this connection it is that the subjects of heat, moisture, atmospheric pressure, electricity, prevalence of certain winds, etc., deserve consideration. But we must confess that this study has been fruitless. In regard to the prevalence of certain winds I have to call attention to the fact that the houses in the tropics are constructed so as to favor as much as possible a complete exposure to the prevailing breezes. A season of persistent winds from the opposite quarters reduces ventilation to a minimum, as all will acknowledge who have felt the oppressive influence of a southwesterly wind in the city of Havana, where every house seems to breathe from the eastward. The prevalence of southwesterly winds has been considered in the Antilles as an element of great importance in the causation of yellow fever. It is very probable that their action is merely such as I have indicated above.

I have used the term contagious-miasmatic in the definition of yellow fever, because it is the one generally employed for the group of infectious diseases in which yellow fever belongs. But there is not, as Prof. William H. Welch very well observes, any essential difference between these and the diseases termed contagious. It is merely a question of degree or facility for transmission. All these diseases require a medium, a vehicle of transportation from the sick to the well. Those diseases in which this medium acquires great importance, to the extent that media apparently the same prove sometimes destructive, sometimes favorable, to the vitality of the infecting agent, we term contagious-miasmatic. They might with propriety be called indirectly or mediately transmissible. Such a disease is yellow fever. So great, in fact, is the importance of the locality and surroundings in this disease that the term infectious, as formerly understood, was properly applied to it. When we are confronted, however, with a doctrine of pure miasmatic origin, we have to place yellow fever with the contagious diseases. For surely yellow fever belongs with those diseases in which the methods of modern research have discovered the presence of a micro-organism in the body, capable, if favorable means are provided, of conveying the disease to other individuals. The general characteristics possessed by yellow fever in common with those diseases are: first, yellow fever is a disease of populous centres; secondly, it is a portable disease and extends along the lines of travel; thirdly, there is no evidence of the disease ever having developed where there was no possibility of infection from other cases; fourthly, one attack confers immunity against a second invasion.

I need not mention cases illustrating the transmission of the disease by fomites. This is admitted, though rather reluctantly, even by the believers in the local origin of the disease. I shall present examples only of the transportation of the disease by sick persons, and these will be taken from observations of the Florida epidemic of 1888.

In the first place, we must bring as evidence the general course of the epidemic. It was confined to Key West during the spring and summer of 1887. Finally, towards the end of summer it made a landing on the mainland, at Tampa, the only port of Florida that was in constant communication with Key West, thus leaving untouched many places that were nearer to the infected area. Some of these became subsequently infected through communication with Tampa. In fact, soon after the outbreak at Tampa and the stampede that followed it we find cases reported and local outbreaks in the track of refugees. Now, the end of summer and beginning of autumn is not the season for the commencement of epidemics in those localities where the disease does not require importation for its development. Here the disease gradually rises with the advent of warm weather.

The outbreaks at Sanford and Bartow occurred around refugees who took sick soon after their arrival from the seat of the epidemic. So constantly has this been my experience that when this link is missing the explanation is often to be found in the fact that the original case was a convalescent, or else the disease was so mild as not to be recognized. It is very probable that children and negroes import the disease in this manner unsuspected.

Another case of importation by individuals is that of the village of Callahan. This town is situated at the intersection of railroads from Fernandina and Jacksonville. Callahan is much nearer to Jacksonville than is Fernandina, but had established a vigorous quarantine against the former place when the fever was declared epidemic in July of 1888. On the occasion of a strike in Fernandina, this city being erroneously reported healthy, the town of Callahan contributed a contingent of guards for the occasion. These guards went and returned without any baggage whatever. On their return they scattered at once to their homes in the country around Callahan. Only one of these guards, Upchurch, remained in the town. He took sick the day after his arrival at his home, one of the oldest houses in the village. Five cases are traceable to this one,—a brother who occupied an adjoining room, three white employees of the railway-station, and a negro who nursed the Upchurch brothers. The station is separated by a narrow road from the house. The employees visited the first Upchurch brother frequently, and used the privy of the house. All this happened between the 9th and 29th of September. One of these employees was treated in the hotel. The two subsequent cases among the whites are traceable to the hotel,—the son of the proprietor on the 13th of October, and a Mr. Rawson, who, after having refugeed, returned to the hotel on the 23d of October and took sick on the 27th. The white population fled from the city, and they appear to have carried no infection with them. Two cases were discovered later in the colored population. Long before this outbreak at Callahan, the town of McClenny, lying farther on the railroad from Jacksonville and Fernandina, had been infected by a refugee, a printer from Jacksonville.

I attach much importance to the following incident. Here the events were predicted and carefully watched for. Mr. Upton owns a saw-mill at

Uptonville, Georgia, on the railroad between Jacksonville and Savannah, seven miles from the inter-state line. He arrived in Uptonville on the 22d of October, after driving over country roads forty miles from Baldwin. The previous history of Mr. Upton is very unsatisfactory. I have found that he was at Callahan on September 25, and visited the second Upchurch case. At any rate, when I saw him at Uptonville on the 24th of October, I decided, together with Dr. Posey, of New Orleans, that he was a convalescent from yellow fever, notwithstanding his protestations and denials. It was not possible to remove Mr. Upton from the village until the 26th. I suspected that other cases would arise from this one, because Mr. Upton's room was very filthy. Furthermore, it had been constantly crowded by villagers. I made a careful inspection of these on the 25th and 26th. On the latter date a Mr. Simmons was taken with yellow fever. He was a night-watchman, and had spent the greater portion of one night in the room with Mr. Upton. On the 27th I removed the whole of the Simmons family to the United States quarantine camp. Mr. Simmons was sent to the hospital, and Mrs. Simmons, with three children, to the camp, which was free from infection. On the 28th Mrs. Simmons was taken sick and died of yellow fever. No other cases occurred. This town had no communication with the infected district. The infecting agent in this case was brought by a convalescent. Yet this man had been probably sick at his own home in Baldwin without conveying the disease to any one. His house here is new, airy, and neatly kept.

The most interesting experience of all, however, is that of a train of refugees from Jacksonville to the mountains of North Carolina. There is evidence to prove that in less than thirty-six hours a source of infection was developed in this train by the refugees themselves. For we find that out of two hundred and eighteen, ten were attacked in the train or soon after arrival; whilst out of more than nine hundred unacclimated refugees leaving Jacksonville under more hygienic circumstances, by a short journey of one hour and a half to Camp Perry, only twenty-four were affected. The train to North Carolina was composed of cars brought into Jacksonville from the North an hour or two before departure; and no baggage was allowed on the train. The excess of cases, it appears, was due to auto-infection of the crowd shut up in the cars over thirty-six hours.

On the other hand, the cases are numerous that would seem to contradict the above experience,—Mr. Upton, for instance, who infected no one at Baldwin; more than one thousand refugees coming to Camp Perry without producing a secondary focus of infection; in the very hospital of the camp several unprotected persons were exposed without any one contracting the disease; and the same may be said of the larger hospital of the Sand Hills, near Jacksonville.

All this illustrates practically what I have said of the importance of the medium. In some localities there is an obstacle to the spread of the disease. But it is evident that, when these obstacles are absent, a case of yellow fever is capable of infecting certain localities, and these may be restricted to very small limits. Many times, in the presence of these apparently contradictory facts, I have looked for the points of difference between the infectable and non-infectable places. I can only say that in the latter there was abundance of fresh air, and an absence of old wood, of packed things, and of crowded people. This is unquestionably one of the most important and most obscure problems in the etiology of the disease.

The disease fails to spread for one of two reasons: either the locality resists infection, that is, is unsuitable for the life of the infecting agent outside of the human organism, or else the conditions of the place create a non-receptivity on the part of the individual. And how insignificant a cause may produce the latter effect! I had an attack of yellow fever, mild, unrecognized, thirty-four years ago, and a barrier was raised forever, somewhere in my organism, against all encroachments of the disease,—an event insignificant in the distance of time, and that has left no other appreciable trace upon my body. Is it not reasonable to suppose that modifications in the surroundings, the food, the air, may also produce similar though temporary modifications,—a slight change in the acidity or alkalinity of a secretion, the substitution in the orifices of the body of one form of micro-organism for another? For instance, I found that during my sojourn in Camp Perry the odor and taste of putrefaction disappeared almost completely in a cavity in one of my teeth. We must not lose sight of apparently unimportant factors in this complicated problem, where even chance must play an important part.

This temporary individual resistance received some support in the fact that a considerable number of persons escape during an epidemic who come into town only during business hours. This measure, however, is not as successful as is generally believed.

Sex.—It appears that in infancy the male sex shows a slight predisposition to the disease. I noted the sex in fifty-six children in Key West, and thirty-four were males.

The previous condition of health, and the social position, have no influence as predisposing causes of the disease.

Period of Incubation.—The period of incubation, as far as it can be determined in the absence of inoculation experiments, may vary from a few hours to fourteen days. The cases in which it extends beyond the seventh day are exceptional. Out of twenty-seven persons who had the disease after removal from an epidemic centre, seven had the disease on the first day, one on the second day, five on the third, three on the fourth, five on the fifth, one on the sixth, two on the seventh, one on the ninth, one on the eleventh, and one on the thirteenth day.

In the last two cases there is a possibility of infection after removal from the infected centre. Of nine hundred unacclimated individuals who left Jacksonville, going into healthy districts, after serving ten days of probation at Camp Perry, none developed the disease.

Morbid Anatomy.—The cadaver presents always a yellow discoloration. The body is frequently mottled, especially in the dependent portions. The features may be somewhat bloated, and there is often a dark discharge from the nose and mouth. I have noticed often in children a purplish discoloration about the pubis, which may be limited to the scrotum or labia. These latter structures may show some evidence of infiltration and excoriation, amounting in very rare cases to actual sloughing. Putrefactive changes and rigor mortis set in early.

I have never dissected the body of a child dead from yellow fever. As the symptoms in the grave cases are precisely the same as those met in the adult, there is no reason to suppose that any difference will be found post mortem.

The muscles present a dark color in contrast with the yellow-stained adipose tissue. The contents of the thoracic cavity are generally normal. The lungs present in rare cases small hemorrhagic foci, and seldom any marked tendency to hypostatic congestion. The left heart I have always found firmly contracted. The muscular wall presents a normal appearance. The cavities contain always some dark fluid blood, very rarely some small coagula. Neither the endocardium nor the pericardium shows evidences of inflammation. Under the microscope I found the muscular fibres generally healthy. A few are found with some granulations about the nucleus.

In the abdomen we notice at once the peculiar color of the liver. This I have never found absent in an uncomplicated case of yellow fever. The organ is not enlarged. It is of a light color in which yellow predominates decidedly. The comparison with boxwood is a good one. In cases of rapidly-fatal termination the discoloration may appear only in patches. The consistence of the organ is not impaired. On section we find the blood-vessels comparatively empty. The microscopic evidences of fatty degeneration are present. Under the microscope we find evidences of some acute interstitial inflammation. The interlobular connective tissue is often swollen and contains in places accumulations of embryonal connective-tissue cells. The liver-cells are either cloudy or decidedly fatty. Some of them are pigmented more than the normal.

The stomach is inflamed. The blood-vessels of the mucous membrane are engorged with blood. In many places near the surface I found minute extravasations of blood. The abundance of leucocytes or embryonal connective tissue around the gastric tubules is evidence of inflammation. The stomach very frequently contains the dark fluid called black vomit, even when it has not been ejected during life.

The kidneys are large, soft, and congested. The microscope reveals the existence of a diffuse nephritis. The form of nephritis present in these cases needs further investigation. It must differ in important particulars from the disturbances of the kidney that occur in other infectious diseases. It is characterized by a prompt invasion and an equally rapid subsidence, and leaves no permanent traces. It does not produce any dropsy.

TEMPERATURE-CHARTS OF YELLOW FEVER.

FIG. 1.

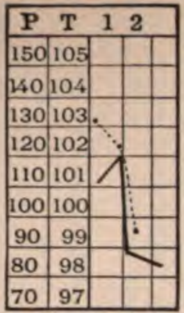


FIG. 1.—Two days' duration. Recovery. Seven years old.

FIG. 2.

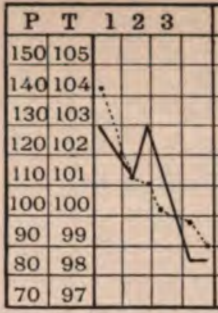


FIG. 3.

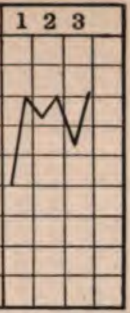


FIG. 2.—Three days' duration. Recovery. Eight years old.
FIG. 3.—Three days' duration. Death. Four years old.

FIG. 4.

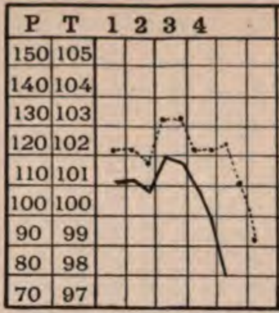


FIG. 5.

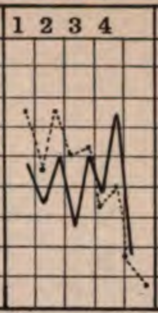


FIG. 6.

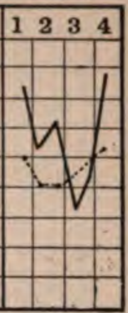


FIG. 4.—Four days' duration. Recovery. Eight years old.
FIG. 5.—Four days' duration. Recovery. Three years old.
FIG. 6.—Four days' duration. Death. Three years old.

FIG. 7.

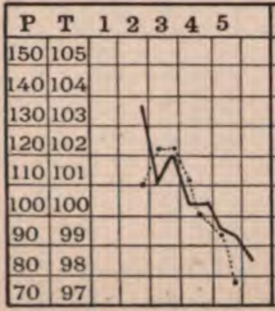


FIG. 8.

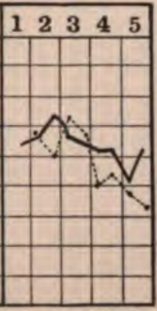


FIG. 7.—Five days' duration. Recovery. Twelve years old.

FIG. 8.—Five days' duration. Death. Four years old.

FIG. 9.

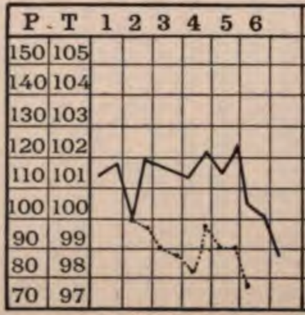


FIG. 10.

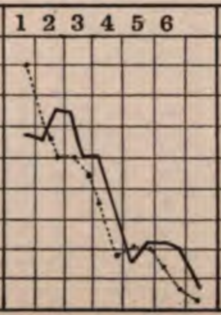


FIG. 11.

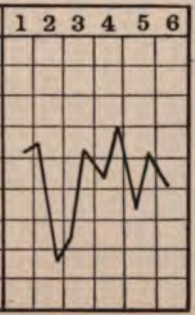


FIG. 9.—Six days' duration. Recovery. Nine years old.
FIG. 10.—Six days' duration. Recovery. Thirteen years old.
FIG. 11.—Six days' duration. Death. Two years old.

FIG. 12.

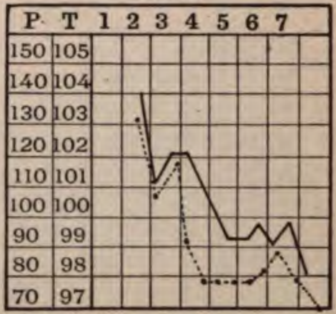


FIG. 12.—Seven days' duration. Recovery. Nine years old.

John Guitéras, M.D.

The spleen is not altered in yellow fever. I have been inclined, with others, to believe that the semilunar ganglia of the solar plexus were often inflamed in yellow fever. But my observations were not sufficiently numerous, and we have the high authority of Woodward against this view.[1]

No histological changes have been found in the blood by Surgeon G. M. Sternberg, U.S.A.[2] The subsequent studies of this investigator have been devoted to the discovery of the specific micro-organism of the disease. He has, I believe, successfully refuted the claims of Freire, Carmona, and Finlay to the discovery of such micro-organisms. In brief, he has discovered no microbe peculiar to the disease in the blood and tissues. The same results were obtained by Dr. Tamayo and his colleagues of the Bacteriological Institute of Havana. In a personal communication Dr. Sternberg informs me that he has lately directed his labors to the examination of the organisms found in the gastro-intestinal contents. The same line of investigation has been followed by the French pathologist Dr. Gibier.

Symptomatology.—*Fever and Pulse.*—The absence of previous records of the disease in childhood justifies the publication of several temperature-charts. I have selected illustrations showing different periods of duration of the disease, and contrasting the favorable with the fatal cases.[3]

Yellow fever presents a more characteristic temperature and pulse-curve than any other of the acute infectious diseases. The distinctive features are observed in making a simultaneous survey of the temperature- and pulse-lines, and they are as distinct in the child as in the adult.

During the initial chilliness and first rise of the temperature, the pulse will rise, as it does in all acute febrile diseases, in proportion with the increase of bodily heat. The invasion takes place generally during the night, and on the morning of the first day the pulse will be found, in children, above 120, and frequently as high as 150. The temperature in favorable cases does not often exceed 103° F. The temperature will generally show an evening exacerbation on the first day, but even at this early date the pulse-curve may start on the line of descent that is so characteristic of the disease. This will certainly happen on the second or third day of the disease, even though the fever may still show a persistent fastigium. Dr. J. C. Faget, of New Orleans, was the first to discern the importance of this phenomenon as a diagnostic sign.

Of course the pulse in children is more subject to accidental variations produced by emotion or exertion, but it is still governed by the same law. The very slow pulse, however, that characterizes the end of the lysis and the convalescence of adults is not generally marked in very young children.

---

[1] Reports of John Guitéras and J. J. Woodward in connection with reports of the Havana Yellow Fever Commission, Supplements Nos. I. and IV. of the Bulletin of the National Board of Health.

[2] Supplement No. I. of the Bulletin of the National Board of Health.

[3] Many valuable clinical notes, and opportunities to study the disease in children, I owe to the kindness of my friends Drs. Bordas, Porter, Monteresi, and Armona, of Key West, Dr. Faget, of New Orleans, and Dr. Caldwell, of Sanford, Florida. See Bordas, "Fiebre amarilla de los Niños," Crónica Médico-Quirúrgica de la Habana, 1887.

The gradual fall of the temperature is often broken by one or more evening exacerbations, which in severe cases constitute a secondary fever. This may extend over several days, giving rise to prolonged cases of ten and fifteen days' duration. In these cases we frequently observe well-marked diurnal remissions independent of all malarial complications. In fact, I find that writers have insisted too much on the continued type of this fever. This was occasioned by the discussions as to whether yellow fever was or was not a malarial disease. As compared with these, yellow fever is certainly a continued fever; but this feature is not specific of the disease. The same may be said of the statement, to which much prominence has been given, that yellow fever is a fever of one paroxysm. As a matter of fact, there is quite often a well-marked remission of the fever between the primary and secondary exacerbations. In fatal cases there is often a marked accentuation of this secondary fever. In fact, an early break in the lysis by a sharp rise of the temperature above that of the fever of invasion is of very bad omen. In mild cases the secondary fever is absent or only slightly indicated.

The Facies.—Jaundice.—The Skin.—The appearance of the face is often sufficiently characteristic on the first day of the disease to warrant a positive diagnosis. Except the jaundice, the appearance is that of measles before the eruption,—the same lively coloration of the skin, the slight puffiness of the lids with red borders, the hyperæmia of the conjunctivæ, the same lustre and watery condition of the eye,—the ferrety eye of typhus, in fact, but always, from the very commencement, with a slight shade of yellow. In the earlier stages of the disease, when the injection and the jaundice, especially the latter, are not well marked, it is an important fact that these phenomena are better observed at a slight distance than on close inspection. Very frequently, and especially in children, on approaching the bedside I have noticed the turgidity of the lips, the suffusion of the eye, and the faint icteric hue that are unmistakable; and yet, on separating the lids, on close inspection absolutely nothing abnormal can be detected.

In the mild cases of children the above may be the only evidence of jaundice. In severe cases, about the third or fourth day the skin assumes a lemon-yellow color. Even before this we may notice from time to time a transient shade of yellow to pass over the red mask of the face, when the movements of expression chase for an instant the blood from the distended vessels. General jaundice is neither as frequent nor as marked in the child as in the adult.

We notice also in children that a very intense injection of the conjunctiva, with ecchymotic spots there and on the surface generally, are rare manifestations. We find very frequently an erythematous condition of the scrotum and labia (as described by French observers), which may lead to infiltration and excoriations. Very rarely we may meet with sloughing of these parts or other regions of the integument. I have noticed in some cases herpes labialis, in others boils, and occasionally a lichenous eruption; but all these are exceptions.

The skin presents alternations of dryness and moisture. It is easy to produce perspiration, but this secretion is not critical. The sweat is always acid.

A peculiar odor sometimes emanates from the bodies of yellow-fever patients. I have never noticed it outside of wards where the patients were treated by inducing profuse perspiration, and I could never detect it in children.

The tongue is generally moist and presents a whitish coating. It is more apt to be pointed than flat. The gums are spongy in some cases, and they bleed easily. A dry tongue with sordes is exceptional with children. It may be encountered in prolonged cases of an adynamic type. The thirst is intense,—more so than in the adult.

Vomiting.—This is not an initial symptom, nor is it as striking a feature as in the adult, because of the comparative absence of painful retching. It is frequently limited to the occasional and even exceptional rejection of food. The matters vomited are generally light-colored, of a very faint greenish-gray shade, and watery. Their reaction is acid. In severe cases the gray color becomes darker, and black striæ appear in the fluid. In these cases the vomiting becomes more frequent and is followed by exhaustion. This earlier stage of the black vomit is quite common in children as compared with adults, and not rarely darker shades occur without fatal results. The true black vomit is not so frequent as in the adult, and, as in the case of the latter, though possibly to a less extent, it is a forerunner of death. This fluid is uniformly black and somewhat syrupy in consistence. These varieties are evidently different degrees depending on the amount of blood exuded into the stomach. They all show, under the microscope, red blood-corpuscles and granular pigment, besides other usual contents of the stomach. I have also found plates of cholesterine. It is very probable that both the biliary and blood pigments contribute to the formation of the dark coloring-matter. I have been shown by Dr. Gibier some cultures of a chromogenic micro-organism found by him in the fluids of the stomach. The culture gave a dark-brown color. It is possible that this discovery may change the views at present entertained as to the color of these fluids.

Vomited matters containing unaltered biliary pigment may resemble, it is said, the black vomit of yellow fever. A piece of white linen immersed in the former is stained of a dark-yellowish color, while in the latter case the stain is brownish black.

The bowels are generally costive, though easily acted upon by cathartics. Exceptionally the dejecta are dark and tarry. Diarrhœa and dysentery are encountered in some cases.

The Urine.—Together with the course of the pulse and temperature, and the facies, the examination of the urine constitutes the basis of the diagnosis of yellow fever. We turn to this more frequently than to any other one symptom as a crucial test in cases of doubt. The evidence we obtain from the urine consists in the presence of albuminuria. This is more common than is generally believed. If persistently looked for, it will be found in almost all cases, whether mild or severe. Albumen may appear in the urine on the first day of the disease, more frequently on the second, and almost certainly on the third day. I have found, especially in children, that albumen may be present only in the urine passed during the evenings, and, in some mild cases, during one evening alone throughout the attack. This explains why the symptom is often found wanting. In fact, the transitory character is a remarkable feature of the albuminuria and the nephritis of this disease. It is an early manifestation, differing from the albuminuria that is encountered in profound adynamic states. This nephritis disappears rapidly, without ever leaving, so far as I know, any permanent traces. It never causes dropsy nor inflammation of the serous membranes. Within a few days we may have albuminuria that may amount to complete coagulation of the fluid on boiling, blood in the urine, and an extraordinary number of epithelial, granular, and blood casts, together with more or less prolonged suppression of the secretion; and all these signs will disappear as rapidly as they came. The albumen may be present on one night only, and casts but two or three days. The amount of albumen varies from a trace to the largest amounts.

I attach much importance to the method of examination of the urine. In the press of work that attends an epidemic, many of the tests for albumen cannot be made with sufficient accuracy. Many times I have found urine that was pronounced non-albuminous after testing by heat and nitric acid, to show a distinct trace by the zone test. Frequently when the amount of albumen is small the cloud is very faint and broad, extending some distance above the point of contact with the acid, and may be taken for a general turbidity of the fluid if a sufficient quantity is not added. The best place to hold the tube is near a window-frame or the free edge of a door, having on one side the shadow of the room, behind the dark profile, and on the other side a free illumination. The urine should be allowed to run from a small filter, holding the tube at an angle, down to the acid. A small piece of filtering-paper can be folded in four and a filter improvised in a moment: so that there is truly no simpler or readier method than this.

The urine is always distinctly acid. Its quantity is almost always diminished, and the solids are increased in proportion to the bulk. At first the urine is clear and of normal color, but it may subsequently become turbid from the presence of blood, of urates, and of mucus and epithelial débris. At this time it assumes also a darker color, from the presence of an excess of coloring-matter, which is often biliary. The diminution of the quantity of urine and infrequency of micturition are very common in children, but the more advanced and serious degrees of suppression are not so frequent as in the adult. If sufficiently prolonged, the suppression leads to uræmic poisoning and a fatal termination.

Hemorrhages.—The gastric hemorrhages have been already mentioned. It only remains to say that in exceptional cases they may occur from any of the mucous membranes or surfaces of the body.

I find the following cases of children recorded in my notes:

	CASES.	DIED.
Hemorrhage from the stomach (varieties of black vomit)	28	9
" from the nose	4	1
" from the mouth	2	1
" from the bowels	1	0
" into subcutaneous tissue—extensive	2	2
Anticipated menstruation	3	0

Nervous Symptoms.—Under this head we find the greatest difference between the symptoms of childhood and those of adult life. Strange to say, we find that the nervous symptoms are perhaps more prominent in the adult than in the child; and this is not dependent solely upon the milder character of the disease in the latter. The difference lies partly in the absence of the moral or emotional element in children. The loquacity, the short-cut phrases and precipitate speech, the excitement, the show of indifference, with unmistakable evidences of fear, all these, that are such prominent features of the disease in the adult, are absent in the young. The wild delirium is seldom present. The child is generally listless and drowsy. The rachialgia and pains in the limbs are mostly wanting, but the headache is present without a doubt, as we may discover by inquiry, or by noting the corrugated brow, the expression of suffering, the evident desire to be let alone, and the turning of the face away from the light. The sleep is generally restless, and the child will wake up delirious or calling for water. His movements then will be somewhat ataxic or jerky, as if premonitory of a convulsive seizure. Convulsions, however, are not so common in the initial stage of yellow fever as they are in the eruptive fevers; nor are they more common than in the adult as later evidences of uræmic toxæmia. As in other fevers of infancy, we must note here the general absence of chills.

Very frequently in grave cases the child becomes semi-unconscious, and lies with the eyes closed, and constantly turning the head from side to side. The respiration then becomes more rapid and sighing at times, the pulse increases in frequency, the temperature may or may not take an upward turn, and the drowsiness deepens into coma. This is the usual termination of fatal cases. Their duration does not exceed three or four days. A few of these cases will die on the fourth or fifth day, after ejecting a large quantity of black vomit.

Other cases run a much longer course, and present all the symptoms of the typhoid state, with a jaundiced and clammy skin, defective renal secretion, albuminuria, and a pulse that is comparatively slow at times. Many of these cases recover. It is in this class of cases that therapeutics plays its most important part.

The cases of infantile eclampsia, with high temperatures, occurring during epidemics of yellow fever are probably the result of the specific infection. Certainly cases of this class, often terminating fatally, are comparatively common in yellow-fever countries. I have noticed in these cases a yellow discoloration of the skin after death. This is, however, merely a suggestion, requiring further investigation.

Complications.—These are rare in the course of the disease in children. Malaria has been discussed as a frequent complication of yellow fever, but without sufficient foundation. I have certainly seen the symptoms that are pointed out as indicative of this complication occurring in localities where there was no possibility of malarial infection. Such symptoms are not rare in the colored people. As I have shown in another part of this paper, a great deal of the malaria of childhood that is supposed to exist in yellow-fever centres is really yellow fever. Indeed, much of the reputation of these cities for malaria depends upon the false interpretation of these facts. These cities very often are not so intensely malarious as they are supposed to be. The opportunity, then, does not frequently offer for a combination of the two poisons in the same individual; and when it does occur the symptomatology of one disease, as a rule, entirely dominates the other for the time being. The mere exaggeration of the diurnal range of temperature cannot be considered sufficient evidence of a malarial complication. This complication has been, in my experience, exceptional, and it has shown itself almost exclusively during the convalescence.

A tendency to phlegmonous inflammations may present itself in the course of yellow fever. These may give rise to phlebitis and lymphangitis that are not, as a rule, of serious character.

Hepatitis, insanity, and paralysis (probably neuritis) I have seen as sequelæ of yellow fever, but never in children.

Relapses are rare in children. Except in cases that assume a typhoid form, convalescence is, as a rule, rapid.

In regard to second attacks, it is only necessary to say that the successful colonization of intertropical America by the Spaniards and Portuguese depends entirely upon the fact that one attack of yellow fever confers an immunity that is practically certain and that lasts throughout life, even though the individual may remove his residence to a colder climate for long periods of time.

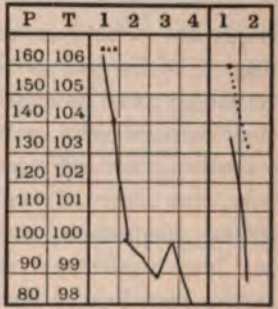
Very rare exceptions to the above rule do occur, and they consist generally of two distinct attacks during the same epidemic.

Diagnosis.—I do not know of any disease with which an ordinary case of yellow fever can be confounded if subjected to observation for two or three days. Yellow fever differs in every particular from the hemorrhagic, the hæmoglobinuric, and the remittent malarial manifestations. The differences are so prominently brought out in tables of differential diagnosis in monographs and treatises that one wonders at the necessity for the construction of them. There are, however, mild cases in children where the diagnosis from ephemeral fever is difficult, at least with our present limited means of observation. Limited, I say, because in those localities where the opportunity offers for a continued investigation of these cases, they are never studied from the point of view of yellow fever.

I cannot insist too much upon the importance of recognizing these cases. The epidemic influence often shows itself first upon children. The first recognized case of an adult may not deserve the importance that is attached to it as the starting-point of the epidemic. I can say this much, that in the absence of cases of actual sickness, because of the impossibility of a house-to-house inspection in a city, I have been able to detect the presence of yellow fever by an analysis of the infantile mortality. A notable increase of the deaths of children from remittent and pernicious fevers, from dentition and meningitis, may be considered as warranting suspicion at least of the existence of a spreading infection of yellow fever. The importance of detecting the first cases out of which this mortality sprung is apparent.

In warm countries the question of diagnosis is complicated by a remarkable liability of children to fevers that may be termed functional fevers because they arise from an excessive demand made upon any of the important functions of the body. The function of heat-inhibition must be overtaxed in the long summers of the tropics. This alone, I believe, is often the primary cause of fever in those regions. The term thermic fever proposed by Wood describes these cases satisfactorily. Short cases of thermic fever will be readily confounded with yellow fever. As a means of diagnosis I can only insist upon the three cardinal points mentioned in the symptomatology,—namely, the relations of the temperature and pulse, the facies, and the albuminuria. Without a careful study of these symptoms the diagnosis may be impossible. In practice it may occur that the facies is the only obtainable evidence. This should be assisted by a careful inquiry as to whether there is any cause to which the fever may be ascribed. The cases represented in the accompanying chart had a very suspicious appearance. At least the face was very much injected. It was, however, more the appearance of scarlet fever, with blood-shot eyes, but without any icteric hue. Furthermore, the children had been taken sick towards noon with a chill, after several hours' exposure to the sun. A case of yellow fever commencing with the violence shown by the first case could not have subsided so rapidly, and would have presented, in all probability, albumen in the urine passed on the second evening. The events of the second day showed that the exclusion of yellow fever was correct. These were not cases of malaria. No quinine was given. The first case was seen

FIG. 13.



Thermic fever of short duration.

with Dr. Bordas and treated with jaborandi. The second case I treated by sponging with cool water and alcohol.

Prognosis.—Yellow fever is much milder in the child than in the adult. In a series of one hundred and sixty-one adults attacked with yellow fever, forty-eight died, or twenty-nine per cent. A series of one hundred and twenty-one children treated during the same epidemic numbered thirteen deaths, or ten per cent. The deaths of colored children must be very rare.

Among the unfavorable symptoms we should notice an extraordinary rise of the temperature during the fastigium, especially about the time when the lysis should commence, or when the temperature has already started on the line of descent. If the pulse rises rapidly at the same time, and the temperature reaches a maximum above that of the initial stage, the prognosis is almost necessarily fatal. A slow pulse, if it steadily loses in volume and resistance, is a grave sign, even though the temperature may be following a favorable course. Great agitation and increasing frequency of the respiration are also of very serious import. The suppression of urine is a grave symptom, though it is more easily overcome in children than in adults. It may be said in general that mild cases have very little albumen in the urine, and fatal cases have much; but many presenting the largest quantities of albumen and casts will recover. It is the rapid increase of the albumen in the second or third day that constitutes the most alarming information derived from this sign. The progressive deepening of the color of the vomited matters is also of very unfavorable omen.

The favorable signs will be evident from the above considerations. But I wish to add that a prolongation of the case beyond the sixth day may be taken as a favorable sign, though the patient may present very alarming symptoms. These are the typhoidal cases of which I have said that they generally recover after a prolonged struggle.

Treatment.—The majority of practitioners in this country who have to contend with yellow fever are of opinion that there is something very important to be done in the management of this disease during the initial stage. If the case is taken in hand early (this is the favorite expression), no danger need be apprehended. One is led almost to believe that there must be some great specific which if promptly administered is sure to exert a decidedly favorable action upon the course of the disease. We find, however, that these advocates of an active early treatment differ very much among themselves; and, furthermore, we find that each particular plan has been condemned as useless or injurious by some physician of great experience. Now, I have no reason to say that these different plans of treatment are in themselves injurious, but the belief in their efficacy has certainly led to a most unfortunate state of the public mind during epidemics of yellow fever. "What is to be done?" "Something must be done," is the cry, and the observers and the patient stand in dread, counting the valuable moments that are lost before the expert yellow-fever doctor and the yellow-fever nurse arrive, and before the mustard bath is ready and the blankets piled upon the patient. And woe betide the patient if none of these things are within easy reach, or if the doctor happens not to believe in the efficacy of the classical sweating! The frame of mind of the patient becomes a very unhappy one, or is made so by the whispered accounts of the numerous cases that have terminated fatally because of the want of a mustard bath or of a dose of castor oil or of calomel. People will dread moving out of the infected area, will avoid going to camps that may be provided for their exit, will become panic-stricken, because they may not have within reach the above-mentioned paraphernalia. Now, if all this fear and tribulation could be avoided, the number of lives saved would, in my opinion, exceed the number that have been saved by any plan of treatment.

The fact is that popular prejudice has held the treatment of yellow fever to the plan that was followed many years ago in the management of acute febrile diseases. The authority of great names and of centres of medical learning has been wanting in many of these sections to bring about the changes that have been elsewhere generally accepted. And yet we have but to open any authoritative treatise on the diseases of the tropics to find a strong condemnation of such plans of treatment. Nor is it difficult to prove that more recoveries occur among patients that are not so treated; because their number includes the isolated cases treated, in a measure, in the open air, and away from the infected area; and in this number is included also a considerable proportion of the cases of negroes, who generally recover because of the mild character of the disease in that race.

Instead of active interference, I must confess to a feeling of helplessness in the presence of the storm of the invasion of yellow fever. I do not know what is the cause, and therefore do not know what to do to control the morbid process.

In the treatment of children it will be found that the following measures may be considered as safe for the relief of more or less dangerous symptoms. If the bowels are inactive, cream of tartar should be given. This may be taken during the evening with a free supply of water. Magnesia is preferred by some, to counteract the acidity of the stomach. In older children, who can take capsules, I use often the compound jalap powder in laxative doses. If they are easily swallowed, these capsules are almost always retained. They appear, in fact, to arrest vomiting, and I have continued to administer them at intervals, in some cases, to the exclusion of other treatment,—only in such doses, however, as will keep up a moderate activity of the intestinal secretions without griping.

If the stomach is very irritable and the food is not retained, calomel should be given in preference to other laxatives, and in minute and frequently-repeated doses. The admixture of lime-water with the milk, or the administration of small doses of carbolic acid with bicarbonate of sodium, or the use of ice, will often prove a good substitute for the calomel.

In other cases our attention will be directed to the relief of headache and restlessness. Cold applications to the head may or may not be soothing.

A sinapism to the back of the neck will be found beneficial. The most decided relief of these symptoms I have obtained from the use of antipyrin or of Dover's powder. Either of them may be recommended during the fastigium of the fever in ordinary cases. In grave cases of short duration I employ the acetate of ammonium with tincture of digitalis, to keep up the activity of the circulation; but I doubt the utility of any medication in these cases. It is certainly pitiful to see these little children treated with blisters to the arms and legs, and taking bromides and aconite for the relief of cerebral symptoms. Many of these patients are treated with large doses of quinine, on the ground that they are cases of pernicious malarial fever; and I have no doubt that the alarming and fatal symptoms may be the result of this medication. Such cases, I believe, were more common in the practice of physicians who denied the existence of yellow fever among the children.

The treatment of the suppression of urine is often a hopeless task. It generally occurs in the desperate cases just mentioned, and there is neither time for nor profit in the employment of special measures for this purpose. The digitalis recommended above will fill this indication, if there is any possibility of filling it. In the more protracted cases, the use of stimulants and tincture of the chloride of iron may overcome the difficulty. If the complication continues, I would advise calomel as the most certain diuretic to be used. I have seldom employed it in children, because the suppression of urine does not often arise as a pressing indication for treatment; but in the adult the effect has been very remarkable. I have given two or three grains every four hours, in capsules, either alone or in combination with small doses of compound jalap powder. The urinary secretion is started often before the third dose is administered. I used calomel as a diuretic in yellow fever for the first time in the epidemic of 1887. On the recommendation of Dr. Sternberg, the bichloride has been since used, with the hope of destroying pathogenic microbes in the intestinal contents. There is no proof that the theory has been made good by the experiment. But I am informed by those who used the bichloride that it certainly had the effect of increasing the secretion of urine and diminishing the amount of albumen.

In regard to the use of cold, which would appear to be indicated in the treatment of yellow fever, I may say that I have employed cold baths and have abandoned them in the febrile diseases of children. Children under the age of seven years are very apt to show evidences of blood-stasis in the cold bath before the internal temperature has been materially reduced.

The influence of therapeutics to determine a favorable result is shown unmistakably only in those cases that are prolonged beyond the sixth day, —the typhoidal cases previously described. In these the black vomit will appear by degrees. As soon as the black striæ begin to show themselves in the vomited matters, or even before, if the lysis is unusually slow and the asthenia marked, we should prescribe the tincture of the chloride of iron in doses of five or ten drops every three or two hours. This treatment is often followed by an arrest of the hemorrhage and diminution of the vomiting.

In these cases great importance attaches to the use of alcohol. Good brandy should be given, diluted with milk, water, or carbonic-acid water. In many cases iced champagne is very well borne by the stomach.

I recommend under the circumstances that special attention be paid to the change of clothing and the removal of the clammy secretion of the skin. This is best done by light sponging with diluted chlorine-water, tepid or cool, as the condition of the patient may require.

In some cases during this stage the jaundice may acquire unusual prominence, and a slight enlargement of the liver will be noticed. I recommend then that the chloride of iron be substituted by chlorate of potassium.

Upon the judicious use of the iron, alcohol, and chlorate of potash, with a nutritious diet, depend, in my opinion, the few triumphs that therapeutics may boast of.

The mortality of yellow fever is considerably reduced when the patients are treated in tents, or in well-ventilated provisional buildings outside of the infected area, provided all crowding of people together be avoided.

The frequent administration of food in small quantities during the lysis is probably of great importance, and the preference should be given to milk. We may substitute for it, at times, strong meat broths, especially when the time allowed for resting has brought together the hours for feeding and the administration of the iron. Soft-boiled eggs are tolerated even before convalescence is well established. Alcoholic preparations containing extract of beef may be used with advantage in the protracted cases. I have seen in the practice of Dr. Charles Faget a preparation of this kind—the elixir Duclos—employed successfully.

The use of cool acidulated drinks is very generally recommended, especially in the early stages. It is stated with much truth that the lemonade if boiled and subsequently cooled will be better borne by the stomach.

Prophylaxis.—The mild character of yellow fever in children and in the negro is a serious obstacle to the prevention of the spread of epidemics, because individuals of those classes may constitute unrecognized sources of infection. Another obstacle is to be found in the unfortunate tendency to conceal the first manifestations of the epidemic. Lastly, the most serious obstacle is to be found in the meagreness of the provisions made for quarantine purposes through the indifference of the public to sanitary matters.

These obstacles, it must be confessed, have rendered comparatively useless all attempts to enforce successfully a reasonable quarantine. I cannot understand, however, why this should be considered proof against the advisability of attempting to restrict or regulate the intercourse with an infected district. Much has been said of the good results obtained by the English with their methods of comparatively free intercourse. The truth is that wherever the territorial circumstances are similar to ours there is nothing in the success of the English that is worthy of admiration and imitation. To speak of yellow fever only, we find that no control has been exerted in the West Indies over the ravages of the disease by any European government. The tendency is rather the reverse, for we find that centres of periodic epidemics (Class II.) are assuming the character of centres of annual epidemic. This has been especially well shown in the island of Cuba since 1761. In the United States, on the other hand, it appears that important centres of the second class have been converted into centres of the third class, or of accidental epidemics. And let not those tropical countries comfort themselves with the feeling of safety that rests upon the belief in the immunity of the native population. They see their commerce prosper, and they wonder at the dread of the disease that is shown in other quarters. But their sense of security is a fallacious dream. Let them reckon by the infantile mortality the price they have to pay for their immunity.

The United States government, after gradually improving its system of maritime quarantine, has very recently undertaken to carry out measures for the restriction of epidemics of yellow fever by land-quarantine. Unfortunately, the means to carry out these measures, and the authority to enforce them, were very limited. But the national health authorities have, I believe, shown what might be done if these limitations were removed. The object in view was the depopulation of infected districts without danger to other sections. For this purpose a probation camp was established at Camp Perry, where persons coming from an infected district were detained for a period of ten days. This experiment was made on a somewhat limited scale, and some of its features evinced a hasty preparation. It was shown, however, that this camp could be kept uninfected, and that more than one thousand people who passed through the establishment could have been received with perfect safety by any community. Unfortunately, the local quarantine authorities, over which the government could have no control, chose to disregard these facts. They were influenced by popular prejudice, and were obliged to treat Camp Perry as though it had been an infected place. Of course this became an obstacle to the usefulness of the camp as a means of depopulating the infected district.

What appears to be wanting to carry out these measures effectually is a permanent organization in the shape of permanent quarantine posts and dépôts, and a standing army, after the fashion of the military organization of the nation. This is the only way of preventing the fatal stampede of the first days of an epidemic. Instead of this, what do we find? A vast disproportion between the appropriations of money made for the War Department and those made for sanitary purposes: in the former case many millions of dollars annually supplied to keep up the defences; in the latter, a few hundred thousand dollars, most of which are made available only after the epidemic invasion is well established. And yet there is no doubt that our imported epidemics have been vastly more destructive to life and capital than our foreign wars.

Another desideratum is the education of the people to a proper understanding of the prominent facts of epidemics. They should know that epidemics commence, as a rule, about concealed cases, that the danger lies in the unknown, but that there is very little risk even in handling cases that are properly labelled yellow fever. These are isolated, and removed from crowded places, and a proper disinfection of their effects should be made by boiling. People should be taught also to travel in times of epidemic with little baggage and such as can be easily and effectually disinfected. It certainly does not seem unreasonable to ask them to give up for the nonce their silks and trappings.

DENGUE.

By RUDOLPH MATAS, M.D.

Definition.—A febrile epidemic disease which is contagious or transmissible and characterized by a polymorphous and often dichronous cutaneous eruption, by very intense muscular and articular pains of a rheumatoid character, and by a cyclical evolution in four periods, the last being that of convalescence, which is prolonged and difficult. This purely symptomatic definition, which we have adopted from Mahé,[1] is properly completed by the addition of the following distinctive features which are peculiar to the history of dengue,—viz., that it is an epidemic disease of intertropical or warm climates (it has exceptionally crossed the thirtieth parallel of north or the twenty-fifth of south latitude); in addition, that it was but very indistinctly recognized and never completely described before the commencement of the nineteenth century; and, lastly, that it is, *as a rule*, a non-fatal malady.

Synonymes.—Unknown or unfamiliar epidemic diseases, which overrun wide and different territorial areas at considerable intervals of time and afflict numerous and varied races of people speaking different languages, usually receive a new name in each particular country which they visit, and the polyglot and word-laden synonymy which results therefrom constitutes only the first difficulty encountered by the investigator. This knowledge, however, is indispensable in order to assimilate all those affections described under different names into one common species. Owing to the intense stiffening and distorting pains which so pre-eminently distinguish the disease, the following names have been given to it: Eruptive Rheumatic Fever; Eruptive Articular Fever; Exanthematic Articular Fever; Articular Fever of Warm Climates (Thaly, Senegal); Rheumatismal Fever with gastric irritation (Furlong, United States); Rheumatic Scarlatina; Arthrodynia (Cock, United States); Trancazo [club-blow], (Santa Cruz de Teneriffe); Pantomima (Cadiz); Stiffneck; Giraffe; Broken-wing; Break-bone Fever, a favorite designation in the United States; Polka Fever (in Brazil) and Dandy Fever, first used by the negroes of St. Thomas, West Indies, in

---

[1] Mahé, Dict. encyclop. des Sciences médicales, art. "Dengue," Prem. Série, tome xxvi., Paris, Masson-Asselin, 1880.

1827, because of the stiffness with which convalescents from the disease are often compelled to walk.

The following may be attributed to the eruptions: Exanthesis Arthrosia, Rosalia, Colorada (Spanish colonies); Calentura Roja (Poggio, in Cadiz and Teneriffe); Simple Red or Exotic Fever (Senegal, Cayenne, Réunion).

Owing to its intense epidemic character and some of its geographical peculiarities, dengue has also been called Spinal Epidemic Fever; Anomalous Fever; Calcutta Fever (Mellis); Epidemic Eruptive Fever of India and Toohutia; Malta, Mauritius, Chinese, and Mediterranean Fever. Then a host of other appellations, some rational, others merely curious and bizarre: thus, Date Fever in Arabia, Port Said, because it usually appears about the date-season; Inflammatory Fever; Three-Days' Fever (India); La Piadosa, "the kind, the charitable fever" (Cadiz); Fiebre Rusa, Gaditana, Influenza Plantaria; Biliary Fever; Bou-Hou or Wailing Fever, Sandwich Islands; N'Dagamonte, N'Dongomonte, N'rogni (indigenous Senegalese); Knee-disease (Arabia); Abou-dabous (Tripolitan Arabs); Kidniga Pepo (Zanzibar); Dunga, Dengue, equivalent to "coquettish," "foppish," in Spanish, from which the word *denguero* or *dandy* (more modern *dude*) is derived. Campbell[1] and others suggest, contrary to the previous explanation of the origin of the word, which is that of Hirsch and Charles, that it is a mere corruption or phonetic English adaptation of the Spanish word *dengue* (dangy, dandy, dengue). Anyway, whatever may be the origin of the word or the reasons which may have led to its adoption, it is a fact that the word dengue has persisted and dominated more than all others in the literature of the subject and has become somewhat official since it was adopted by the Committee of the College of Physicians and Surgeons of London in 1869 in their System of Nosological Classification, and has since been universally adopted to designate it by all writers, irrespective of nationality.

History.—Though vaguely described, it is pretty certain that dengue prevailed epidemically and quite extensively during the latter part of the last century. Beyond this, the early history of the disease is lost. If we were to hold strictly to the dates indicated, it would appear according to the chronicler Gaberti, quoted by Pruner, that in 1779 a disease corresponding n its description to dengue prevailed extensively on the Arabian coast and n Cairo, Egypt. Almost at the same time a similar disease prevailed on he Coromandel coast, if we are to accept the evidence of a French missionary, Persin. In 1779, David Brylon described the disease as it prevailed n Java under the name of articular fever. In 1780, Benjamin Rush appears to have observed it in Philadelphia under the name of bilious remittent fever (Wood). Pezet also recognized it in Lima, Peru, in 1818.

These citations would tend to prove that dengue had become disseminated over a very large area before the advent of this century, since it had been recognized in at least three very distinct points represented by the three continents Asia, Africa, and America. It might even be added that it had also prevailed in Europe, if it had been observed, as is pretended with good reasons, in Cadiz and Seville in 1785 by Nineto de Pinta.

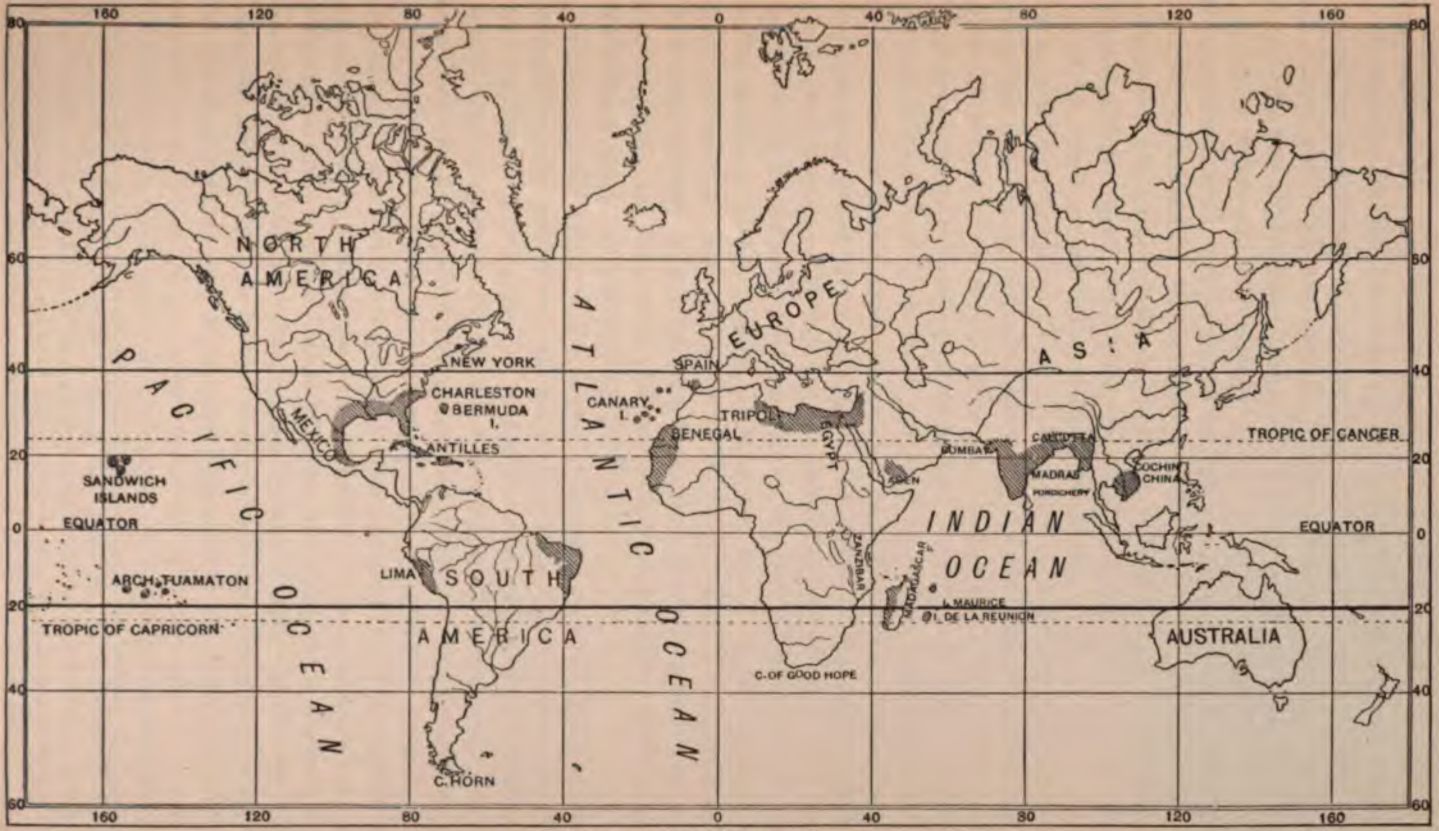
---

[1] Language of Medicine, 1888, Appleton, New York.

It was certainly in India, however, that dengue was first well described from 1824 to 1826, when such men as Mellis, Twining, Cawel, and Mouat contributed to its literature. A little later, from 1826 to 1828, the great Antillean epidemic which so extensively prevailed in the Southern United States was observed and described, with all the care and attention possible at that time, by a host of most competent observers, American, English, Spanish, and French. Since that time, as our knowledge of the disease has increased, its clinical differentiation been perfected, and our means of communication, both by steam and by electricity, so largely and increasingly facilitated, the prevalence of the disease has been much more frequently noted, though its epidemic itinerary has rarely transgressed the boundaries of tropical and temperate latitudes. Limiting ourselves to the Western Hemisphere alone, we will notice that in 1826 it prevailed in Savannah, Georgia; in 1827 to 1828, in St. Thomas, Santa Cruz, and other Antilles, Colombia, Bogota, Carthagena, Hayti, Jamaica, Charleston, Mobile, and sporadically Boston, New York, and Philadelphia (Stedman, Cock, Furlong, etc.); in 1839 to 1844, in Iberville, Louisiana, and Mobile, Alabama; in 1846 to 1848, in Brazil, where almost all the inhabitants were attacked with the disease (polka fever); in 1848–50, in New Orleans, and along all the Gulf and South Atlantic States,—a most extensive epidemic (Dickson); in 1850 to 1852, in Peru; in 1854, in Havana; in 1855 to 1856, in Martinique (Ballot); in 1861, reappearance of dengue in Texas and the Southern United States; in 1866, reappearance of dengue in the Southern United States; in 1874 to 1875, epidemic in Martinique,—imported, it is said, by immigrants; in 1880, in Charleston, Savannah, New Orleans, and other cities of the Southern States; in 1885, a most extensive and violent epidemic, exclusively limited to the State of Texas.

The sum of evidence furnished by the history and geography of dengue would indicate that this disease was originally an Asiatic tropical infection, starting perhaps in India, which was subsequently diffused to its present wide geographical area of distribution by new discoveries, increased commercial relations, etc. Furthermore, that, though the disease cannot be said to possess perennial endemic foci of prevalence, still it is known to prevail sporadically and with great frequency in Northern Egypt, India, and perhaps our own United States; Europe, with the solitary exception of the warmer provinces of Spain, having thus far enjoyed almost complete immunity.

We will not dwell further on this aspect of our subject, as it is not our particular province to do so, especially in so narrowly limited an article as this. Any one further interested in this field of research will find a more ample consideration of it in Hirsch's "Handbuch der historisch-geogra-



Geographical distribution of Dengue.

phischen Pathologie," Creighton's English translation, Sydenham Library, 1887, or in Mahé's learned article, *loc. cit.*, from which the major portion of the preceding data has been condensed.

Etiology.—Dengue is essentially an epidemic disease, though it sometimes occurs sporadically in the countries in which it more habitually prevails. Age, sex, social condition, race, and nationality appear to have no influence on the production of dengue. Infants at the breast, only a few weeks after birth, are stricken side by side with septuagenarians. The infirm valetudinarian and the most healthy and vigorous youth are alike exposed to its influence. There are few diseases which are such great levellers as this. Climatic conditions, insomuch as they are related to thermic influence, are of importance. It has been justly observed that it is particularly in the summer months that dengue breaks out in the regions situated just outside of the tropics. There are some exceptions to this, especially in North America. Geographical distribution of the disease shows plainly (see map) that its prevalence is influenced in a great measure by the distribution of heat on the surface of the globe. In the Northern Hemisphere it has rarely crossed, at least as an epidemic, the thirty-second parallel in America and the thirty-sixth in Europe; in the Southern Hemisphere it has never gone below the twenty-first; but within these extreme limits there are few diseases which have spread so thoroughly over a large surface. The extent of its itinerary, the rapidity of its march, the multitudes of victims[1] which it strikes at once, its benign character which contrasts so markedly with the apparent gravity of its symptoms, are all peculiarities which tend to bring it in relief and throw it in strong contrast with most other pandemic maladies, with perhaps only one exception,—influenza,—with which it has great epidemiological affinities.

After a careful study of the disease it becomes evident that the great thoroughfares, the public ways of the world's travel, are the routes also followed by dengue in its migrations, and that, consequently, the more numerous and frequent the transit of human beings along these routes, the greater the rapidity of the spread and the greater the frequency of its visitations.

A great deal has been said in regard to the atmospheric, telluric, and climatic conditions which are favorable and unfavorable to the spread of dengue, but almost all this class of information is of a very misty and unstable sort, so that, outside of what has been said in regard to the favorable influence of heat, little more can be positively affirmed. It has been asserted that altitude has a decided effect in unfavorably influencing the infection, but this influence, the most competent observers (Catholendy, Mahé, and others) inform us, is due merely to the difference in temperature, and not of altitude alone. In Réunion, the high table-lands of Salazie and of Cafrés (nearly two thousand metres above the sea), and even the much lower level of St.-Denis in the same island (seven hundred to eight hundred feet), were never troubled with the disease, while, on the contrary, the lower country below these points was completely swept by it. Like other great epidemic diseases, it prefers large populated centres, not, of course, because of an *inherent* preference, but on account of the greater pabulum. Though a warm climate appears to be almost an indispensable requisite for its proper propagation, it is a notable fact that cold does not markedly diminish the spread of dengue epidemics. (*Vide* Thomas, *loc. cit.*, epidemic of Savannah, Georgia, most intense in the exceptionally cold winter months of 1880 to 1881.) Again, as will be noticed farther on, one attack of the disease is not protective from further attacks, though on this point there is some division of opinion.

---

[1] It is by millions that we must count the victims of the disease in the Indian epidemic of 1872. At Réunion, dengue swept over the whole island in three months; there, at St.-Denis, out of a population of thirty thousand, twenty thousand were stricken with the disease. In the recent epidemic of 1885 which prevailed with so much violence in Texas, McLaughlin estimates that in the city of Austin alone, out of a population of twenty-two thousand, sixteen thousand persons were attacked in the course of a few months.

From these remarks it will be gleaned that little, if anything, is actually known of the auxiliary agencies or factors which influence the development of the dengue poison, whether in a favorable or an unfavorable direction. As to the *vera causa*, the essential ferment or contagium, which is the *sine qua non* in the etiology of the disease, our knowledge was still more deplorably scanty until the advances of modern bacteriological research gave strength to the numerous suggestions that had been offered by almost every writer on the disease since the earlier glimmering of the panspermist doctrine. All ideas in this direction were entirely conjectural and hypothetical until 1885, when the unusually fertile and rich field offered by the great epidemic which prevailed in the State of Texas was utilized by Dr. J. W. McLaughlin, of Austin, and the first step taken to examine the etiological problem with all the lights that modern science could lend to the inquiry.

McLaughlin's investigations[1] were not completed, but the facts obtained by him are sufficiently striking and valuable, if confirmed by further investigators, to support the most sanguine expectations that may be entertained as to the early and positive discovery of the essential etiological factor.

As a prominent and active practitioner in Austin, Dr. McLaughlin enjoyed exceptional advantages for obtaining material and assuring the genuineness of the cases investigated. These investigations covered the space of six months. The work actually performed during this time is embraced in the following paragraphs:

1. Blood which was obtained from living subjects during the various stages of dengue was microscopically examined (*a*) directly, that is, without the addition of any chemical reagents; (*b*) after it had been subjected to the action of certain chemical reagents,—viz., glacial acetic acid with and

---

[1] Researches into the Etiology of Dengue. By J. W. McLaughlin (President Texas Microscopical Society, Austin, Texas), Jour. Amer. Med. Assoc., June 19, 1886.

without dilution, caustic potash in solution, both weak and strong, chloroform, and ether.

2. This blood was carefully dried upon sterilized cover-glasses, by passing them through the flame of a spirit-lamp, and then subjected to the action of various staining reagents.

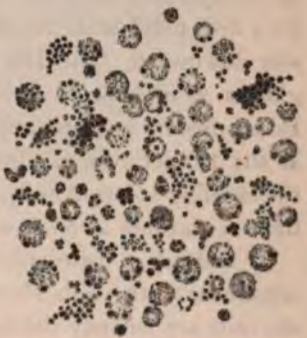
3. Dengue blood, obtained from living subjects, was introduced upon the points of a platinum wire into test-tubes containing sterilized culture-jelly prepared for this purpose. These tubes were closed with plugs of sterilized cotton, then placed in an incubator, where the temperature was kept at 100° F. for the growth of such organisms as were contained in the blood.

4. Blood was drawn directly from the veins of a living subject into a series of sterilized glass bulbs which were united by a capillary tube. This was performed in such a manner that it seems impossible for germs from the air or by other accidental means to have gained an entrance into these bulbs. These were also kept in an incubator at a temperature of 100° F.

5. The matter vomited and urine passed by dengue subjects were subjected to microscopic examination.

Dr. McLaughlin summarizes the results obtained from the preceding methods of examination as follows: "In the blood examined directly or after its treatment with the chemical reagents already referred to, stained or unstained, I *invariably* found, often in great numbers, in the cell elements as well as in the plasma, micrococci about $\frac{1}{20}$ to $\frac{1}{30}$ the diameter of the red cells, spherical in shape and red or purplish in color. When these were seen in great numbers, one layer was superimposed upon another; frequently seen in the cultures they appeared of a black or brownish color, but when seen singly or in thin layers in the blood or in cultures the red color is always distinct and characteristic.

"During the development of this organism, at some period in its life-history, from causes which I do not understand, it becomes surrounded with a gelatinous envelope: this I have frequently observed in the blood and in the culture alike."[1]



Blood of dengue fever, from culture-bulb, showing micrococci in the red cells and in the plasma. In the latter they are found singly and in zooglœa masses. (From McLaughlin's paper.)

McLaughlin always succeeded in growing in culture-tubes upon the surface of jelly micrococci, and no other form of bacteria, which in color, size, and behavior are identical with those seen in dengue blood. The blood contained in a series of glass bulbs was examined, some after the lapse of six weeks, some at three months: in both instances it was found that the blood contained a pure culture of micrococci which in all respects were the same as those previously seen in fresh blood. The blood for examination was obtained from about forty typical cases of dengue at various times and places and during the various stages of the disease. The results obtained from examinations of these different specimens were entirely uniform. By systematic attempts at staining with Bismarck brown, vesuvin, gentian-violet, methyl-violet, fuchsin, methyl-blue, aniline-green, picro-carmine, and eosin, Dr. McLaughlin came also to the conclusions, first, "that the dengue micrococci do not stain with aniline as readily as do other forms of bacteria;" second, "methyl-aniline blue in a weak solution of caustic potash furnishes a staining-fluid for which the cocci of dengue manifest an especial affinity. With all the other dyes named the results were negative; *i.e.*, all parts of the picture were stained alike,—cells and organisms,—and they were all decolorized with equal facility when washed in one-per-cent. solution of acetic acid and then in absolute alcohol. With the methyl-blue potash solution, however, a very different result was obtained: this dye, in the solution referred to, manifested such an affinity or elective action for the organisms of dengue that these would retain the blue color after this had been extracted from the blood-cells by the decolorizing agents named.

---

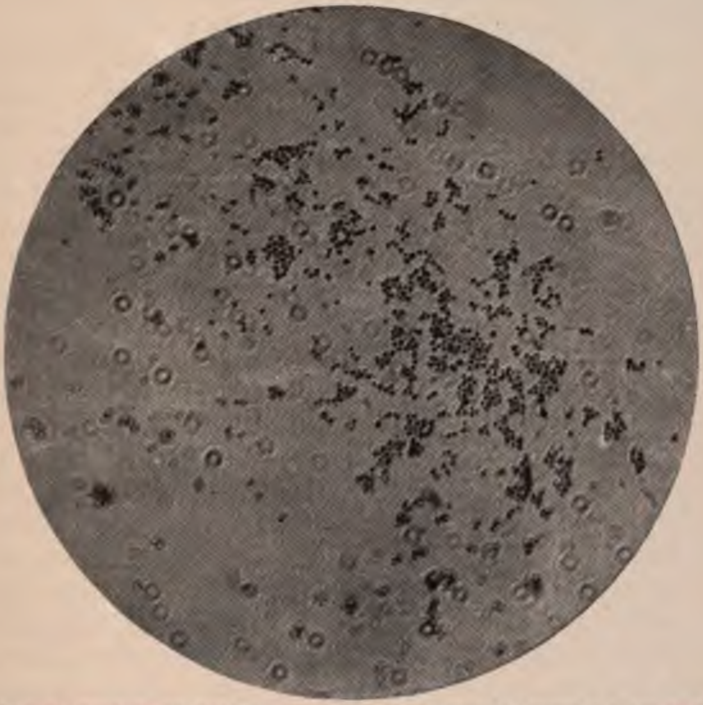
[1] It is worth noting that in the examination of dengue blood made in Calcutta in 1872 by Cunningham and Charles some curious observations were recorded which appear to confirm the observation of McLaughlin, notwithstanding the technical imperfections of the then existing methods: "The moment the blood was collected from the veins it was treated

"The manner of preparing this solution and the method of staining with it which were adopted are as follows: Concentrated alcoholic solution of methyl-blue, 30 c.cm.; solution of caustic potash 1:10000, 100 c.cm. In a dish filled with this fluid the cover-glasses were floated with the blood-side downward. The dish was then covered, to exclude dust, and the cover-glasses were kept in this condition from twelve to twenty-four hours. Better results were obtained by keeping the staining-fluid during this time at the temperature of 100° F. The cover-glasses are then removed from the staining-solution and washed in the one-per-cent. solution of acetic acid, then in absolute alcohol, until the color is entirely or sufficiently removed.

"I think a better picture is obtained, and the relative position of the organisms to the cells shown, if the process of extraction is arrested before the cells are entirely decolorized: they should then be mounted in Canada

---

with a solution of osmic acid, and submitted afterwards to the action of an almost saturated solution of potassium acetate, with the view of fixing the white blood-corpuscles, in order to examine the specimens subsequently and more leisurely." It was found that a considerable increase in the number of Hayem's hæmoplasts (Bizzozero's blood-plates) had taken place. These little corpuscles were abundant, sometimes isolated and free, *at other times aggregated in small masses and held together by a granular and somewhat gelatinous material.* This condition of the blood persisted for a few days, and then the blood rapidly returned to its normal appearance and condition. These modifications were very appreciable from the third day, and would last as late as the sixth. These signs were wanting, however, in several cases of fever which had been diagnosticated as cases of dengue fever. (*Vide* E. Charles, Clinical Lectures on Dengue, Calcutta, The Lancet, 1871, 1872, 1873; quoted also by Mahé, *loc. cit.*).



Photomicrograph of McLaughlin's Dengue Micrococcus Culture: taken with 2.0 mm. hom. imm., 1.40° apert. apochromatic objective, Zeiss, and No. 6 Zeiss apochromatic projection eye-piece. Magnified 1250 diameters. (By W. M. Gray, M.D., U. S. Army Museum, Washington.)

balsam and examined with a high power ($\frac{1}{12}$ H 1 objective Grunow's N. Y., a histological stand, and Abbé's illuminating apparatus were used in these researches) and with a large diaphragm and open condenser. The blood-cells should show a faint blue color, whilst the micrococci, which are to be seen in the blood-cells and plasma, will be stained an intense blue. The inability of these organisms to hold the other aniline dyes, acid or basic, to which they were exposed, their uniform size, their presence in the blood-cells, their ability to resist the destructive action of acids, alkalies, ether, etc., it would seem, are sufficiently distinctive to differentiate them from protoplasmic granules or the products of cell-disintegration. An additional reason for regarding them as micro-organisms exists in the fact that they can be and have been grown upon culture-media outside of the body."

We have thus far almost literally quoted Dr. McLaughlin's account of his method of distinguishing the peculiar micrococcus which he has discovered in the blood. We will not continue with him in his detailed description of the methods adopted for the cultivation of the micro-organism out of the body, which he succeeded in growing very successfully. To go further into other technical details would be entirely unpractical and out of place in this contribution: in fact, the stress which we have laid upon the technical directions recommended by McLaughlin for the recognition of his micro-organism would be certainly out of place were it not for the consideration that by thus quoting them *in extenso* we are not only illustrating the pains-taking method adopted by McLaughlin in his careful research, but are also laying before the reader a method which is so simple that, if the practitioner be a simple clinical microscopist, he will find no difficulty in repeating it quickly and correctly. He will, therefore, be materially assisted in carrying out a test-investigation which, if corroborative of McLaughlin's observations, will certainly prove of immense value in the sometimes difficult and quite perplexing differential diagnosis of this remarkable disease. If in this respect alone McLaughlin's discoveries are proved true and correct, then he will have rendered a service which should make science grateful to him.

Now, as regards the *etiological* value of McLaughlin's observations, little can yet be said: the observations thus far are limited to the discovery of a micro-organism and its cultivation out of the body in blood and other culture-media; the other conditions to prove the essential etiological value of a micro-organism, laid down by Koch and other writers, are wanting, notably the experimental reproduction of the disease in the lower animals and man by the inoculation of the cultured colonies. The very careful precautions, the uniform results, and the generally conscientious methods of research followed by Dr. McLaughlin would lead us to believe that there can hardly be any doubt as to the close causal relationship of the micrococcus that he has discovered with the disease, though we must wait patiently for another opportunity to present itself before the full evidence needed to establish the absolute importance of this micrococcus in the causation of this disease can be indisputably asserted. In the mean time we sincerely hope that the means of recognizing the micro-organism, so much insisted upon in these pages, will be borne in mind and will be utilized at each and every opportunity.[1]

Clinical History and Symptomatology.—*Varieties.*—That dengue fever is a disease which presents a variety of types is a statement that will be borne out by any one who has had an opportunity of personally observing this affection. In this respect it resembles most of the epidemic diseases which prevail in widely different territorial areas and among a great variety of races. It is strikingly like yellow fever in this respect, and the distinction made by Charles, of Calcutta, of *denguis latens*, *denguis mitis*, and *denguis maligna* is true in so far as these terms represent the various degrees of intensity with which the dengue poison may affect the organism. It would be strange indeed if in this respect dengue should differ from the other infections with which we are more constantly brought in contact. It is owing to these differences in the degree of intoxication that clinical confusion is liable to be created in the differential diagnosis whether of the milder or the graver extremes, which are at times rendered especially obscure in tropical countries, owing to the wealth of these countries, as a rule, in all the varieties of allied forms of epidemic pyrexiæ. The extremely severe or mild types and, consequently, more perplexing varieties of this disease, which may be regarded, fortunately, as its exceptional manifestations, because diverging from the ruling type, will be considered subsequently. At present, in order more clearly to grasp the physiognomy of this disease, it is preferable to describe that group of symptoms which when present must be considered peculiarly distinctive and characteristic, and, when regarded in their *ensemble*, pathognomonic of dengue. There are certain symptoms which are common to the vast majority of the cases,—those which have given to dengue its independent position in the nosology, and the absence of any of which is always sure to lead to doubts and perplexity when clinically dealing with the malady.

With Mahé and other competent observers, we will premise by recognizing in the clinical evolution of the more typical cases of this disease four leading phases, which are—

1. The onset with fever and, ordinarily, a transitory eruption,—the initial rash.

2. A remission, more or less long, more or less pronounced.

---

[1] In connection with the etiology of dengue it is of interest to mention the statements that have been made as to the transmissibility and epizootic prevalence of the disease among the lower animals. Cristobal Cubillas, as early as 1784, stated that in the epidemic of Cadiz a great number of animals were affected with the disease. According to the Bombay daily papers, a great many beasts in the Baroda district were afflicted with dengue, horses and cattle being especially liable (Martialis). In 1871, in the Baroda district during the prevalence of dengue a great many domestic animals were stricken as if paralyzed in the extremities, but rapidly recovered in three to four days. These interesting observations should certainly be confirmed and investigated. (Mahé.)

3. A second eruption, more pronounced than the first, the terminal rash with or without fever.

4. Desquamation and convalescence.

Period of Incubation.—This appears to be from a few minutes' duration (Bordier, Mahé, Rochard, and others) to five or six days (Fayrer), and to average four days (Catholendy).

It is almost universally admitted that a simple case of dengue has usually but few, if any, prodromata (Hirsch, Thomas, Holliday, Wise, Dunckley, Martialis, Charles, Fayrer, and others). These do exist sometimes, however; and Poggio, who is one of the most experienced writers on this disease, states as a result of his study of eight hundred cases in the Cadiz epidemics of 1865 and 1866 that the fever was constantly preceded by a prodromic period of variable duration, lasting from twelve to twenty-four hours, and characterized by signs of malaise, depression, lassitude, frequent yawning, repugnance to exertion, cephalalgia, and a sense of heaviness about the supra-orbital region. In our experience during the epidemic which prevailed in New Orleans in 1880 we were impressed, along with other observers, with the belief that the disease usually gives little, if any, warning of its approach.

In infants and younger children, as Thomas and other observers have remarked, the disease often begins with a convulsion, a child being waked up at night with a spasm. If the child is old enough to speak, it will complain more often of feeling cold or chilly along the back, and shortly after of cephalalgia, rachialgia, and arthralgic pains. This symptomatic tripod —cephalalgia, rachialgia, and arthralgia—should at once awaken the suspicions of the practitioner during an epidemic of the disease. The behavior of smaller children, of infants especially, will depend almost entirely upon the intensity of the attack; if the infection is slight or moderate, the little patients will give expression to their suffering by their great and sudden restlessness, agitation, and manifest discomfort, by constantly crying or moaning, and not infrequently by the repeated vomiting, especially of the breast-milk in nurslings. More serious are those cases in which the infant or child, after having had a convulsion, remains listless, apathetic, or in a stupor. In these cases the gastro-intestinal disturbance is more pronounced, vomiting being quite frequent, the vomit usually consisting of ingesta, mucus, gastric secretions, and bile. These cases are almost always associated with a high temperature, and will need very careful watching. All these phenomena may be entirely wanting, and the disease may abruptly present itself with fever and the characteristic pains. Stedman, Martialis, and Charles cite curious illustrations of the occasional lightning-like suddenness of the invasion: in more than one instance persons have been attacked in the act of imitating the painful distortions produced by the disease, and have been forced to continue a *bona fide* mimicry in spite of themselves.

No matter how the attack begins, an indefinable prostration seizes the patient, and fever begins; in adolescents and adults the pulse becomes hard and rapid, oscillating between 100 and 120 and even 140 (Twining) to the minute. In younger children the pulse is often so frequent that it is impossible to count it. The respiration in children is apt to be particularly hurried, though usually in proportion with the fever. The temperature begins to rise at once, and attains its maximum usually in from twelve to twenty-four hours, rarely after three days, and very rarely after five or seven days. The fastigium is generally very short, and the defervescence is rapid and characterized by a succession of remissions and exacerbations, which continue until the temperature has fallen one or one and a half degrees lower than the natural heat of the body.[1] During the next few days, if the temperature is closely watched with a thermometer, it will be found that it fluctuates from a degree below to one or two degrees above the normal heat. As will be again stated below, by the end of the sixth or seventh day there is a very slight rise again, being a secondary fever, but, as a rule, this heat soon subsides, and the temperature remains normal unless there is relapse, which is not uncommon even in the mildest forms of the disease after complete defervescence. The pulse often becomes slower than natural, and occasionally runs down in adults to sixty or sixty-five beats per minute (Thomas), though this marked retardation in the pulse-beat is not to be compared in its constancy and significance with that which is so notable a feature of the later stages of yellow fever.

It is during the first, pyretic stadium that the initial eruption is observed. In India this first rash has been observed in one-half of the cases, according to Martialis,—in two-thirds of them according to Charles. This eruption is usually very transitory and lasts only as long as the first or febrile period of the disease: it varies in intensity from a slight blush to a well-marked scarlatiniform erythema. It causes no desquamation and leaves no trace behind it.

During this period of invasion, or pyretic period, representing the first phase of the disease, one of the most constant and truly reliable and earliest symptoms is the cephalalgia, with the severe pain, which has already been mentioned, in some joints, usually one of the interphalangeal of the upper extremities, which rapidly extends to all the other joints and bones, this pain during the progress of the disease passing from one joint to another, as in metastasis. The severe pain in the head is very distressing, and, from the results that now and then follow, must be due to a slight hyperæmia of the brain or its meninges. "The heavy aching and throbbing of the temples and balls of the eyes, with dilated pupils, are marked and significant symptoms" (Thomas).

---

[1] Dr. D'Aquin, of New Orleans, asserted that there was a continuous and steady rise in temperature until the highest point was reached on the first, second, or third day of the attack; then a short stadium of a few hours; then a remission, soon to be followed by another fever, with rise in temperature, but never reaching the point of first maximum temperature. Holliday, also of New Orleans, and an equally competent and experienced observer, in commenting upon the above says ("Dengue or Dandy Fever," Trans. Amer. Pub. Health Assoc., vol. vi., 1881, p. 168), "These views are *not* corroborated by the reports received by me, which establish a distinct *daily* remission, with a fall in thermometer of from one to three degrees, with, of course, a corresponding rise."

The observations of D'Aquin have been confirmed by Vauvray, who observed the disease in Egypt, and quite recently by De Brun, who observed the epidemic which prevailed in 1888–89 in Beyrout, Syria (*Semaine Médicale*, March 6, 1889); while, on the other hand, Thomas, of Savannah, and Martialis, in India, and numerous other observers, speak of the remission as given in the text, and agree with Holliday. The truth of the matter is that, outside of the fact that there is, first, a paroxysm of fever, secondly, a remission, and then (usually), thirdly, a milder secondary thermic paroxysm, there is no fixed and *diagnostic* fever-chart of dengue. There is no doubt that there are cases presenting the D'Aquin and Vauvray tracings, but there is no doubt also that the type observed by Martialis, Holliday, and Thomas is equally, if not more, common, at least in our Southern epidemics.

To these febrile and painful phenomena symptoms of gastric disturbance are often added. The tongue becomes coated with a thick dirty-white fur in the centre, which contrasts with the red edges (J. Rochard, Fayrer, Mahé, and others); it is more often noticed in the experience of other observers (Holliday and others) that the tongue is uniformly coated with thick fur, as in most fevers, *and is not* notably red at the edges, differing markedly in this respect from the tongue usually seen in yellow fever. There is nausea, sometimes mucous or bilious vomiting, constipation, more rarely diarrhœa.

The urine is usually dark, scant, sedimentary, and, *as a rule, non-albuminous.*[1]

Pari passu with the subsidence of the fever, which takes place usually after the first forty-eight hours (Fayrer), often after three days, and more rarely after five, seven, or eight days (Mahé), the characteristic pains diminish and "critical" phenomena are liable to take place, such as profuse sweating, diarrhœa, and not rarely epistaxis, as in typhus. During this period of remission the patient is left exceedingly prostrated, stiff, and without appetite, if the attack has been at all severe: the prostration is particularly noticeable in children, in whom it is accompanied by extreme restlessness, not infrequently by delirium and insomnia (Holliday). Sometimes, on the contrary, the sick believe themselves entirely well, and will insist on getting out of bed. This apyretic period or intermission, though it is usually only a simple remission, may last one or two days (Wilde), three or four days (Fayrer), two to three days, or may be absent (Ballot). During the remission the third phase of dengue presents itself, and is recognized in the peculiar eruption or series of eruptions which give to dengue the characteristics of an exanthem. This eruption is polymorphous in character, sometimes erythematous like scarlatina, and at other times like miliaria, urticaria, and herpes. This terminal rash is more frequent in dengue than the initial eruption. It indicates the *lysis* of the disease, unless there be one or more relapses,—an occurrence which is far from rare. The eruption is usually accompanied by a very moderate rise of temperature; it sometimes appears in successive crops; it is also rare for the pains to return in this period. This secondary febrile movement and eruption lasts about two or three days, and then the fourth phase of the disease, or desquamation and convalescence, is inaugurated.

---

[1] The condition of the urine in tropical pyrexiæ is always a matter of great importance, both from the diagnostic and from the prognostic stand-point. The earliest observers of dengue satisfy themselves with noting simply the color, quantity, and reaction, and these are, as usual, most diversely described. Other observers, however, are much more satisfactory. Thus, Morgan noticed a specific gravity of 1004 to 1040, acid, *non-albuminous;* Chipperfield, acid, specific gravity av. 1010, *non-albuminous.* Goodeve detected an occasional *trace* of albumen in four cases in the Indian epidemic of 1853; while Charles and Martialis *never detected it* in the epidemic of 1872. In China, at Amoy, Muller and Manson *failed to find albumen.* Albuminuria was detected *only once* by Ballot in the epidemic of Martinique in 1860, and twice in Cochin China by French observers in 1873 (Mahé). Albumen was observed but exceptionally by Holliday and his colaborers (*loc. cit.*) in Louisiana. Enough has been said to prove that albuminous urine is an exceptional occurrence in dengue, which differentiates it markedly in this respect from yellow fever.

With the disappearance of the terminal eruption there is a general subsidence in all the acute phenomena, and convalescence may be fairly regarded as begun. The desquamation of the skin is more or less marked according to the intensity of the eruption. This process is frequently associated with intense itching: usually, however, the process is quickly effected, and convalescence, properly speaking, started; at other times, especially in weak, infirm, strumous, rachitic, or ill-fed children, and in aged persons, it is very slow in taking place and convalescence is equally retarded.

The preceding synopsis of the clinical history and symptomatology of dengue will cover with great probability the majority of cases. In the midst of large epidemics, however, there are all gradations of dengue-infection, some cases being so exceedingly mild as to be only perceptible, and others so violent that the identity of the disease is lost and the liability to confusion with others, commonly malignant and destructive fevers, enormously increased. In the mild forms of the disease the patients are scarcely ill, and, where it is not easy to decide as to their exact nature, "a trifling malaise, a white tongue, bitter taste in the mouth, headache, prickly sensation in the eyes, very slight pain in the limbs without fever, may only lead to a suspicion of the real nature of the case, until a symptom (the terminal rash) appears which shows you what you have had to deal with; and even this last symptom may be missing." Of the malignant form Charles says, "Drowsiness may have passed into coma; the temperature verges on the hyperpyretic [one case, 109½° F., fatal, quoted by Holliday, of New Orleans]; the heart fails, and the lungs are œdematous, while the whole surface is highly cyanotic." These cases have been popularly termed "black fever," and are very justly dreaded. Happily, such cases are rare.

Complications and Sequelæ.—Numerous complications have been mentioned in connection with this disease, but it is doubtful if any, outside of those related to the extreme prostration of the nervous system, can with any propriety be regarded as proper complications of dengue. Malaria is one of the poisons that is most likely to mark the course of the disease by giving to it its peculiar seal.

A typhoid condition was noticed by Poggio in the epidemic of Teneriffe in three cases.

In the pyretic stage, symptoms of cerebral hyperæmia, culminating in convulsions, coma, and even meningitis, have been noticed, but these are usually phenomena which result from hyperpyrexia. The eruption is likely, when very intense, to give rise to inflammatory œdema of the face simulating erysipelas, especially when in the neighborhood of the eyes, nose, or ears. Coryza, bronchitis, gastric and intestinal catarrh, dysentery, and catarrhal jaundice are occasional though not constant complications affecting the *primæ viæ*. Endocarditis, pericarditis, pleurisy, and arthritis (Zuelzer, Mooden Sheriff, Dunckley, Thomas) have been cited in support of the rheumatic character of the poison; but these complications are so very rare as barely to deserve notice, and are utterly unworthy of being regarded as evidence in favor of the nature of a causal agent. Glaucoma (Thomas), amaurosis, dementia, and transitory paralyses, neurasthenia and neuralgias, myalgias, and other evidences of the profound impression of the poison on the nervous system, are more deserving of attention. Epistaxis and hæmatemesis, in younger children especially, are not very rare, and may lead to serious confusion in discriminating from yellow fever. Ptyalism (Martialis, Morgan), orchitis and orchialgia (Mellis, Aitken, Martialis), cervical glandular enlargement, and other lymphadenopathies have been noticed (Christie, epidemic of Zanzibar, 1871, Martialis, Fouqué, Catholendy). The kidneys are rarely implicated.

Course, Duration, Complications.—Enough has been said in the section devoted to the clinical history and symptomatology to indicate approximately the course of the disease. The duration of dengue is exceedingly variable, or at least differently estimated whenever writers have attempted to calculate it. Thus, we find it four to six days (Ballot in Martinique, Morice in Cochin China); three to eight days (Thomas); from five to six days (Catholendy at Réunion); four to five days (Poggio and Vauvray); eight days (Fayrer); but it must be borne in mind that these figures are very far from giving a true idea of a fever which can be completely evolved within extremes of two or three days' minimum and persist, or leave its distinct impress on the patient, for weeks and even months (Mahé).

The convalescence is still more indefinite in its duration. Occasionally it is rapid and satisfactory, in robust children especially, as in the epidemic of Réunion or in that of 1860 in Martinique (Mahé). At Port Said, Vauvray tells us, the patients were up and about in thirty-six or forty-eight hours after the fever. *Per contra*, in other epidemics—and in this respect we may safely say that the majority of epidemics of dengue resemble one another—the convalescence is not completely effected in weeks, especially, as already stated, among the weak and infirm.

Among the sequelæ or relics of the disease, Manson and others have noticed crops of furuncles and abscesses and a general tendency to pus-formation in various parts of the body, which are also noticeable in other prostrating fevers, and which in the light of modern bacteriological research would properly be classed under the head of *secondary mixed infections*, the various staphylococci and streptococci of suppuration giving rise to the abscesses, the special micro-organism of dengue being supposed to be non-pyogenic.

Relapses.—The frequency of relapses is universally admitted as being one of the distinctive features of the clinical career of dengue. Poggio calls attention to this in his observations, and is astonished that one attack should protect so little from another. The frequency of these relapses was estimated at fifteen per cent. at Réunion in 1869 (Baret, Mahé). A distinction must be made in regard to the term "relapse" in connection with dengue. A relapse, properly speaking, is the repetition of an attack of the disease after *complete* recovery therefrom, and must not be mistaken for the exacerbations which follow frequently after the primitive remission and before convalescence is completed (Rochard). Relapses, even in this sense, are very common while epidemics are in progress: in fact, Thomas considers that once having had the disease makes a person more liable to it than before, at least during an epidemic. The same author cites instances of patients whom he treated for dengue in Savannah in 1880 who had had the disease in previous epidemics.

Prognosis.—Dengue almost invariably ends in recovery. In four epidemics out of eleven, notwithstanding the thousands of victims, not one person was recorded as having succumbed to the disease; in the other seven, but few deaths were noticed,—at furthest a total of five in the severest epidemics. In Gorée, Senegal, Thaly lost one case only out of one thousand patients; Mooden Sheriff states that in Madras dengue was sometimes fatal in adults from pericarditis and in children from convulsions; out of three thousand six hundred and forty-seven cases in India collected by this observer, twenty died, and the mortality was distributed as follows: adult males, seven, female, one, children, twelve,—showing the greater dànger to the latter class of sufferers. This experience with very young children is supported by the observations of epidemics in New Orleans and in the South generally, confirming in this respect the general law that the extremes of age are always more seriously exposed to the risks of disease. The proper prognostic elements which belong to this disease have not been yet sufficiently defined. Rey, quoted by Mahé and Rochard, states that the more precocious and confluent the eruption the more readily are the symptoms subdued and the career of the disease abbreviated. If, on the contrary, the eruption is discrete, disseminated, rare about the extremities and more abundant in the face, cheeks, and neck, the fever will be accompanied by greater malaise, anxiety, and cephalalgia.

Differential Diagnosis.—The positive or absolute diagnosis of dengue is exceedingly difficult to establish in the beginning of an epidemic, just as, on the other hand, it impresses itself at once on the profession and the laity, once the epidemic has been recognized and admitted. The sporadic cases which occasionally occur in places where dengue may be considered endemic offer particular difficulties if the special group of symptoms characterizing the disease are not well pronounced. Once, however, the attention of the attendant has been awakened, it is difficult to confound this affection with any other: how mistake the acute, sudden stiffening "break-bone" neuralgic pains for the inflammatory rheumatic pain, the fever so rapidly lighted, its eruption simple and double, the remission, the relapses, the non-albuminous urine, and finally the rapid spread and lack of fatality which always characterize it? Still, the differential diagnosis is difficult and at times impossible, especially when dealing, as already stated, first, with single, very mild, *atypical* cases; secondly, with very malignant and *rare* hemorrhagic cases in children; thirdly, when other allied infections, like yellow fever and malarial fever, are prevailing extensively and simultaneously with dengue. The differentiation between dengue and the two last-mentioned diseases is a matter of very great importance to Southern practitioners and all those who, living within the areas which are exposed to the visitations of the three poisons, especially at the commencement of epidemics, are made to appreciate the disastrous consequences of an error in diagnosis.

Differentiation between Yellow Fever and Dengue.—It must be admitted that dengue is a most frequent companion of yellow fever in its epidemic eruptions. There is great truth in the language of Porcher when he says,[1] "It is a significant fact that we have never been able to distinguish accurately between the two, to say of every case and at every stage of these two diseases, 'This is yellow fever, this only break-bone;' yet the extreme, well-marked examples of undeniable yellow fever were as different in every material respect from the lighter form of break-bone coexisting with it, as black is from white. . . . The one is a disease characterized at its inception always by high temperature, by the supervention of albuminuria, hemorrhages, black vomit, convulsions, bronzing of the skin, fatty degeneration of the liver, and often by death; the other light, fugitive, often almost ephemeral, only producing weakness and prostration, and never fatal. And yet they often shaded insensibly into each other and no distinct lines of demarcation could be drawn by any one. No one could give us a single diagnostic point. It was a mere question of plus and minus, the decision never being positive or based upon established scientific reasons. . . . The milder cases were called break-bone, and the severe yellow fever; yet we were in constant dread lest a case thought to be break-bone should prove to be yellow fever by an aggravation of its symptoms. Those who were what may be called very sick, who had high fever or some violent symptoms of some days' duration, who got well 'by the skin of their teeth,' or who died, enjoyed the uncertain, and, with regard to the latter, posthumous, credit of having had the real disease."

It cannot be said that Dr. Porcher has greatly exaggerated the situation in presenting the case as he has done, for when it comes to an absolute, positive, and unfailing test we have none. For this reason we have insisted so much upon the examination of the blood in all suspicious cases, in order that McLaughlin's promising microscopic test may be confirmed and made available.

---

[1] "On Dengue in South Carolina in 1880," Trans. Amer. Public Health Assoc., art. xxxviii.

Pending the confirmation of these observations, we must depend upon the clinical manifestations, which, when brought in contact with those of yellow fever, as has been so ably done by Holliday, will no doubt prove of great assistance.

Yellow Fever.	*Dengue.*
Single paroxysms.	Single paroxysms quite often. Two paroxysms with a remission between them.
Temperature rising regularly.	Temperature rising irregularly.
Duration, seventy-two hours.	Duration, three to five days.
Tongue, white centre, red edges, pointed; conjunctivæ very much congested.	Tongue broad, white, deeply indented by teeth, edges rarely very red.
Stomach irritable.	Nausea complained of.
Vomiting frequent.	Vomiting rare.
Violent pains in back and head; great jactitation; hebetude great; eruption rare.	Pains occurring early, much *more* severe; a general early appearance of eruption.
Jaundice appearing early; symptoms of nervous exhaustion evident and alarming.	Conjunctivæ rarely very red; præcordial tenderness on pressure rarely well marked; nervous exhaustion profound, though rarely alarming; jaundice never observed.
Secretions all suffering; urine scanty, often albuminous; suppression frequent.	Secretions natural; urine *usually* normal, sometimes and *exceptionally* traces of albumen.
Hemorrhages frequent and alarming, and *black vomit* an urgent symptom.	Hemorrhage *slight*, insignificant; if at all present, black vomit *very rare*.
Recovery exceptional.	Recovery the rule.

This comparison (slightly modified) was based not only upon a large personal experience, but also upon a careful analytical study of the opinions of over sixty physicians who had a long experience with both diseases in New Orleans and other parts of Louisiana.

Malaria and Dengue.—Dengue has been frequently mistaken for malarial remittent fever, especially in the earlier, more perplexing periods of dengue epidemics, before the type of the disease is fully recognized. Still, though there may with more or less frequency be remissions, relapses, and perhaps intermissions and occasional so-called critical (profuse) sweatings (not preceded by violent or regular chills), the *pains*, the absence of regularity in the accesses and of paroxysms, the absence of gastric, hepatic, and splenic complications, of glandular enlargements, of the malarial cachexia, sufficiently mark the distinction between break-bone fever and the malarial infections (Porcher). Finally, two other tests in cases of great doubt offer themselves to the physician: first, the examination of the blood, which can be readily applied at the bedside, and which, by revealing the presence or absence of the *plasmodium malariæ* of Marchiafava and Celli (distinctly confirmed and admitted by Sternberg, Osler, Councilman, and others), will decide whether the case is malarial or not; and, second, the therapeutic or *quinine* test: quinine has no influence whatever upon the career of pure dengue,—unless this be complicated with malaria, when quinine will clear the case of the malarial infection,—while it will certainly modify the clinical career of a pure malarial case.

Again, the arthralgic and myalgic pains of dengue have led to confusion of this disease with rheumatism, and even with gout (Morice), especially when there is some swelling and redness over the joints, as in the cases observed by Martialis, Sheriff, and various other observers. In our experience true evidences of arthritis, such as redness and swelling, are exceptional rather than usual phenomena in this disease. The eruption of dengue and the whole complexus of symptoms usually allow of a ready differentiation between the two diseases.

Finally, the eruption with fever has not infrequently caused dengue to be mistaken for scarlet fever, measles, or the early stage of small-pox; but the pains, the peculiarities of the dengue fever-chart, and the absence of sequelæ, as well as the subsequent career of the cases, will promptly dispel any doubts that may exist as to diagnosis.

The eruption of dengue has also led to confusion in the first days of the disease with simple erythema, febrile urticaria, erythema nodosum, and erysipelas; but it will usually suffice for the practitioner simply to bear in mind the characteristics of dengue and of these diseases to avoid any possibility of confusion.

Pathological Anatomy and Pathology.—It is self-evident that a disease so rarely fatal as dengue must of necessity possess a very scant store of anatomical data. Only three necropsies of pure cases (?) of dengue have been recorded (A. Hirsch), and in these the observations made were limited almost exclusively to the morbid appearances of the knee-joints, which were found hyperæmic. These observations are hardly worth mentioning. The few deaths that occur from this disease are usually traceable to complications, hyperpyrexia, and exhaustion. The pathological anatomy of dengue is, in reality, yet to be written.

The pathology of the disease is entirely speculative. Nothing definite is known, and what has been said in connection with the clinical history and in the way of interpreting the insignificance of the clinical phenomena is all that could be added here. That there is an infection that is admitted in the blood cannot be doubted; how or by what route it gains entrance into the economy is yet unknown. It is presumable that the poison after admission to the circulation acts primarily upon the nervous system, as the intense cephalalgia and neurotic manifestations would lead us to suppose. That the poison acts on the joints and muscles like that of rheumatism we doubt, because of the simple neuralgic and usually *non-inflammatory* character of the articular affection. The exceptions to this are too few to allow us to admit the analogy with the rheumatic poison. Still, the stiffness of the muscles remains, and this favors the rheumatic theory. The eruption with the inflammation and swelling of the lymphatic glands we are inclined to associate, with Schmidt,[1] with the final elimination of the infectious poison from the organism.

Treatment.—A self-limited disease, almost always ending in recovery, must needs rarely call for active therapeutic interference.

Though our acquaintance with dengue is comparatively recent, yet its treatment, like that of other great epidemic diseases, has gone through various phases of doctrinal therapeutics, all of which have left their imprint behind them. In the great Indian epidemic of 1824–26 (Twining, Mouat, Cawel, and others), and in the Cadiz epidemic of 1784 (C. Cubillas), the antiphlogistic method was given an ample opportunity to test its claims (large venous depletion, active and profuse emeto-catharsis). It is stated, however, that even at this early period British practitioners reserved the sanguineous depletions for special and well-marked indications.

One century afterwards, in the Teneriffe and Cadiz epidemics of 1867 and 1868, Poggio denounced venesection as pernicious in the treatment of dengue. He adopted the expectant method, much as it is practised to-day.

In the Indian epidemic of 1872 the British practice consisted largely in the administration of diaphoretics (acetate of ammonia), tincture of belladonna for the pains, iced drinks, cold sponging of the body, especially in hyperthermic cases, and stimulants. Quinine, in large doses, had gradually become fashionable, and a host of other more recent remedies were added to meet the symptoms.

It is a notable fact that, notwithstanding the frequent and almost radical changes in the methods of treatment, the mortality of dengue has remained unaffected; it has always been the same,—practically *nil*. In this respect it has been most benignly different from yellow fever, which has proved its contempt of therapeutics by its persistent malignity in spite of the "systems" and "doctrines" that have been brought to bear against it.

Notwithstanding our evident inability to jugulate or even seriously check the progress of the disease, there is unquestionably much that the physician can do to mitigate the sufferings of the patient and to comfort him generally in his transit through this most cruel ordeal. In fact, a careful observer like Mouat noticed in his earlier practice in India (1827) that the disease did appear to last longer and to present more serious features when left to follow its course undisturbed.

In the majority of tropical diseases an evacuant medication at the onset seems generally to be followed by good results, and experience has attested the fact that dengue is no exception to this general rule, all observers appearing to agree on this point. For this reason, it will be proper to begin the treatment of the first stage by administering an emetic of ipecac syrup, followed after the emesis by a laxative. In infants at the breast, aromatic syrup of rhubarb is a very generally administered and popular laxative; several large spoonfuls of prune tea sweetened with syrup of manna will also act efficiently in the same direction, and may be given to advantage to older children where the tea is combined with a few leaves of senna. Calomel has always been, of course, a most popular laxative with English-speaking practitioners, but it is very doubtful if it has any special advantages; if used at all, the more elegant and efficient modern triturates with sodium bicarbonate should be prescribed. Husband's and Henry's magnesia, cream of tartar in lemonade flavored with some pleasant and tart syrup (raspberry, strawberry, currant, etc.), will be found palatable, if iced, even by the most fastidious and difficult children. Iced citrate-of-magnesia lemonade is usually well taken by older children. After the laxative, a hot mustard foot-bath will prove most grateful to the patient, as it tends markedly to relieve the intense cephalalgia of the invasion. In infants and little children, the convulsive phenomena which occasionally characterize the invasion will tend to be shortened by a general mustard warm bath prepared according to Trousseau's recommendations, by simply immersing a small bagful of mustard-meal in a tub of hot water and pressing the bag in the water without mixing the meal with the water. Furthermore, potassium bromide is especially effective in diminishing the reflex excitability of children, and will prove more than usually effective in this condition. In cases of marked cerebral hyperæmia, and in adults or adolescents especially, one or two leeches to the mastoid process or a few small wet cups to the nape of the neck will greatly lighten and relieve the head-symptoms. Good judgment is necessary in the exhibition of this depletory treatment, which should always be reserved for sthenic and plethoric children and adults, who usually constitute the small minority of the population of warm climates. Cold applications to the head, iced in summer, with camphorated sedative water, bay rum, cologne, or any other pleasant evaporating preparation, will always be grateful, if not indispensable, to the patient. This wetting of the head should always be done in a thorough and efficient manner, parents and friends, in tropical countries particularly, being afraid, for some mysterious reason, of cold water in fever. For this reason, this method of cooling the head should be demonstrated to them by the physician himself: the little patient should be laid across the bed with the head projecting beyond the edge, allowing it to rest in a basin or bowl of water mixed with the evaporating lotion, ice being also added if the initial cephalalgia is intense; the head should be then gently but freely douched with the water; a little shampooing of the head aided by fanning will complete the process, and the patient will be made thereby infinitely more comfortable, no matter what his age. By this means alone the convulsive manifestations and agitation of many children will be lulled and averted.

---

[1] Article "Dengue," Pepper's System of Medicine, vol. i.

After these preliminaries, which are especially indicated in the earlier period of the invasion,—though the cooling of the head as above described will always be in order during the pyretic period,—the practitioner must prepare to combat more permanently the tendency to continued elevation of temperature and hyperthermia, as also the myalgic and especially arthritic pains which characterize the further course of the disease. In the first highly painful stage of the disease, I am satisfied that the remedies usually trusted in the more common pyrexiæ of children are not to be relied upon: aconite, with liquor ammonii acetatis and mistura potassii citratis, quinine, etc., all of which have been faithfully tried in this affection, are mere placebos, and amount to nothing in the way of securing positive relief to the sufferer. Here the improved antipyretic therapeutics of the present day finds an admirable field of application. Thomas (*loc. cit.*) says that in cases exhibiting the rheumatic type, particularly when the temperature runs high, sodium salicylate will be found to be very efficient, safe, and pleasant. This experience I can, with the majority of observers, personally confirm. But it cannot be doubted that even in this type of the disease sodium salicylate is inferior in both its antithermic and its analgesic properties to several of the synthetically prepared alkaloids of the aromatic series of the carbon compounds, of which kairin, thallin, antipyrin, and antifebrin are most prominent members. Of these there is no question, at present, as to the superiority, reliability, and safety of antipyrin and antifebrin, the former of which has been tried by De Brun in the epidemic of dengue at Beyrout, Syria, already referred to. Observations on the action of these antithermics in dengue are still insufficient and lacking to complete our thorough understanding of their comparative merits in this disease. But it appears to the writer that no matter which one of these two agents is employed, it will be found reliable and superior to the older febrifuges. Antipyrin and antifebrin act well with children, can be made palatable, especially the latter, and, when properly exhibited, seldom give rise to objectionable after-effects. Argutinsky recommends the following minimal doses of antipyrin: for children under one year of age three grains thrice daily, at intervals of three hours; for those of from one to three years, five grains; for children between this age and five years, from five to six grains three times, at intervals of two hours; for children of from six to eight years, from eight to ten grains daily, at intervals of two hours; and for children of from ten to twelve years, from ten to twenty grains thrice daily, at intervals of one hour. The dose of thallin is about one-fourth of that of antipyrin, but it is a dangerous remedy and is rapidly being forgotten. Antifebrin affords advantages in the opinion of most observers (Beaumetz, Huchard, Sée, etc.) over any of these agents. The dose is one-half that of antipyrin, though its full action is produced more slowly. The writer prefers to use a weak emulsion of antifebrin in syrup of acacia (gr. i–ʒi) when dealing with small children, and administering the remedy at frequent intervals (half an hour), carefully watching the effect with the thermometer: this is certainly the safest and most effective plan when dealing with children of unknown idiosyncrasies. It should be remembered that these agents are also active when administered by enema.

Notwithstanding the worthlessness of quinine in combating the pure dengue poison it will be prudent to administer it as a rule in malarious localities, where the marsh-poison is constantly seeking opportunities to manifest itself. If antifebrin and antipyrin fail in their analgesic effect, chloral will be found to be a valuable agent to soothe the pain and to procure rest. Brun has obtained better results with it than with opium or morphine: still, in some cases the latter preparations will have to be appealed to, especially in grown children, to relieve the pain, as under other circumstances opium and its preparations should be administered cautiously to children. In grown children or in adults some benefit may be obtained in relieving the insomnia and agitation by administering at night the new hypnotics sulphonal (adult dose, gr. x–xxx) or paraldehyde (adult dose, ʒss–ʒi).

Liniments are prescribed often, but they do not appear to add to the comfort of the patient, or, if of benefit, are so only by impressing the mind: camphorated soap liniment, chloroform liniment, aconite, etc., will be found sufficient. Digitalis is expressly indicated in very depressed conditions and in those in which the heart appears to suffer. In very tedious cases the proper nutrition of the patient is a matter of great importance, especially in children and old subjects.

The eruptions call for little interference: when pruritus becomes excessive during the period of desquamation, German green soap, corrosive sublimate soap (Bergmann's formula), carbolic acid solution, one or two per cent., or a salve of vaseline and hydronaphthol, may be tried with expectation of relief. Aitken advised the application of an emulsion of sweet almonds containing ammonium hydrochlorate. A salve containing cocaine hydrochlorate, three or four per cent., will be found available, if recently-reported experience with it in this field is to be trusted.

Convalescence requires the greatest care. A reconstructive and analeptic treatment, and generous diet, are here called for. The preparations of iron and cinchona are here as well indicated as in most of the prostrating diseases of tropical climates. The persistent anorexia is a feature peculiar to the convalescence of dengue, and it may prove very difficult to overcome it. Tincture of gentian, bitter infusions, or small fragments of rhubarb-root, immediately before meals, appear to give the best results (Rochard). H. Rey has found arsenic especially valuable. Nux vomica, in the form of tincture, in progressively increasing doses, according to the plan recommended by Musser, of Philadelphia, would find here an excellent field of application. Cod-liver oil in strumous children, and massage in all ages, will prove especially useful. Finally, we would advise, with H. Rey and Rochard, Fayrer, and other experienced tropical practitioners, the use of cold baths or douches as among the best means to return to the economy its wonted energy. If all means fail to return health to a prostrated dengue patient, the last resort of tropical therapeutics must be appealed to,—*i.e.*, a change of air, sea-voyage, and change of residence to a more temperate or cold climate.

CHOLERA INFECTIOSA.

BY E. O. SHAKESPEARE, M.D.

Definition.—Cholera Infectiosa or Asiatica is an infectious disease of a specific character, due to a specific agent which primarily attacks the intestinal canal. This agent is transportable from place to place, and is endowed with the power of rapid multiplication, both within and without the human organism, under favorable circumstances.

This agent is in all probability a vegetable parasite,—namely, the comma bacillus of Koch. In the process of development and growth of this parasite, both within and without the human body, a specific poison or *ptomaine*—a chemical alkaloid possessing specific chemical and physical properties—is produced; the primary action in the human system is upon the mucous membrane of the intestinal canal, chiefly the small intestine, and the ultimate result is the desquamation and destruction of the epithelial elements.

This poison elaborated in the intestines is absorbed, produces an inflammatory irritation of the tissues immediately underlying the epithelia, and finally enters the blood-circulation. In the blood it attacks the red corpuscles, causing destruction of some and alteration of the function of many, and it causes great disturbances of the nervous system, the gravity of its effects falling principally upon the vaso-motor and respiratory centres.

The infecting agent exists in the intestinal contents of those suffering from the disease, and is discharged with the alvine evacuations, and sometimes also with the ejections from the stomach. Both dejecta and ejecta, hence, contain the infectious principle, and under favorable circumstances are capable of conveying the disease, either directly or indirectly, from the sick to the healthy. *Cholera Asiatica is, therefore, an infectious disease, and is capable of being conveyed from person to person and from place to place, and under favorable circumstances of becoming epidemic.* It should, consequently, be properly named *cholera infectiosa.*

Means and Manner of Infection.—Since the infectious agent exists in the evacuations both from the stomach and from the anus, various materials become capable of conveying the infection of this disease: such as clothing soiled with this matter; hands fouled with it; articles of food and drink which have been contaminated with it. It is by means of soiled clothing and personal effects upon which this agent is preserved in a more or less moist condition that the infectious principle is conveyed long distances both by land and by sea. The contamination of watercourses and small streams by vomit or dejecta is perhaps the most frequent and certainly the most rapid means of producing a sudden and widely-extended outbreak of cholera infectiosa. The watercourses are not infrequently also contaminated by washing therein the personal effects of cholera patients.

Regarding the comma bacillus of Koch as the infectious agent, it has been established by numerous and exact experiments that this microbe is not only able to live for a considerable length of time in water, but is even capable of enormous multiplication therein, especially if the water contain a certain amount of organic or vegetable material. The use of such contaminated water for drinking, bathing, and culinary purposes is perhaps the most frequent mode of introduction into the human organism of the contagious principle of cholera infectiosa.

The universal practice of the watering of milk also renders this article exceedingly and especially dangerous to children during periods of the prevalence of cholera; and, where extensive and sudden local outbreaks of the disease cannot be attributed directly to the use of contaminated water, it is generally the milk which conveys the cause of infection. Other articles of food are in a far less degree liable to contamination, but there are numerous examples of infection occasioned by thoughtless or accidental contamination of vegetables, fruits, and other nutritive material. Experience has abundantly proved two laws which have an important bearing upon the spread of cholera. The tendency to infection varies exceedingly among individuals, and is with the vast majority exceedingly small. Disturbed conditions of the digestive apparatus greatly increase the susceptibility of an individual and render him far more liable to an attack after exposure to the infection. It is exceedingly improbable that the infectious principle is ever conveyed to the healthy by the medium of the air: it is certainly never transported to any considerable distance in this manner. It is very doubtful, if even possible, that infection may take place through the lungs. It is certain that it cannot be effected by cutaneous absorption. The disease, therefore, cannot be properly termed truly contagious in the common use of that word. It is extremely doubtful if there be a single well-authenticated case upon record where the disease has been conveyed in any other manner than by the introduction of the infectious principle into the stomach.

Still, regarding the comma bacillus of Koch as the infecting agent, it has been abundantly proved that the normal acid juices of the stomach are capable of destroying it. It is, therefore, not surprising that the examples are multiplied where water and other ingesta known to be infected have been swallowed, intentionally or accidentally, by healthy persons, without harm. If, however, this living infectious principle, the comma bacilli of Koch, escape beyond the pylorus and pass into the small intestine, the contents of which have an alkaline reaction, multiplication with enormous rapidity therein, elaboration in considerable quantity of the poisonous ptomaine, and the establishment of the disease which we recognize as cholera infectiosa, become possible.

Prophylaxis.—The considerations already advanced suggest more or less reliable prophylactic measures. If the stomach be properly guarded against the introduction of the living infecting principle, the individual will be necessarily protected against the danger of an attack. Protective measures may be considered from two stand-points: first, with regard to the person suffering from an attack of the disease; second, with regard to the healthy individual exposed to infection.

A. With regard to the person suffering from an attack of the disease: The evacuations from the stomach and the bowels should be immediately disinfected; where this is thoroughly accomplished, it is impossible for the infection to spread beyond the attacked. The dejecta and the vomited matter should be passed into a vessel containing a quart or more of a strong solution of carbolic acid, one part to twenty of water; and immediately after the evacuation a sufficient amount of the disinfectant should be added to make the whole quantity equal to the bulk of the evacuated material; the whole should then be gently stirred, and afterwards allowed to stand for fifteen to twenty minutes, when it should be removed and emptied into a pit containing unslacked lime, and be immediately covered by a quantity of the same material. If circumstances render it impossible thus to dispose of the disinfected evacuations, they should be emptied into a large earthen vessel containing a quantity equal to their bulk of a solution of bichloride of mercury, one part to a thousand, and stirred thoroughly therein; after remaining there for an hour or more, they may be emptied into a drain which leads to the sewer. The clothing of the patient, as well as the soiled bed-linen, immediately after removal should be disinfected by thoroughly soaking for an hour or more in a large quantity, more than sufficient to cover them, of a strong solution of carbolic acid, one part to twenty; or they should be immediately subjected to the prolonged action of boiling water or steam. The anus, hands, and mouth of the patient should also, immediately after an evacuation, be washed with a disinfectant, —in this case, however, weaker than above indicated, say one part to ten thousand of bichloride of mercury and water, for the anus and hands, and for the mouth water slightly acidulated with sulphuric acid. The hands of the attendants, also, should be washed with the same weak solution of bichloride of mercury after handling the patient. Under no circumstances should the attendant, or any one else, eat in the same room with the sick; and, as an invariable rule which should be scrupulously observed, no person who has been in direct contact with the sick or with any of his personal effects should eat without first thoroughly cleansing and disinfecting the hands.

B. With regard to the healthy person exposed to the infectious principle of the disease: Remembering what has already been remarked concerning an increased susceptibility to infection by reason of disturbance of the digestive apparatus, it is strenuously insisted upon that all causes, of whatever nature, of disturbance of the functions of the stomach and intestines, should be studiously avoided: such as intemperance of all kinds, either in drinking or in eating; all irregularities of personal habits, either as to time of meals, occupation, exercise, or hours of sleep; all emotional excitements should be removed; in short, every circumstance which experience has shown may exercise a disturbing influence upon these important functions should be carefully guarded against; the use of articles of food which are liable to occasion indigestion, or to cause an unusual or unhealthy activity of the digestive apparatus, should be interdicted; the child should be carefully prevented from indulging in exhausting sport or exercise, and should be carefully shielded against intemperate weather; it is all-important that the functions of the skin should be kept regular and active by a sufficient amount of seasonable clothing by day and by night; particular care should be taken that revulsions of blood, produced by chills, from the cutaneous surface to the internal organs, especially the abdominal, may not occur, and in this connection it is strongly recommended that the abdomen be enveloped at night by a broad band of flannel, in order that during the restlessness of the little one in sleep the skin of the abdomen may not be exposed to the direct action of the air; cold baths should be avoided; the surface of the body should be washed at not too frequent intervals, by sponging with tepid water, and afterwards dried thoroughly by vigorous rubbing with a rough towel; meanwhile, the body should be protected from draughts. Irregularity and intemperance in eating and drinking have already been alluded to. It is important that the little one be prevented from imbibition of large quantities of water or other fluids at intervals between meals, for, if there were no other reason, it is a well-known physiological fact that in the intervals of digestion the reaction of the gastric juices is neutral and sometimes even slightly alkaline. If contaminated water or milk should be swallowed in large quantity during this interval, it is clear that the probability of the living infecting agent passing through the pylorus into the small intestine is greatly increased, and the possibility of an attack much enhanced. In a house where a cholera patient is suffering, the children should be kept out of the sick-room. But if, as often occurs among the class of people who are mostly the sufferers from cholera,—the poor and the squalid,—there be only one common room for the use of the family, the child should on no account be permitted to occupy the same bed as the sick, and should during the day, as also during the night, be kept as much as possible from contact with the sick-bed.

Attention to the preparation of food is a matter of extreme importance to all persons exposed to the infection of cholera, and especially to children. It goes without saying, that the materials consumed should be perfectly fresh and sound in every respect, and that the water and milk employed should be absolutely free from the living infecting principle, as well as pure and healthy. As a guarantee against the possibility of infection by means of the water or milk, both should be thoroughly boiled before use, and, as it is possible for the cholera-microbe to multiply rapidly both in water and in milk, these articles should not be used except after very *recent* boiling. Coffee and tea should be *recently* made and *served hot.* All food should be *thoroughly* and *recently* cooked. No raw food of any description, except possibly a moderate quantity of perfectly fresh, ripe, and absolutely clean fruit, should be eaten. *Salads* and other such articles should be *interdicted.* Bread, as well as butter, should be carefully protected against the possibility of contamination. The culinary utensils and table-ware should be *scrupulously cleaned* with *boiling water.*

The hygienic condition of the dwelling and its surroundings should be made as perfect as possible. All decaying animal or vegetable matter should be removed. The house-drains should be free and clean and flushed with a sufficient amount of water at intervals, followed by the emptying therein of a liberal quantity of strong solution of copperas in water, or of a five-per-cent. solution of carbolic acid. The cess-pits and the privies should be kept clean and free from odor by the use of unslacked lime, large quantities of copperas, or other similar inexpensive materials. The supply for *drinking-water* should be *scrupulously guarded from possible contamination of any kind.*

Among the precautions to be enforced against a threatened attack of cholera infectiosa in any one, but especially in the young, one of exceeding importance is watchfulness over the condition of the alimentary canal. In a large number, perhaps the majority, of instances, an attack of cholera is preceded some hours or days by derangements of the digestive apparatus, such as distress or a sense of fulness or heaviness in the stomach, of gastralgia or nausea, or of occasional vomiting; or the disorders may be limited to the intestines only, and be manifested by vague general abdominal uneasiness, or slight fleeting pains, or active peristaltic movements which can be seen or felt through the abdominal walls; and all or any of these may be associated or end with diarrhœa, and sometimes with tendency to disproportionate prostration; or, again, the disorders of the stomach and intestines may be combined.

If these disturbances of the alimentary tract are promptly discovered and remedied, many an attack of cholera will be thereby avoided. In such cases, absolute rest in bed, and, if possible, also total abstinence for a day or two from food, should be enjoined; if there be reason to infer the presence in the stomach of undigested food, a single emetic dose of ipecac should be administered; or if there be visible peristaltic movements of the intestines, or diarrhœa, these should be controlled respectively by small doses of opium in a convenient form, and of such drugs as salol, naphthalin, salicylate of bismuth, or analogous compounds.

What has been thus far said applies especially to individuals; but, unfortunately, in this disease public interests and relations must also be regarded, and from this stand-point, *so long as there are in the locality only a few scattered cases of the disease*, the utmost efforts should be made to prevent the establishment of an epidemic.

The presence of the comma bacilli of Koch in the alvine evacuations or in the vomited material from a suspicious case once determined, the duty of the attending physician and of the health officer becomes plain. The safety of the other inmates of the dwelling, and, what is of infinitely greater importance, that of the general community as regards public health and commercial interests, demand that the most skilful and intelligent physicians and nurses be procured for the sick and *kept in constant attendance.* If the dwelling be a hovel of the poor, as is usually the case, the inmates should be removed without delay to clean, healthful, and commodious quarters; if the attacked is already in a desperate condition, where every prolonged disturbance increases the probability of a fatal termination, he should not be moved.

Inasmuch as the safety not only of the health and trade of the population of the locality involved and its immediate surroundings, but also of those of great states and sometimes nations in communication with it, is seriously threatened by the escape and spread of the infectious principle elaborated in and discharged from the intestines of the person suffering an attack of cholera infectiosa, skilful and constant attendance is imperatively called for, and should be provided at the public expense. Furthermore, that same public has a paramount interest and absolute right to be *assured* that every rational precaution against the spread and dissemination of the infectious agent is scrupulously and conscientiously enforced. This is tantamount to saying that the care of the attacked and of the dwelling, as well as the custody or close surveillance of all persons associated or in communication with him or it, should be under the strict control and direction of the jeopardized public *through its own intelligent and responsible agents.* The prevention of the spread of infection—of such enormous importance to the public—should under no circumstances whatever be trusted in any degree to the ignorance or carelessness or conflicting interests of the inmates of the infected dwelling; neither should it be left to the chance of uncertainty through the incompetence or neglect or whimsical notions or personal interests or lack of authority of the private physician. Of course this means temporary invasion of the private rights and restriction of the personal liberty of a few individuals, whose own security is thereby enhanced. But the public safety demands a temporary sacrifice of private rights under these circumstances, and there should be no hesitation or vacillation in requiring it.

It is obvious that the evacuations of the intestinal canal of the attacked should, without loss of time, be carefully disinfected. But by no means all suffering an attack of cholera infectiosa are, especially in the earlier stages of the disease, so ill that they cannot be out of bed, and even out of doors engaged in their ordinary vocations. Yet experience has abundantly proved that those suffering "a walking attack" carry in their intestinal canal the infectious agent of cholera, and are capable, under favoring circumstances, of establishing a centre of infection wherever in their movements they may chance to void those intestinal contents. Hence the necessity of temporarily restricting the liberty of all inmates of the infected dwelling and of all persons in close communication with it, whether at the time of infection they are evidently suffering or not. All such persons should be isolated and kept under strict surveillance until the extreme limit of the period of incubation (say, five days) has fully elapsed, *counting from the commencement of the surveillance.* If during these five days no sign of even a slight or "walking attack" has made its appearance, and finally if a culture-test, as hereafter described, of the fæces has indicated the absence of the comma bacilli of Koch, the individual temporarily restrained of his liberty both for his own benefit and for that of the public may be without danger restored again to the full enjoyment thereof.

As suggestions for the practical application of public measures of prevention, we incorporate here an abstract, which we communicated to the *Medical News*, October 15, 1887, of the section on Prevention comprised in our Official Report of Cholera in Europe and India, 1884 to 1886:

"Measures of prevention, to give the greatest possible guarantee of success in extinguishing an incipient epidemic of cholera, should, in the first place, be based upon the most exact knowledge we possess of the cause, mode of attack, and manner of spread of the disease; and, in the second place, these measures should be intelligently, thoroughly, and rigidly enforced.

"What are the considerations involved in the first category? Probably nine-tenths of intelligent and experienced physicians all over the world, even including those of India, have for years admitted that there is most convincing proof that the active cause of the disease is a specific, material, living entity, of extremely minute size, endowed with the power of self-propagation and of exceedingly rapid multiplication in enormous numbers; that among animals it naturally attacks man alone, assailing him only by way of the intestinal canal; that the evacuations from the bowels contain the active cause of the disease, and that when this agent in any manner—as through drinking-water, milk, food, or the handling or washing of contaminated personal effects, etc.—reaches the intestines of another susceptible person, the disease may be thereby transmitted from the sick to the healthy; that the active agent exists in the dejecta of the lightest and most imperceptible no less than the severest and most deadly forms of the disease, and is known to be transportable from place to place through the movements of man and his personal effects.

"Proceeding from this basis, logical deduction and common experience alike demonstrate the absolute necessity and efficiency of such measures of prevention as the following:

"*a.* Speedy recognition and isolation of the sick; their proper treatment; absolute and rapid destruction of the infectious agent of the disease not only in the dejecta and vomit, but also in clothing, bedding, and in or upon whatever else it finds a resting-place.

"*b.* The convalescents should remain isolated from the healthy so long as their stools possibly contain any of the infecting agent; before mingling again with the well they should be immersed in a disinfecting bath, and afterward be clothed from the skin outward with perfectly clean vestments which cannot possibly contain any of the infectious material.

"*c.* The dead should be well wrapped in cloth thoroughly saturated in a solution of corrosive sublimate, one to five hundred, and without delay, cortége, or lengthy ceremonial, buried near the place of death in a deep grave, remote as possible from water which may under any circumstances be used for drinking, washing, culinary, or other domestic purposes. (Cremation, of course, is by far the safest way of disposing of cholera cadavers.)

"*d.* Those handling the sick or the dead should be careful to disinfect their hands and soiled clothing at once, and especially before touching articles of food, or drinking or culinary vessels.

"*e.* In the case of maritime quarantine, the well should be disembarked and placed under observation in quarters spacious enough to avoid crowding, and so well appointed and furnished that none will suffer real hardships.

"*f.* Once having reached the station, those under observation should be separated in groups of not more than twelve to twenty-four, and the various groups should under no pretext intermingle; the quarters for each group should afford stationary lavatories and water-closets in perfect working condition, adequate to the needs of the individuals constituting the group, and supplied with proper means of disinfection; there should be a bed raised above the floor, proper coverings, and a chair, for each member of the group, each person being required to use only his own bed; there should be a common table of sufficient size to seat around it all the members of the group, who should be served their meals from a central kitchen, and with table-furniture belonging to the station and cleaned by the common kitchen scullions.

"*g.* Drinking-water, free from possible contamination and of the best quality, should be distributed in the quarters of each group as it is needed, and in such a manner that it is received in drinking-cups only; there should be no water-buckets or other large vessels in which handkerchiefs, small vestments, children's diapers, etc., can be washed by the members of any group.

"*h.* Immediately after being separated into groups in their respective quarters, every person under observation should be obliged to strip and get into a bath (a disinfecting one is preferable), and afterward be clothed with fresh, clean vestments from the skin outward. Every article of clothing previously worn should be taken away and properly disinfected.

"*i.* Then all the personal effects should be at once removed to a separate building, washed,—if possible,—and thoroughly disinfected, or, if necessary, destroyed. After disinfection they should be temporarily returned to the members of groups when occasion requires a further change of clothing.

"*k*. Under no circumstances whatever should washing of clothing by those under observation be permitted. All used clothing should be first thoroughly disinfected (by boiling, when possible), and then should be cleansed, the disinfection and washing being done by a sufficiently trained and absolutely reliable corps of employees supplied with adequate appliances.

"*l*. All those under observation should be mustered in their own quarters and be subjected to a close medical inspection, *while on their feet*, at least twice every day, in order to discover and isolate as soon as possible new cases which may develop; and of course the clothing and bedding of these new cases should be treated without delay in the manner already mentioned. In the mean time, a watch should be set over the water-closets, for the purpose of discovering cases of diarrhœa, and, when discovered, such cases should be temporarily separated from the rest; they should receive judicious medical attention at once, and precautions should be taken as if they were undoubted, but mild, cases of cholera.

"*m*. The quarters should be kept thoroughly clean, and every surface upon which infectious material could possibly be deposited, including the floors, should be washed with a strong disinfectant twice daily, and oftener when necessary; evacuations from the bowels should be passed into a strong disinfectant; the hopper of the closet should be then flushed, and finally drenched with a quantity of the same disinfectant.

"*n*. For the proper attention to the sick, there should be two or more competent and experienced physicians, assisted by a sufficient corps of intelligent and efficient nurses, with hours of duty so arranged that a physician with a sufficient number of nurses shall be in constant attendance in the wards of the hospital.

"*o*. For the prompt recognition and separation of new cases, their temporary medical attention, the proper treatment of discovered cases of diarrhœa or cholerine, and of other maladies, and the immediate correction of every insanitary practice or condition by constant, vigilant, and intelligent supervision, there should be at least two or more competent and experienced physicians, with hours of service so arranged that a physician is on duty night and day among those under observation; and he should have subject to his orders, at any and every moment, a sufficient and efficient corps of nurses and laborers to carry out properly and promptly his directions.

"*p*. In order to prevent the intermingling of the various groups, to enforce obedience and order, and to make it absolutely impossible for the quarantined and their personal effects to have any communication with the exterior, a well-organized and sufficiently large police-corps should patrol the borders of the stations and the buildings day and night.

"*q*. Any group among whom there have developed no new cases of cholera, or of choleraic diarrhœa, during the preceding eight or ten days, may be regarded as harmless, and allowed to leave quarantine after each one is finally immersed in a disinfecting bath and reclothed with clean garments from the skin outward, the garments removed being destroyed, or thoroughly disinfected and cleansed as above indicated.

"As yet, no reference has been made to the crew, ship, and cargo. What has been said of the treatment of those under observation applies to every one of the ship's inhabitants. The observation, isolation, and cleansing of the crew and their effects could safely be performed aboard ship if necessary. The ship should be thoroughly cleansed and disinfected, particular attention being given to the quarters of the emigrants and crew."

The discovery of the comma bacillus of cholera infectiosa by Koch in 1883 has rendered it possible, for the first time in the history of epidemics of this disease, for the physician and the health officer to be absolutely assured of the infectious nature of suspicious cases. It is almost the universal experience of those whose duty it is to deal with the first appearance of this disease in a locality, that there is great doubt and contention concerning its real nature; and, as a rule, it is only after these doubts have been settled, unfortunately, by the starting and the spreading of an epidemic, that the authorities become convinced of the necessity of enforcing efficient restrictive measures. It is, however, then too late to protect the community from the misfortunes which follow in the track of this devastating scourge. It is only in the judicious handling of *the first suspected cases*, as a rule, that public preventive measures can be enforced with much hope of satisfactory results. Commonly, the course of the epidemic passes beyond control of the authorities after it has once commenced actively to spread; at least its effective management is then exceedingly difficult.

The discovery of Koch—since amply confirmed in all parts of the world by competent observers—that in the presence of the comma bacillus of cholera is furnished a practicable, rapid, and reliable means of the certain diagnosis of epidemic or infectious cholera, has its greatest value for the protection of the public health just at this point. The innumerable observations which have been made since that announcement have positively shown that, even if this microbe may not be actually the exciting cause of the disease, it is certainly its invariable accompaniment, and has never yet been found associated with any other disease. It may therefore be regarded as absolutely certain that whenever the comma bacillus of Koch is recognized in the evacuations from the bowels or from the mouth of any person, we have to do with a case of cholera infectiosa, and the most thorough measures to prevent the spread of infection are imperatively called for.

It is not necessary in an article of the scope and purpose of this to relate in any detail the history of this discovery and of the observations which have subsequently been made concerning it. We may state in this connection that for ourselves we not only regard the presence of the comma bacillus as an absolute proof of the existence of the infectious principle which causes cholera infectiosa, but are also strongly constrained to recognize in this microbe the active living principle which constitutes the cause of infection as well, although we freely admit that the proof of the latter is as yet by no means so strong and unanswerable as that of the former.

Morphology of the Cholera-Microbe.—The comma bacillus of Koch should perhaps be more properly classed as a spirillum than as a bacillus. Koch himself now is inclined to this opinion, and at present places this micro-organism between the bacilli and the spirilli, as partaking of some of the peculiarities of both. In the human organism this microbe is limited to the digestive tract. It exists in enormous numbers in the intestinal contents in the early stages of cholera, and is especially numerous in cases of extremely rapid development, such as the *foudroyante.* The numbers gradually decrease with the duration of the disease, until they finally disappear, having already become rare when the dejecta begin to present again their natural color and consistence. They swarm in myriads in the serous fluid of the intestinal contents, but are far more numerous in the desquamated flakes of the intestinal epithelium, those bodies which give to the discharges a *rice-water* aspect. They furthermore have been found in some numbers in the lumen of the follicles of Lieberkühn; they have sometimes been found below the epithelia, between it and the subjacent basement-membrane, especially in the Lieberkühnian follicles; and occasionally they have been found to penetrate to some distance into the surrounding connective tissue of the intestinal mucosa. It is doubtful if in the human being they ever reach the circulating blood or extend as far along the lymphatic tracts as the location of the lymph-glands, and they have never been found in the lacteals: so that in the human subject the habitat of this micro-organism may be said to be limited strictly to the innermost portion of the intestinal canal.

It occasionally happens that the intestinal contents, especially in extremely rapid cases of cholera, contain almost exclusively myriads of the comma bacilli, as shown by microscopic examination. But in the vast majority of cases these micro-organisms are intermixed with various other species of bacteria in considerable numbers, and often the other bacteria are so numerous in proportion to the comma bacilli that it is difficult to find more than two or three of the latter in a given field of the microscope.

Photo. No. 4 of the preceding article on "Outlines of Practical Bacteriology,"[1] by the author, is a photo-micrograph of the intestinal contents of a case of cholera of the latter category. By reference to it, it is seen that there are not more than two comma bacilli present in the microscopic field represented, whilst the other bacteria are far more numerous. In those cases where the comma bacilli exist in the intestinal contents in nearly pure culture and but little intermixed with other bacteria, microscopic examination alone is sufficient to base a diagnosis upon. But in such cases as those represented by the photo-micrograph above mentioned, microscopic examination alone can easily mislead. The very few curved bacilli found may possibly not be those of the comma bacilli of Koch, since there are numerous other curved bacilli which more or less closely resemble morphologically those of cholera infectiosa. In these cases, and in fact in the vast majority of instances, for the purpose of diagnosis it is safe only to resort to culture methods for the certain recognition of the comma bacilli of Koch, and the procedure is then as follows. A series of three test-tubes containing neutral or slightly alkaline sterilized flesh-peptone-gelatin are inoculated with a minute quantity of the intestinal contents as evacuated from the bowels, and plate-cultures are made from each after the manner described in detail in the preceding article above mentioned. After twenty-four or forty-eight hours it is usually found that the colonies developing from the micro-organisms contained in the intestinal contents are almost all those of the comma bacilli of Koch. For some reason, not well determined, it is found that under such circumstances few other colonies will develop in the gelatin, so that, notwithstanding the abundant intermixture of various other bacteria in the intestinal contents itself, the gelatin shows almost a pure culture of the comma bacilli. This was, in fact, the case with the intestinal contents represented in Photo. No. 4 above referred to. After twenty-four or forty-eight hours, a microscopic examination under a low power, say thirty diameters, of the gelatin plate, shows the comma-bacilli colonies presenting the appearance represented in Photo. No. 7, which is a reproduction of a photo-micrograph of a colony in a gelatin plate made from the intestinal contents represented in Photo. No. 4. By reference to Photo. No. 7 the following peculiarities of the aspect of the comma-bacillus colony will be noted. There is a rough granular centre of a gray or brownish-yellow aspect, having the appearance of a collection of highly refracting, finely-broken glass, surrounded by a clear circular zone in which are a few scattered, dark, refracting granules. This colony really rests at the centre of the bottom of a saucer-like cavity depressed below the general surface of the surrounding solid gelatin, and containing clear fluid. In their development the colonies of comma bacilli have the faculty of liquefying the surrounding gelatin. It is necessary to wait until the development of the colony has sufficiently far advanced to produce this liquefying effect in order to be sure that one has under observation a genuine colony of the comma bacilli of Koch. For in the earliest appearance of the cholera colonies the surrounding ring of fluid is so narrow as to escape observation, and there are bacteria which, in their development in flesh-peptone-gelatin plate-culture, present a granular aspect so nearly resembling that of the central portion of this comma-bacillus colony as to make it impossible, or at least exceedingly difficult, to distinguish between them. Photo. No. 6 represents a colony of a micrococcus sometimes found in the air; the aspect of this colony is like broken glass also. This latter colony, however, as may be seen, is more or less regularly circular in outline, and by reference to Photo. No. 7 it is observed that the outline of the comma-bacillus colony is quite irregular; in fact, it is often found to be much more irregular than here seen. In the earlier stages of development of the comma-bacillus colonies, however, their outlines are more or less regularly circular, and they are then difficult to differentiate. It is necessary to proceed a step further in the examination of the comma-bacillus colony in the plate-culture before becoming assured of its character. A microscopic examination of the individual bacilli constituting the colony must be made, and it is well in addition to make an inoculation from such an individual colony into a test-tube containing solid flesh-peptone-gelatin in the manner described in the preceding article before mentioned. After twenty-four or forty-eight hours, the puncture made by the inoculating needle shows along its track a growth of bacteria more or less advanced, according to the length of time which has elapsed since the inoculation, and according to the temperature to which the test-tube has been subsequently exposed.

---

[1] See page 147, *ante.*

Photo. No. 10 is a photograph, natural size, of such an inoculated gelatin-tube, forty-eight hours after inoculation. The surface of the solid gelatin within the tube is seen to be inclined. The culture now presents quite a characteristic aspect. The upper portion of the culture is more or less funnel-shaped, with the appearance of an air-bubble at the top. The fluid contained in the funnel-shaped cavity beneath the apparent air-bubble (which latter in reality is an optical delusion, the portion of the funnel represented by the air-bubble being only an empty cavity containing air) is clear or slightly opalescent. At the bottom of the funnel and along the narrow neck below is deposited a grayish, finely-granular mass, consisting of the subsided bacilli. Microscopic examination under a high power of the individual bacilli constituting the colony of Photo. No. 7 is made in the manner indicated in the preceding article. If the microscopic examination both of the plate-colony and of the material of the gelatin-test-tube culture shows only curved bacilli, it may at once be inferred that one has to do with a case of genuine cholera infectiosa. In order to be absolutely certain of this, however, at the same time that the gelatin-tube culture is made from the plate-colony, the surface of a potato prepared after the manner indicated in the preceding article mentioned should also be inoculated from the same colony, and the potato should be placed for twenty-four hours in the culture-oven. A growth consisting of curved bacilli upon the potato is positive proof that the bacilli are none other than those of cholera infectiosa.

Photo. No. 13 is a photo-micrograph of a cover-glass preparation made from the colony represented in Photo. No. 7, which was obtained from the intestinal contents represented in Photo. No. 4. By reference to Photo. No. 13 it will be seen that we have a pure culture of curved bacilli. The forms here shown are in the main those of a simple curve in one direction; but there are numerous instances of a figure S, caused by the juxtaposition or attachment end to end of two bacilli curved in opposite directions. Under cultivation the comma bacillus of Koch is not always found presenting exactly the morphology here shown. Growths in certain media, especially those not most favorable for the development of the bacillus, show more or less numerous threads of a more or less spiral form. These spirilli may be more or less jointed and consist of a spiral chain of a number of individual curved bacilli joined end to end more or less closely, or they may present the appearance of a continuous curved or spiral filament without interruption. The spiral forms which often develop in cultures of the comma bacillus, and, in fact, of all other curved bacilli heretofore cultivated artificially, are well represented in Photo. No. 12, which is a photo-micrograph of a cover-glass preparation from a pure culture of the curved bacillus of Finkler (so called, of *cholera nostras*). Photo. No. 11 also shows a few examples of short spirilli in a pure culture of the Deneke or cheese bacillus. Comparison of these photographs with that reproduced in Photo. No. 13 also shows the morphological resemblance of the individual curved bacilli of Koch, Finkler, and Deneke. Other curved bacilli—such as those of the mouth, one form of which was isolated and cultivated by Miller, those sometimes found in intestinal contents in cases of dysentery, those frequently found in leucorrhœal discharges, those occasionally found in scrapings from pneumonic lungs, those often found in ordinary running water and in numerous other places—also so closely resemble the comma bacillus of Koch morphologically that it is impossible, or at least unsafe, to undertake to distinguish between them, by means of the microscope alone, for the purpose of recognition, except under the circumstances above mentioned. For distinction between the curved bacilli of cholera infectiosa and other curved bacilli it is a *sine qua non that plate-cultures, tube-cultures,* and *potato-cultures be resorted to,* in addition to microscopic examination. The appearance of gelatin-plate colonies of the curved bacillus of Finkler is shown in Photo. No. 5, which is a reproduction of a photo-micrograph of such a colony. As is seen, the outline of the colony is regularly circular; its aspect is finely granular, and its color is yellowish gray. The appearance of the gelatin-tube culture of the curved bacillus of Finkler is represented in the two tubes to the right in Photo. No. 9, as compared with the appearance of gelatin-tube cultures of the curved bacillus of cholera infectiosa, as shown in the two tubes to the left in the same photo. The curved bacilli of Finkler also have the power of liquefying gelatin, but in their growth in ten-per-cent. gelatin there is no appearance of an air-bubble at any time, but development is far more rapid than in the case of cholera infectiosa, and the fluid contained in the liquefied portion completely fills the cavity, and is of a homogeneous opaque aspect. The curved bacillus of Deneke in its growth in the gelatin-plate forms a colony which also has the power of rendering the surrounding gelatin fluid, and the aspect of the colony slightly resembles that of genuine cholera. But its development is much more rapid, the fluidification of the gelatin proceeds with much greater speed, and the zone of fluid surrounding the central colony which rests in the bottom of the saucer-like depression is not clear, but more or less cloudy. The growth in gelatin-tubes is funnel-shaped, and there is an apparent air-bubble at the top. When, however, resort to the potato-culture is had, it is found that the comma bacillus of Koch grows abundantly upon it at normal room-temperature and in the culture-oven, while that of Deneke refuses to grow. Photo. No. 8 represents a number of gelatin-plate colonies, which do not render the surrounding gelatin fluid, of a curved bacillus morphologically identical with that of cholera infectiosa, isolated by the writer from the water of a well which constituted the common water-supply of a small village in Sicily where an outbreak of cholera had occurred. It is sufficient, as these photographs indicate, to cultivate these different organisms in gelatin-plates, in order to distinguish readily between them. In the experience of the author in the examination of the intestinal contents of cholera infectiosa at the quarantine station in New York, and at various places in Spain, France, Italy, Sicily, and India, the comma bacillus of Koch has been invariably present, whilst none of the other curved bacilli have been found therein. He has compared its biological characters with those of all other curved bacilli known, and he has no hesitation in affirming his opinion that there are none of them identical with it.

Various methods of recognizing the presence of the comma bacillus of Koch without resort to the microscope and to plate-cultures have been proposed for practical use, such as those of Bujwid, Buchner, Gruber, Brieger, and others; but in our opinion, however valuable they may be in exceptional cases as supplementary to those above described, they should by no means be substituted in the place of the latter.

Symptomatology.—The symptoms met with in cholera infectiosa are extremely varied, according to the stage and the character and rapidity of the attack. Clinicians who have treated of this disease have generally recognized four stages: *a*, of premonitory diarrhœa; *b*, of serous diarrhœa; *c*, of collapse, algidity, or asphyxia; *d*, of reaction.

a. As to whether the premonitory diarrhœa is to be properly regarded as the actual commencement and an essential part of the disease, or whether it is to be considered as a frequent predisposing simple disorder of the digestive apparatus, opinions have differed, and experienced physicians are still far from harmonious. Whilst in most epidemics of cholera perhaps the majority of sufferers experience the so-called premonitory diarrhœa, yet observers have repeatedly noted its general absence. And, again, where such diarrhœas have been widely prevalent, common experience has shown that only a comparatively small percentage develop into recognized choleraic attacks. If the premonitory diarrhœa indicates a genuine invasion of the organism by the specific infection of the disease, certain it is, nevertheless, that there are many grave and even fatal attacks without its presence. But it is in the experience of all who have had much to do with epidemics of cholera that any one of the recognized stages of the disease may be wanting. It therefore seems unwarrantable, on the ground of its frequent absence, to exclude the first stage of premonitory diarrhœa as a part of the real disease. And from the stand-point of therapeutics it is wise to treat this stage as the commencement of an attack of cholera, which if neglected at this time may ultimately have a fatal termination. If not controlled, the diarrhœa may, after persisting for hours or days, be followed by the onset of symptoms which remove all doubt, during the existence of an epidemic, of the commencement of an attack of the dreaded disease. It is during the night that this onset occurs in the majority of cases.

b. Serous diarrhœa is the symptom which, with its usual accompaniment of intense thirst, nausea, or vomiting, cold, shrunken, wrinkled skin, sunken eyeballs, husky voice, weak, frequent, thready pulse, great prostration, restlessness, anxiety, and cramps, by far the most frequently marks both for the family of the sufferer and for the physician the commencement of the feared attack. If diarrhœa has been present, the alvine evacuations undergo usually a striking and more or less characteristic change, as well as often become much more copious and frequent. Up to this point the disease has been essentially localized, and the intensity of action of the specific poison has fallen upon the lining of the intestinal canal. The intestinal epithelia lose their functions and vitality and desquamate in flakes. Probably even before this the specific poison has reached the circulation and induced a paralysis of the intestinal capillaries and venules. With the desquamated flakes of epithelia the lumen of the intestine now contains serous fluid exuded from the paralyzed capillaries. The intestinal contents are free of bile, resemble a more or less thick meal-gruel or macaroni- or rice-water, and the alvine evacuations present the well-known appearance of such material, but often somewhat foamy, and they are strongly alkaline in reaction. Besides the symptoms above indicated, any of which may be wanting or but little pronounced, there is now more or less suppression of urine. This symptom has by some authors been ascribed to a mechanical effect of the enormous exudation of the fluid of the blood into the intestinal canal. But there is a pathological state of the secretory elements of the kidneys now present which is closely analogous if not identical to that existing in many of the infectious fevers, and it is highly probable that the suppression of urine is in great part the result of the poisonous action upon the kidney of the specific ptomaine contained in the blood.

Whilst serous diarrhœa is customarily an exceedingly prominent symptom in cholera infectiosa, yet there are genuine cases of the disease where it is totally absent,—the so-called cases of *cholera sicca*, dry cholera. In these cases, although there may be no diarrhœa at all, the autopsy shows almost invariably an enormous quantity of the grumous fluid retained in the intestinal canal which it distends. Moreover, the characteristic aspect of the intestinal contents and alvine discharges above described is by no means invariable: instead of a colorless material there may be a yellowish or even a bloody tinge, and there may be a certain admixture of ordinary intestinal contents. The intellect is generally clear.

c. The stage of serous diarrhœa or of rice-water discharges from the bowels, with the accompanying symptoms, lasts for a variable period of two or three to several hours. Reaction may occur at the end, or, what is more frequently the case, collapse may set in. In this stage the vomiting ceases, the serous discharges are interrupted, or the contents of the intestines dribble away unceasingly and involuntarily. The heart almost stops its pulsations; the thickened blood almost ceases to flow; respiration becomes extremely shallow, slow, and irregular; aphonia is complete, as also is anuria; the surface is cold as marble, and livid, especially that of the orbit, nose, lips, fingers, and toes. Even the tongue and the breath are cold. This stage may last for several hours, to end in death or reaction.

Although the surface-temperature, as estimated by the hand or by the ordinary application of the surface-thermometer, is usually below the normal both in the stage of serous diarrhœa and in that of collapse, the temperature of the rectum is higher than in health, and in some cases is greatly elevated. Indeed, notwithstanding the striking coldness of the cutaneous surfaces, at times cadaveric, the patient is usually sensible of the most consuming internal heat. And if death supervene during these stages, the temperature of the corpse may ascend several degrees above the normal body-heat and remain there for some hours. Another post-mortem phenomenon, which is sometimes startling to the uninitiated, is the not infrequent occurrence of marked muscular contractions of the muscles of the face and extremities, productive of various facial contortions and movements of the limbs.

d. The stage of reaction succeeds that of serous diarrhœa or of collapse. In the most fortunate cases convalescence begins at once and proceeds regularly to the rapid restoration of health, with the appearance of bile and of normal fæces in the intestinal canal. But if destruction of the intestinal epithelia and involvement of the subjacent connective tissue of the mucosa have been extensive, or if the formed elements of the blood have seriously suffered, as not infrequently happens, especially in the so-called toxic form of cholera, one of three issues may follow: there may be prolonged anæmia with all its usual sequences, or there may be a long-continued series of digestive derangements, and in either case a very tardy re-establishment of health; or the denuded and inflamed intestinal surfaces may afford entrance to septic germs, and the unfortunate patient pass from the active choleraic seizure into a scarcely less dangerous typhoid condition of reactionary septic fever.

During the stage of reaction numerous and extremely varied cutaneous manifestations have been frequently noted. A few authors have also recorded the occasional occurrence of various cutaneous eruptions during some of the earlier stages of the disease, even during that of premonitory diarrhœa.

According to the character, gravity, and rapidity of the attack, besides those already noted, various qualifying terms have been employed in the description of cholera, among which the principal are *cholerine*, *cholera fou-*

droyante, cholera toxica. By cholerine is meant an exceedingly mild form of the infectious disease, without the development of the stage of collapse or of typhoid reaction. The term *foudroyante* is applied to those exceedingly rapid and grave cases which run their frightful course from beginning to end in a very few hours. In cholera toxica there seems but little evidence of localization of the initial attack upon the intestinal canal; but the nervous centres and the great internal organs are quickly overwhelmed with toxic quantities of the poison.

Differential Diagnosis.—During the prevalence of an epidemic of cholera there is usually no difficulty in recognizing an attack if seen in the stage of serous diarrhœa or of collapse. During the stage of typhoid reaction the course of the fever and the state of the nervous system constitute an ensemble of symptoms very closely resembling the typhoid state of several febrile diseases in which septicæmia plays an important *rôle.* The typhoid condition in cholera infectiosa is, as has already been mentioned, essentially a septicæmia, but it has as complications, which more or less influence the type of the fever, usually serious disorders of the liver and kidneys. The history of the attack, together with the prevalence of the disease, will remove all doubt. As to the stage of premonitory diarrhœa, we have already declared that for the purpose of treatment it is always safe to regard it as the beginning of an attack of cholera infectiosa.

But there is always a time in the course of cholera invasions when the difficulty of an absolute diagnosis is very great, if not really impossible, unless recourse be had to the most recent discoveries. Yet, as we have pointed out already, this is the time when for the purposes of prophylaxis an absolute differential diagnosis should be promptly made. The physician is at such times often required to decide between the presence of Asiatic cholera, cholera nostras, pernicious malarial fever, or some form of ptomaine or mineral poisoning. The existence of the comma bacillus of Koch either in the dejecta or in the vomit determines at once the presence of cholera infectiosa; its absence and the presence in the blood of the plasmodium malariæ prove the existence of malaria.

Treatment.—Knowledge of efficient methods of treatment of cholera infectiosa has by no means kept pace with that of the etiology and prophylaxis of the disease. Unless the so-called methods of *hypodermoclysis* and *enteroclysis* shall prove as effective as the recent experience of some Italian observers would seem to indicate, there appears to have been no marked advance made in the therapeutics of severe attacks of cholera. In this class of cases the mortality varies from thirty per cent. to eighty or ninety per cent., and seems to be far more influenced by the period and intensity of the epidemic and by hygienic surroundings than by therapeutic interference. It has often happened that the ratio of deaths to the number of pronounced attacks has been nearly as great under the management of skilful and experienced European physicians as in the hands of native East Indian attendants whose chief reliance is upon charms and invocations. Whilst this is true of severe attacks of cholera, nevertheless there is scarcely any grave disease which is more manageable if it be properly and promptly treated during the earlier stages. If the mortality of cases in the later stages often rises above ninety per cent. in spite of active and intelligent interference, judicious management of the disease in the earlier stages is usually followed by as great a percentage of cure or abortion of the attacks.

In the stage of premonitory diarrhœa, absolute rest in bed, with warm clothing and abstention from food, should be enjoined; and appropriate doses of laudanum, either alone or in conjunction with some form of camphor, such as chlorodyne, should be administered; or salicylate or tannate of bismuth may be used. Nearly always this simple treatment will prove efficient.

If, however, the simple diarrhœa persists or shows a tendency to assume the serous type, with or without vomiting, coldness, prostration, and cramps, more vigorous treatment is urgently called for. The body should be enveloped in hot flannels, and heat applied to the extremities; cramps should be combated by local frictions, either dry or with whiskey-and-salt or the like; vomiting should be checked, if possible, by swallowing small lumps of cracked ice, and by sinapisms applied over the epigastrium. The two remedies which appear to be most efficacious in this stage of the attack are, however, the warm bath and tannic *enteroclyses*. The temperature of the bath should be 38° or 39° C., and the patient should be immersed in it to the chin and kept there for twenty minutes. After removal from the bath the surface should be very quickly dried and enveloped again in hot flannels, and warm aromatic drinks be given. The bath may be repeated *pro re nata* in two or more hours. The effect of the warm bath, in arresting or allaying the vomiting and in quieting the general nervous system, as well as in restoring warmth to the cutaneous surface, arresting the cramps, stimulating the flagging circulation of the blood, and relieving the general prostration, is often marked to the eye of the observer; and it is usually so comforting to the patient that, although objected to at first, it is frequently called for after being once experienced.

But the *enteroclyses* of tannic acid introduced by Prof. Cantani, of Naples, and so frequently used by other Italian physicians during the recent cholera epidemic in Italy, would seem to afford the greatest reliance in the treatment both of the premonitory diarrhœa and of the active stages of the disease.

If a slight attack of a seemingly simple diarrhœa does not yield at once to rest in bed and the administration of a dose or two of warm infusion of chamomile to which chlorodyne or laudanum has been added in proper quantity, then recourse should be had without loss of time to the warm *enteroclysis* of tannic acid. This enteroclysis is essentially an injection into the colon per rectum of a considerable quantity of warm water holding in solution a certain percentage of tannin. The rectal syringe by means of which the injection is made is furnished with an elastic tube three metres in length, with a nozzle at the free extremity and a cock at the proximal end. With such an instrument not only the whole length of the colon can be filled with the desired fluid, but also not infrequently a quantity can be made to pass beyond the ileo-cæcal valve into the small intestine.

The tannic solution recommended by Cantani is constituted for an adult as follows:

℞ Boiled water or infusion of chamomile, *warm*,	2 litres;
Tannin,	5 to 10 grammes;
Laudanum,	30 to 50 drops;
Powdered gum-arabic,	50 grammes.

The temperature of the mixture and the quantity to be injected should vary, according to the age of the patient and other circumstances, in the judgment of the physician. The most convenient time for administration of an enteroclyster is immediately following an evacuation.

It is the experience of those who have followed this method of treatment that in almost every case of cholera taken at the beginning it has proved successful in a surprising manner in arresting the diarrhœa and stopping the disease.

In the language of Ramello, "If all of those who suffer from diarrhœa in time of cholera would at once have recourse to tannic enteroclysters, the grave cases of this disease would be very rare." But the first and the second phase of the disease, in which medical treatment promises its most certain triumphs, are usually through neglect passed before the physician is called. When first seen the patient has generally advanced far towards or is already in the stage of collapse, when the system is nearly overwhelmed by the quantity of specific poison already absorbed from the intestinal canal and by the excrementitious substance retained in the economy through the failure of the liver and the kidneys to perform their excretory functions, and when neither the substances swallowed per os nor those injected per rectum are longer absorbed.

In this desperate condition the warm bath repeated every hour or two may be resorted to with some prospect of benefit. But it should be supplemented by an attempt to restore to the tissues of the body the large quantities of fluids which have been lost, and to wash out from them some of the excrementitious substances which have not been eliminated. For this purpose intravenous injections have been proposed, and during the last epidemic were more or less extensively practised, but without great success, as a rule. Another method of accomplishing the end in view, less objectionable and more easy of application, has also been proposed by Cantani, of Naples, and practised by him and his countrymen with great success, as reported. It has been named *hypodermoclysis*. It is claimed that this method is neither irrational nor dangerous nor painful nor difficult nor lengthy nor inapplicable in a large number of cases. The method of hypodermoclysis is based by Cantani upon the following reasonable considerations:

1. The death of cholera patients supervenes either by asphyxia in the algid period or in consequence of a tumultuous reaction in the typhoid stage, because the organism through diarrhœa and vomiting has lost a very large quantity of its aqueous constituents, and has retained or cannot longer eliminate the excrementitious materials,—the products of combustion and of decay,—on account of suppression of the functions of the kidneys.

2. Recovery occurs when absorption is resumed, in the intestinal canal, of the fluids which furnish to the blood and to the tissues the water which is indispensable to them.

The office of the physician is therefore to introduce this water into the blood and tissues: not being able to do so either by the stomach or by the rectum, he should have recourse to subcutaneous injections, and in this rational manner satisfy the need of the whole organism.

Cantani suggests as the most successful time for resort to hypodermoclysis the first indications of insufficiency of water in the body, such as discoloration of the skin, cramps, coldness, etc.,—that is to say, in the beginning of the algid period.

The formula for the fluid used by Cantani for hypodermoclysis is, for an adult, as follows:

℞ Pure sodium chloride, 80 grammes;
Sodium carbonate, 6 grammes.
Dissolve in 2 litres of boiled water.

The quantity to be injected each time varies, according to circumstances, from one to two and one-half litres. The temperature of the solution should be 38° C., unless that of the rectum be very low, in which case it has been sometimes raised as high as 43° C.

The apparatus required is very simple. One of the best forms consists of an ordinary fountain syringe having a long elastic tube, to the distal end of which is attached a fine-pointed metallic canula supplied with a cock.

The operation is as simple as the apparatus. The region preferred is either the mammary or the ileo-costal. A fold of the skin is raised, and the canula, previously filled with fluid, is inserted quite a distance between the skin and the subjacent fascia. The fountain of the syringe is elevated until the fluid begins to flow by gravity. In fifteen to twenty minutes one to two litres can be thus injected. During the process the current should be interrupted at intervals by means of the cock. Upon withdrawal of the canula after completion of the operation, the tumor should be gently rubbed, when the fluid will very soon be absorbed.

The warm bath, in conjunction with hypodermoclysis, appears to exercise a powerful influence upon absorption also.

After hypodermoclysis, hypodermic injections of stimulants, often so urgently called for, especially during the stage of collapse or rigidity, become active, whilst they have before been inert.

If after a first injection the coldness and the wrinkling of the skin persist and the secretion of urine be not re-established,—if, in a word, we be convinced that the tissues are not yet supplied with the water which they have lost,—the operation should be repeated some hours later.

"In the majority of cases, however, after the first hypodermoclysis, if the internal losses have not been such as to be incompatible with a good reaction, the circulation is re-established, the eyes open, bathed once more with their natural fluids, and show an expression of consciousness. Little by little the lividity of the skin diminishes, and the timbre of the voice becomes normal. In less than an hour, a person who was at the mouth of the grave is restored to life.

"The physician who knows how to use, with courage and reliance, laudanum, tannic enteroclysis, warm baths, and hypodermoclysis will have to record among the victims of cholera only those unfortunates who when he was called were already well advanced in the stage of cyanosis and collapse."

Such are the confident expressions of an author who has repeatedly seen the marvellous results of this new practice. In summarizing the treatment he says:

"First Period of cholera, improperly called *premonitory diarrhœa:* Rest in bed, warm infusions with laudanum or chlorodyne and cognac; warm bottles to the feet, warm general baths, and *warm tannic enteroclysters.*—Certain cure.

"Second Period, specific or rice-form diarrhœa: Always warm baths, lemonade acidulated by chlorohydric or tannic acid, with laudanum, spirituous liquors, warm tannic enteroclysters, lumps of ice swallowed.—Cure almost certain.

"Third Period, vomiting, diarrhœa always more profuse, cramps and coldness, commencing cyanosis: Hypodermoclysis and warm baths, alternated with tannic enteroclysis, hypodermic injections of stimulants, revulsives externally.—Very many cures."

In the stage of typhoid reaction the skill, judgment, experience, and watchfulness of the physician are taxed to the utmost. In the selection of the line of treatment to be followed, it should be always borne in mind that we have to do with *a fever of a septic character* consequent upon extensive abrasion or destruction of the mucous surfaces of the intestinal canal, and complicated by serious involvement of the liver, of the kidneys, sometimes also of the blood, and of the general nervous system.

Prognosis.—The mortality of cholera infectiosa, as is known, is sometimes frightful. It is usually greatest in the earlier course of the epidemic, and it is limited almost entirely to those who neglect to invoke the aid of the physician until the attack has become exceedingly grave. If the patient is seen early and is promptly, judiciously, and *constantly* cared for, the danger of a fatal issue is not great. If the practice of enteroclysis and hypodermoclysis meet the claims made for them, the disease will be robbed of many of its terrors.

JOINED TWINS.[1]

By WILLIAM WRIGHT JAGGARD, M.D.

Joined Twins, or double monsters, are those malformations that appear to be two individuals which are united to a greater or less degree, and which have mutually influenced development at the site of union. In very many cases, each of the individuals which constitute the monster is symmetrically developed, and the vice of development at the site of union affects both individuals in an equal degree. In other cases, however, one individual is larger and more perfectly developed than the other. The larger frame will then represent the main body, or parent stock, while the smaller individual will appear as a parasitic appendage. There are, then, two general groups of joined twins: 1, the equal or perfect double monsters, and, 2, the unequal or parasitic double monsters. This division is merely for the sake of convenient discussion, since a difference of degree alone exists between coequal duplex individuals and those congenital tumors that contain bone, nerve, fat, and the like, of a second fœtus.

Isidore Geoffroy Saint-Hilaire has formulated certain general laws with reference to the attitudes of the individuals that enter into the constitution of double monsters. These laws may be regarded as demonstrated. They are:

When two or more individuals are united in the composition of a monster, double or more than double, the union takes place between homologous surfaces of the bodies. Thus, in a double monster, if one of the individuals is adherent by the ventral surface, it is to the ventral surface of the fellow that it is generally united, and not to the dorsal nor to one of the lateral surfaces. The same law is true of triplex monsters.

Furthermore, if the two individuals that compose a double monster are compared, they will be found to be placed and their organs disposed more or less symmetrically upon the two sides of the line or of the plane of their union. The same law is true of triplex monsters.

Genesis.—Double monsters and homologous twins are the product of a single ovum. The view is no longer tenable that twins within separate envelopes may come in the course of their development to be so close to each other that the membranous partition disappears, and coalescence of the fœtuses takes place.

---

[1] The manuscript of this article was received too late for it to appear in the place originally assigned to it.

Opinions, however, differ widely as to the mode in which double monsters originate in a single ovum. There still remain a few observers who think these beings arise from the fertilization of a single ovum, containing two germinative vesicles, and from the fusion of the two resultant germinal masses. But the weight of evidence and opinion is greatly in favor of the notion that double monsters have their origin not only in a single ovum, but also in a single blastodermic vesicle. While the proposition is generally accepted that *all double monsters originate in a single ovum and are developed out of a single germinative vesicle*, numerous hypotheses have been suggested to explain the modality of the further growth and development of these beings. At the present time, two principal hypotheses, apparently contradictory propositions, are vigorously defended in the explanation of the phenomena. They are, 1, the hypothesis of Fusion, and, 2, the hypothesis of Fission. To these may be added a third supposition,—though of minor significance,—the hypothesis of Radiation. These three attempts at the interpretation of the phenomena by no means exhaust the possibilities in the conception of the genesis of double monsters, with the records of which the literature of the subject abounds.

1. *The Hypothesis of Fusion.*—According to this hypothesis, when the first traces in the germinal disk become visible, two distinct embryonic areas are already present, which either persist entirely separate and develop into homologous twins, or which unite with each other to a variable extent. This notion is maintained chiefly in France, and Claudius, Panum, and B. S. Schultze may be named among its defenders.

The Germans reject this hypothesis, calling it "naïve," for the following reasons:

1. Fœtal malformations present an uninterrupted transition-series from a supernumerary terminal phalanx to a complete double monster. It is not easily conceivable that a supernumerary terminal phalanx could arise from a twin pregnancy in which all of the one twin except its terminal phalanx had disappeared. In fœtal malformations, moreover, there is no evidence of the disappearance of any organ: the rudimentary organs appear only as aplastic.

2. Double monsters in all their variations show a certain regularity and symmetry of formation; only synonymous organs and systems are conjoined, a condition that would not occur in case of accidental fusion. An apparent exception to this rule is observed in craniopagus, in which the frontal may be found joined to the parietal bone.

3. The fusion of two embryos has never been observed. When two embryos lie too close to each other in the cavum uteri, one is often found pressed out flat,—fœtus papyraceus,—but it is never seen to be fused with its fellow (Perls).

2. *The Hypothesis of Fission* is that the blastoderm in all cases is simple in the beginning, but that this primitive, single germinal mass splits up more or less completely, and gives origin in this mode either to joined twins or to individuals that are separate. This hypothesis also includes the notion of external gemmation or budding. Among the sponsors of the hypothesis may be mentioned the names of Reichert, Dönitz, Förster, Bruch, Dittmar, Virchow, Ahlfeld, Öllacher, and Gerlach. As already intimated, the hypothesis of fission is warmly defended in Germany. This view offers a plausible explanation of the uninterrupted transition-series of malformations from polydactylus to the complete double monster, and the weight of probable evidence is in its favor. In this connection, mention must also be made of the important experimental observation of Gerlach, who has succeeded in producing anterior duplicity in at least one case. Valentin,[1] at a much earlier period, claimed to have brought about duplicity of development in the embryo of the chick by artificial fission, but the facts in his cases have been seriously questioned. Perls[2] makes an ingenious attempt at the reconciliation of these two hypotheses, which, as before remarked, are apparently contradictory propositions.

3. *Hypothesis of Radiation.*—Rauber has announced a third hypothesis, which differs in essential points from the two views just mentioned. This observer assumes a principle of radiation as the basis of his hypothesis. In the earliest period of ontogenesis, the primitive embryonal germ sustains a radial arrangement to the centre of the area pellucida. Rauber calls this mode of development monoradial. In the genesis of a multiple monstrosity, several primitive embryonal germs arrange themselves in a radial fashion, and the mode of development is termed pluriradial. The process of radiation is described in the following words: "Just as under normal conditions the anterior embryonal germ of the vertebrates appears as a projection, as a ray, from the marginal elevation, so the multiple formations appear as multiple projections, or rays, from the marginal elevation."

Causation.—The discussion of the cause of double monsters is foreign to the purpose of this paper, inasmuch as the subject is of a purely speculative nature. Mention, however, must be made of the views of Marchand, that refer the duplicity of the embryonic germ to factors that are in operation before segmentation of the yolk,—that is, to conditions of the ovum or spermatozoid before fertilization. Like Fol, he is of the opinion that the penetration of the vitelline membrane by two or more spermatozoids may give origin to two or more centres of segmentation.

The influence of maternal impressions in the explanation of the occurrence of double monsters is often invoked, and, unfortunately, not merely by the laity. Although, as remarked by Parvin, the belief that the unborn child may be affected through the mother's mind has the alleged criterion of truth that it is universal and perennial, nevertheless the notion rests upon no basis in objective fact. Experience teaches that fright, bodily violence, and the like may cause structural alterations in the ovum as the effect of hemorrhage, partial rupture of the membranes, incomplete loss of their contents, that sometimes result in the production of monstrosities. But the direct influence of the mental attitude of the mother upon the physical development of the fœtus in utero, in the absence of any such coarse physical cause, is an item of absolute conjecture. Then it must be borne in mind that all gross errors of development are announced at a very early period in the history of the embryo,—before the expiration of the eighth week,—a period considerably antecedent to the time frequently appointed by the popular mind for the operation of maternal impressions.

---

[1] Arch. f. phys. Heilk., 1851.

[2] Allgemeine Pathologie, 1886, p. 668.

Nomenclature.—The nomenclature of double monsters is extraordinarily rich. In this article the terms employed by Gurlt and Förster will be chiefly used.

When the common portion is relatively extensive, the monster creates the impression of being a single individual of which some particular part is duplex, and is less suggestive of two individuals. Under these conditions, the designation is selected that is made up out of the name of the duplex portion, and the termination *-didymus* (δίδυμος, "double"), or the prefix δι-, is used.

On the other hand, when the common portion is relatively small, the monster creates the impression of being two individuals that are united, and is less suggestive of a single being. Now the name is chosen from the portion common to the two individuals, and the termination *-pagus* (πήγνυμι, to "unite"), or the prefix συν-, is employed. Then there are also analogical terms, as *Janiceps*.

Classification.—As remarked by Harrison Allen, it is an open question to what extent a malformation like a double monster can be called a genus, and to what degree the subdivision of each genus may be designated a species, so long as naturalists are not agreed as to the restriction of these terms in normal plants and animals. For the purpose of discussion, however, some systematic arrangement of these objects is absolutely necessary. The original classification, proposed by Isidore Geoffroy Saint-Hilaire, has been variously modified by Gurlt, Förster, and others, in accordance with the hypothesis of the development of these beings by the fission of a single germinal area.

The axial structures may be split from above downward, giving origin to double monsters that are joined at their caudal extremity, but whose cerebro-spinal axes diverge in varying degrees as they ascend,—hence *Terata katadidyma*, monsters with downward cleavage.

Double monsters may be joined at the cephalic pole with divergence of the caudal extremities. In this manner are produced beings that are single above and double below,—*Terata anadidyma*, monsters with upward cleavage.

Finally, fission may occur at the same time from above downward and from below upward, but the process is arrested before complete separation, and there result beings whose cephalic and caudal extremities are divergent while the intermediate region is continuous in a greater or less degree,—*Terata anakatadidyma,* monsters with both upward and downward cleavage.

DOUBLE MONSTERS.

A. TERATA KATADIDYMA.
- Monocranus.
- Dicranus.
- Diprosopus.
  - Iniodymus (Fig. 1).
  - Opodymus (Fig. 2).
- Dicephalus.
  - D. dibrachius (Fig. 3).
  - D. tribrachius.
  - D. tetrabrachius (Figs. 4 and 5).
  - D. tetrabrachius tripus (Fig. 6).
  - D. parasiticus.
- Ischiopagus.
  - I. tetrapus (Figs. 7 and 8).
- Pygopagus (Figs. 9 and 10).
  - P. parasiticus.

B. TERATA ANADIDYMA.
- Dipygus.
  - D. dibrachius tetrapus (Figs. 11 and 12).
  - D. dibrachius tripus parasiticus (Fig. 13).
- Syncephalus.
  - Janiceps asymmetros (Figs. 14 and 15).
  - Janiceps (Fig. 16).
- Craniopagus (Figs. 17 and 18).
  - C. parasiticus.

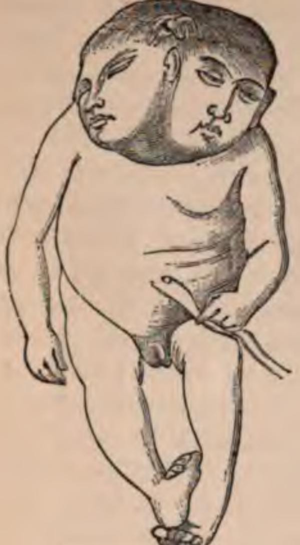
C. TERATA ANAKATADIDYMA.
- Prosopo-thoracopagus (Fig. 19).
  - P. parasiticus.
- Thoracopagus.
  - Xiphopagus (Fig. 20).
  - Sternopagus (Figs. 21 and 22).
    - S. parasiticus (Figs. 23 and 24).

TERATA KATADIDYMA. *Monocranus,*—single cranium, face double in part, three or four eyes, brain duplex in various degrees.

Dicranus,—double cranium, face single or double and fused, lower jaw single.

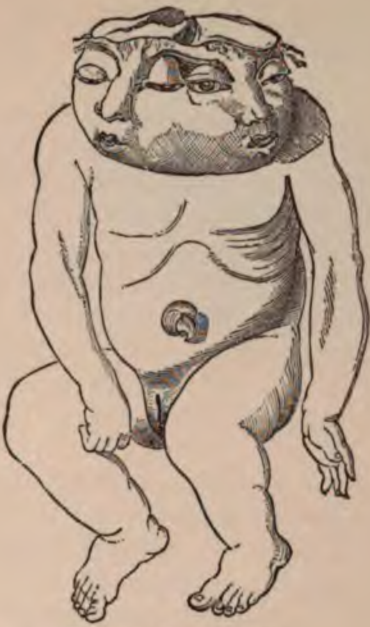
Diprosopus,—face more or less double; the faces, and in part the

FIG. 1.



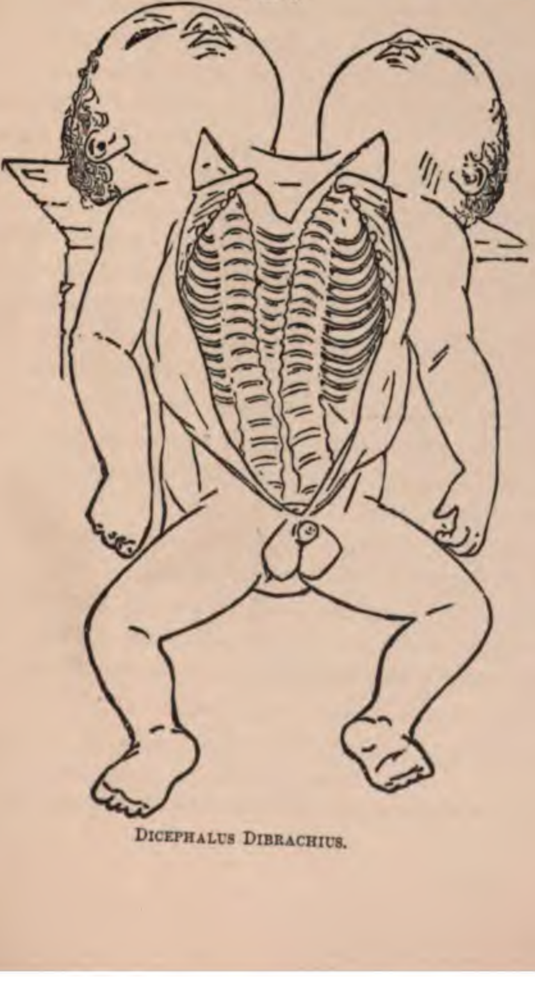
INIODYMUS (Depaul).

FIG. 2.



OPODYMUS (Soemmering).

FIG. 3.



DICEPHALUS DIBRACHIUS.

cranium to the occiput, completely separate (Iniodymus), or the faces are separated to the zygomatic arch (Opodymus); in both cases, double lower jaw (Figs. 1 and 2).

Dicephalus,—characterized by the presence of two distinct and separate heads, either equal or unequal, with various degrees of duplicity in the vertebral column. The bodies are joined laterally, the faces look forward and commonly in the same direction. According to Fisher,[1] out of five hundred cases of human double monsters that are recorded, about one-third belong to this genus. The female sex preponderates in the proportion of two to one. In very rare cases, one of the embryonal masses persists in a rudimentary state, and forms an appendix to its fellow,—*Dicephalus parasiticus.* Under Dicephalus are included the following species:

Dicephalus dibrachius.—A. Otterson[2] has given the following excellent report of a case of this species:

History.—Mother, a multipara; utero-gestation was unattended by any event at all noteworthy; earlier stages of labor prolonged; a head was finally delivered by forceps, after which no progress was made for some hours. Dr. Otterson attempted to introduce his hand into the uterus for exploration; partial version of the retained parts, engagement of the breech; efficient contractions with expulsion of the breech and body, followed by the second head. The child was dead when delivered. The mother made an uninterrupted recovery.

The specimen was presented to the Museum of the Brooklyn Anatomical and Surgical Society, and has been accurately described in the following words by Lewis S. Pilcher[3] (Fig. 3):

The child has two distinct and perfect heads and necks, one trunk, two upper and two lower extremities; length, forty-eight centimetres. The two heads differ slightly in size; circumference of the left head thirty-six centimetres, that of the right head thirty-three centimetres. There is no anal orifice. The genitals are male, single and perfect.

Skeleton.—The vertebral columns are distinct and perfect throughout; they approach each other gradually from above downward as far as to the lumbar region, whence they run parallel to each other, being separated by a small interval; the sacra, each distinct and perfect, articulate with each other by means of an interarticular fibro-cartilage that unites the contiguous auricular surfaces of the two bones. From each sacrum springs the innominate bone that forms the wall of the pelvis upon that side; at the symphysis pubis the two unite as usual. The corresponding dorsal vertebræ of the two columns are united by a series of bony arches formed by coalesced ribs; each arch or compound rib has two normal heads, one at either extremity, by which it articulates with the proper vertebræ. The ribs, which spring from the free sides of the two vertebral columns, are connected to a common single sternum in front by unusually long costal cartilages, and thus complete the thorax.

---

[1] Transactions New York State Medical Society, 1865, 1866, 1867, and 1868.

[2] Annals of Brooklyn Anatomical Society, 1880.

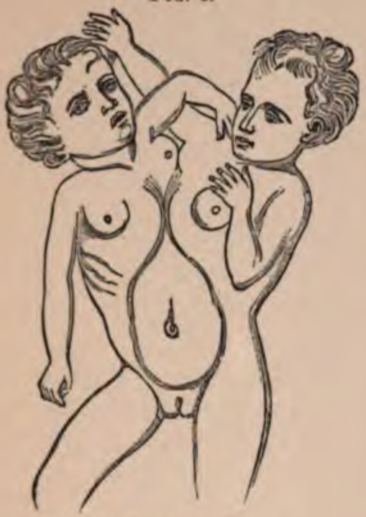
[3] Annals of the Anatomical and Surgical Society, Brooklyn.

The clavicle and scapula of either side are normally related to the sternum and ribs. Resting upon the posterior face of the upper compound ribs, in the middle of the back between the two series of dorsal spines, is a compound scapula formed by the fusion of two bones along their anterior edges; an acromion process, club-shaped, projects forward from the middle of the upper edge of this compound scapula; articulating with this process, and passing directly forward to articulate with the sternum at its upper border, the episternal notch affording an articulating surface, is a slender compound clavicle.

Respiratory System.—Two sets of respiratory organs are present, each independent and perfect. There are four pleural sacs. By the blending of the pleural layers that lie in contact in the middle line, a fibro-serous septum is formed that divides the thorax into two cavities posteriorly; these middle pleural sacs and their contents are hidden from view anteriorly by a large pericardial sac, with the posterior wall of which the anterior margin of the septum described becomes blended.

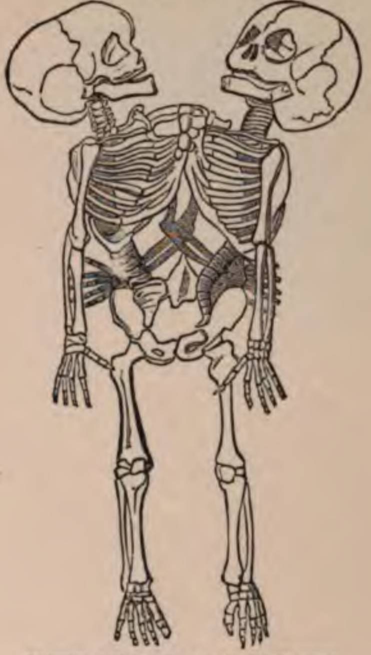
Circulatory System.—The pericardium lies in the middle line, directly behind the sternum, and extends to some distance on each side of it. The sac is single, and encloses a compound heart, the ventricular portions of which remain separate, while the auricles are blended together. Constituting the left mass of the heart are two ventricles and one auricle (the left), which are of normal size, shape, and relative position. The origins and relation of the aorta and pulmonary artery upon this side are normal. Into the auricle enter four pulmonary veins. The elements of the right mass are more changed: there is but one ventricle, which, however, is larger than either of the ventricles of the other mass; from the right side of its base springs a second aorta; there is no pulmonary artery on this side. There is no apparent attempt at differentiation of the auricles: there is simply a single capacious auricle, which is blended with the right auricle from the left mass, forming a huge venous reservoir. At the right posterior side of this reservoir enter two small pulmonary veins from the right pair of lungs. A single ascending vena cava collects the blood from the lower portion of the body; above, the left innominate vein of the left child crosses transversely its neck to the point of junction of the two necks, receiving the right internal jugular in its course; here it is joined by the left internal jugular of the right neck, and by a large anomalous vein from behind; the large descending vena cava thus formed descends in a straight course to the middle of the compound auricle. The right innominate vein, formed by the right internal jugular and right subclavian veins of the right neck, empties into the compound auricle at its right side. The two aortas descend each upon the left side of their proper vertebral columns; they do not unite below, nor bifurcate, but each diverging continues as a common iliac, and,

FIG. 4.



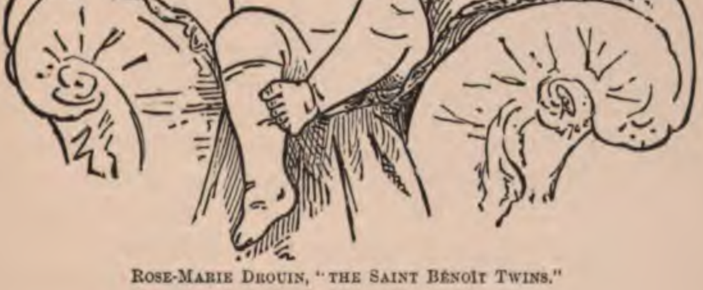
RITA-CHRISTINA (Serres).

FIG. 5.



DICEPHALUS TETRABRACHIUS (Serres).

FIG. 6.



ROSE-MARIE DROUIN, "THE SAINT BÉNOÎT TWINS."

after giving off the umbilical artery, passes on to be distributed to a lower limb.

Digestive System.—There are two stomachs. The left is of normal shape and size, and occupies its usual place in the abdomen. To its cardiac end is attached a normal spleen,—the only one present. The right stomach is smaller, pyriform, hidden behind the liver, and lies very obliquely, with its pylorus pointing towards the pylorus of the other. Its duodenum joins at once the left duodenum, and the two bowels appear fused together for about one-sixth of their entire length; a well-marked longitudinal groove so marks the fused bowel that the appearance of a double-barrelled gun is produced; transverse section shows that they are divided by a membranous septum into two distinct tubes, each with its own mesentery; this persists through a length equal to one-third of the whole; then they again fuse, and the double-barrelled arrangement persists through a length somewhat greater than at the beginning; the small intestine finally becomes single, and continues thus to its junction with the large intestine, which likewise remains single to its termination; at the point of beginning of the single tube a small nipple-like diverticulum exists. The rectum descends to the bottom of the pelvis, where it ends in a *cul-de-sac.* The liver, upon its surface, appears to be a simple organ, but from its posterior inferior border project supernumerary lobes, the evident remains of a second liver. There is but one gall-bladder.

Genito-Urinary System.—There are three kidneys,—a large compound kidney lying in the mid-lumbar sulcus, and one in either lateral lumbar region. The left kidney is greatly atrophied. The bladder is single; the genital organs are single and well developed.

Nervous System.—Each head and neck and each lateral half of the body is supplied by its own cerebro-spinal axis; along the line of fusion only is there any communication between the branches of the two axes.

The important question as to the viability of this monster, apart from the accidents of birth, has been answered in the negative by Pilcher, from consideration of the structure of the heart and great vessels. The left mass of the compound heart was complete, and apparently equal to the performance of its functions, so far as they related to the left child; but the right mass, composed of but one auricle and one ventricle, with no pulmonary artery, was obviously unequal to its task. There was present no anastomosis between any large arteries of the two systems to permit the admixture of arterial blood with the venous current of the right system. Accordingly, had the monster been born alive, the right child would have perished at once from asphyxia, and the speedy death of the left child would have followed.

Dicephalus tribrachius.

Dicephalus tetrabrachius is an example of anterior duplicity in which cleavage extends downward through the thorax as far as to the abdomen. These beings possess two hearts and two pairs of lungs, and are viable. Occasionally the sacrum and ilia are double.

A specimen of dicephalus tetrabrachius dipus is presented in the well-known case of Rita-Christina, who died at Paris, November 23, 1829, having reached an age of eight months and eleven days. The autopsy was conducted by Serres, from whose report Figs. 4 and 5 are taken.

Sometimes a third lower extremity, either rudimentary or perfect, is present,—dicephalus tetrabrachius tripus (Fig. 6). Of this species the living female double monster known as the St. Bénoît twins is a typical example.

Prof. Duncan C. MacCallum, of Montreal, gives[1] the following description of this specimen:

These children are the products of a second gestation. They were born at St. Bénoît, county of Two Mountains, Quebec, on February 28, 1878. The mother is a fine, healthy-looking woman, aged twenty-six years. Her labor lasted seven hours, commencing at one A.M. and terminating at eight A.M. One head and body were born first; this was shortly followed by the lower extremities, and immediately afterwards the second body and head were expelled.

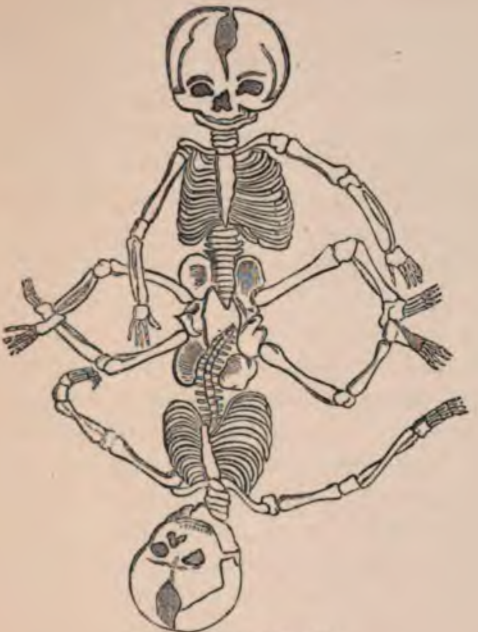
The names Marie and Rose have been given to the right and left child respectively; surname, Drouin.

Marie is more strongly developed and healthier-looking than her sister Rose, who is smaller, darker, and more delicate-looking. They are both bright, lively, and intelligent-looking children. The two bodies, from the heads as far as the abdomen, are well formed, perfectly developed, and in a state of good nutrition. The union between them commences at the lower part of the thorax of each, and from that part downwards they present the appearance of one female child; that is, there is but one abdomen with one navel, a genital fissure with the external organs of generation of the female, and two inferior extremities. The floating ribs are distinct in each, as is also the ensiform cartilage. The lateral halves of the abdomen and the inferior extremities correspond in size and development respectively to the body of the same side; and the same remark applies to the labia majora. The spinal columns are distinct and appear to meet at a pelvis common to both, although the fusion of the children commences at some distance above their junction. From near the extremity of each spine a fissure extends downward and inward, meeting its fellow of the opposite side at the cleft between the buttocks near the anus, including a somewhat elevated soft fleshy mass, thicker below than above. At a central point between these fissures, at the distance of sixty-four millimetres from the point where the vertebral columns meet, and eighty-nine millimetres from the anus, there projects a rudimentary limb with a very movable attachment. This limb, which measures one hundred and twenty-seven millimetres in length, and is provided with a joint, tapers to a fine point, which is furnished with a distinct nail. It is very sensitive, and contracts strongly when slightly irritated.

---

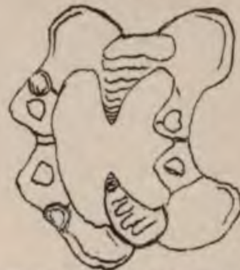
[1] Canada Medical and Surgical Journal, October, 1878.

FIG. 7.



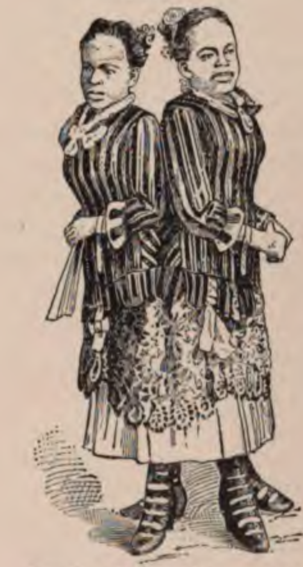
ISCHIOPAGUS (Prochaska).

FIG. 8.



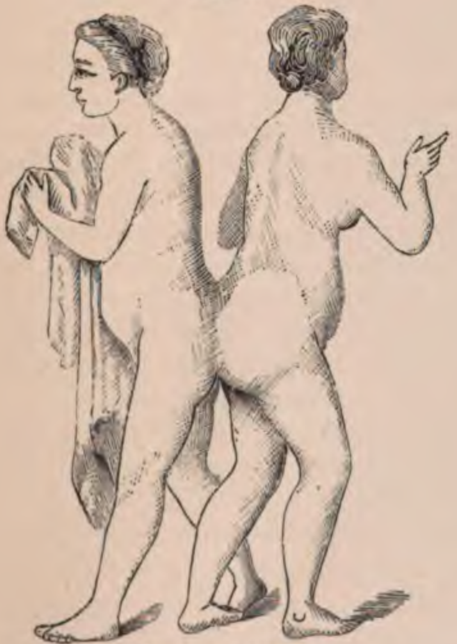
PELVIS OF AN ISCHIOPAGUS (after Du Verney).

FIG. 10.



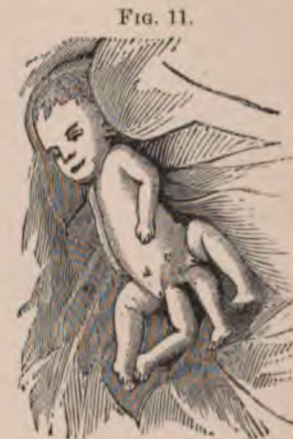
THE TWO-HEADED NIGHTINGALE.

FIG. 9.



HELEN AND JUDITH.

FIG. 11.



DIPYGUS TETRAPUS.—Four-legged female infant, J. Myrtle Corban (from photograph taken in Nashville, Tennessee, June 16, 1868).

The spinal, respiratory, circulatory, and digestive systems of these children are quite distinct. They have each a separate diaphragm, and the abdominal muscles on each side of the mesial line and the limbs of that side are supplied with blood by the vessels and are under control of the nervous system of the corresponding child. They have each a distinct stomach and a separate alimentary canal (the two alimentary canals probably joining at a point close to the common anus). It would follow that the accessory organs of the digestive system are distinct for each child. The two fissures behind are evidently the original clefts between the buttocks of each child, one buttock remaining in its integrity, whilst the other in a rudimentary condition is fused with that of the opposite child, forming the soft fleshy mass from the upper part of which the rudimentary limb projects.

The St. Bénoît twins were seen and photographed by Lewis S. Pilcher when they had attained the age of thirteen months. Pilcher's observations correspond with those of MacCallum. He says, "Its vital functions were all being performed regularly and properly, and the mental development of the two parts was equal to that usual in children of its age. It apparently had as good an expectation of living to maturity as any other infant."

It is claimed that the St. Bénoît twins survived their birth a longer period than any other recorded case of the three-footed, four-armed dicephalic monster. MacLaurin's case[1] lived only two months. The St. Bénoît twins lived about three years. The alleged cause of death was extreme exposure and fatigue consequent upon exhibition in various museums.

Ischiopagus.—The ischiopagi are characterized by the pelvic union of two individuals, with common umbilicus, placed end to end and in a similar position,—that is, the trunks, necks, and faces are in the same plane, and at right angles to the direction of the lower extremities (Saint-Hilaire). The pelvic bones, duplex, form a ring, from which four, or in case of asymmetrical disposition three, lower extremities proceed.

Preenay records an example of this species. The twins, female, had club-feet; they lived for several months. Serres's case lived four weeks. (Figs. 7 and 8.)

Pygopagus.—According to Veit,[2] this genus includes those double monsters in which two complete individuals are united in the region of the buttocks by the sacrum and the coccyx, or only by the latter and the adjacent soft parts. Each individual has a distinct umbilicus. Förster has collected ten examples of this monstrosity. All were viable. Of the internal organs, only the rectum, and in a number of the cases the vagina, were single.

The best-known example of this genus is presented in the case of the Hungarian sisters Helen and Judith, who were born in 1701 and died in 1723. According to Geoffroy Saint-Hilaire, who has described this remarkable case in detail, at the birth of the twins Helen presented by the vertex and was delivered as far as the umbilicus; after the lapse of three hours the rest of her body was expelled, followed at once by the birth of Judith, who presented by the feet (Fig. 9).

---

[1] Philosophical Transactions, London, 1723, vol. xxxii. p. 346.

[2] Die Geburten missgestalteter, kranker und todter Kinder, Halle, 1850.

Helen and Judith are alleged examples of the influence of maternal impressions. Dr. Torkos[1] begins the description of this monstrosity by citing the proof which it furnishes of the influence of the imagination of the mother on the fœtus; for at the commencement of her pregnancy the mother witnessed, with extreme attention, two dogs glued together during the act of coition, their heads turned each to its respective side, and she was unable to efface this picture from her mind.

In the cases of pygopagi reported by Norman, Jungmann-Kleinwächter, the delivery of the twins occurred in much the same way as in the case of Helen and Judith. At a recent period the two-headed nightingale, Christie and Millie, has attracted attention (Fig. 10).

R. P. Harris informs us that the celebrated South Carolina twins, born July 11, 1851, were brought into the world by the same method, but the mother, having a large pelvis, "had a brief and easy" delivery. The larger of the two girls also came first, as in the Tzoni case of 1701. I believe these twins are still living.

The attempt at separating the two individuals in pygopagus by operative procedure proved fatal in one case.

TERATA ANADIDYMA.—The double monsters that are examples of posterior duplicity seldom present symmetrically developed forms in the human race.

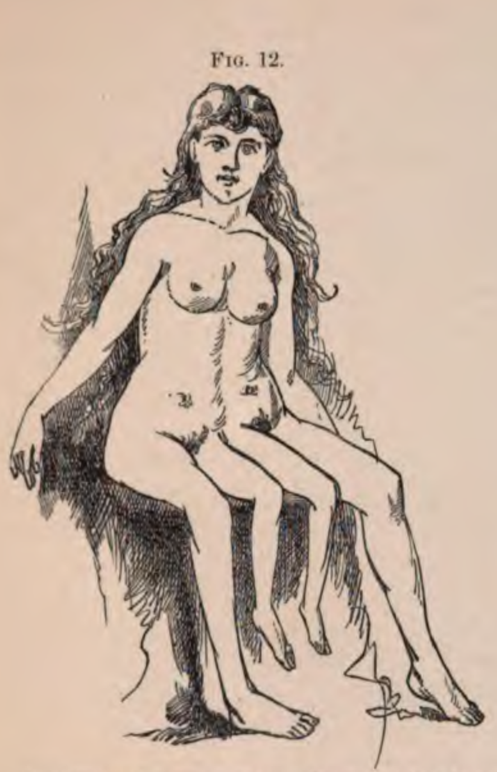
Dipygus.—The dipygi are examples of posterior duplicity in which cleavage upward occurs through the pelvis and the posterior aspect of the lower portion of the spinal column. These monsters consist of a single head and neck, single or double thorax, two or four upper extremities, single or double abdomen, and three or four lower extremities. Symmetrical development of the duplex regions is rare. Commonly, asymmetrical forms—dipygus parasiticus—are observed, in which larger or smaller portions of the parasite protrude from the autosite. It has been noted that the higher the development of the parasite the nearer to the cephalic end of the autosite it comes to lie. Thus, if the parasite is composed of a trunk and upper and lower extremities, it is attached to the mouth, neck, or chest of the autosite; if composed only of lower extremities, these take their origin in the pelvis of the autosite. When the spinal column and pelvis are single, the transition into polymelia of the lower extremities is presented. The parasite is always without a heart, and its blood-vessels are derived from the autosite.

As at present informed, the most remarkable example of posterior dichotomy on record is the case of Mrs. Clinton Bicknel, *née* J. Myrtle Corban, of Blount County, Alabama. This being is an example of dipygus

---

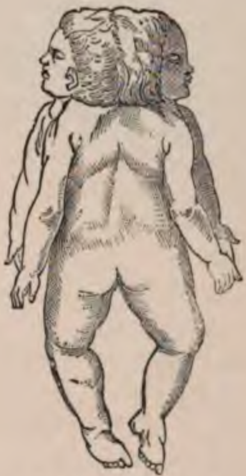
[1] Histoire des Accouchements, etc., G. J. Witkowski, Paris, 1887.

FIG. 12.



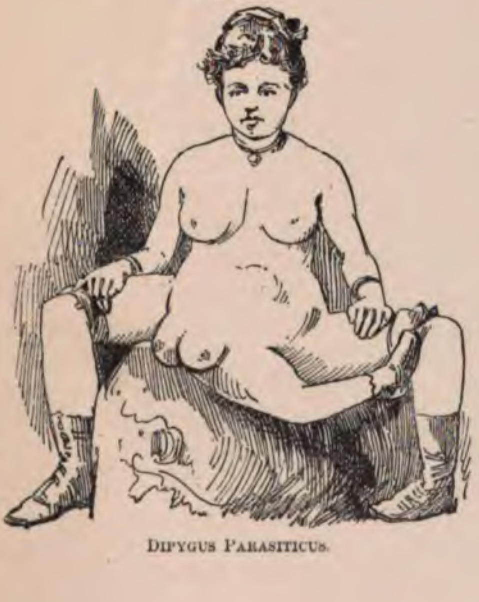
DIPYGUS DIBRACHIUS TETRAPUS. (From American Journal of Obstetrics and Diseases of Women and Children, December, 1888.)

FIG. 16.



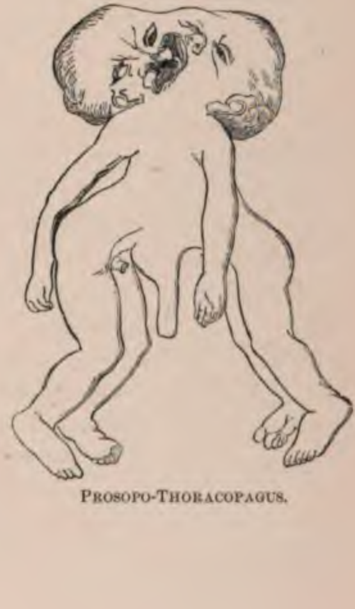
JANICEPS (Bordenave).

FIG. 13.



DIPYGUS PARASITICUS.

FIG. 19.



PROSOPO-THORACOPAGUS.

dibrachius tetrapus. Joseph Jones and Paul F. Eve, of the University of Nashville, made an examination of this individual soon after her birth, and have written the following history:[1]

"Josephine Myrtle is the third child of W. H. and Nancy Corban, aged twenty-five and thirty-four years, the wife being the senior by nine years. They are so much alike in appearance—both having red hair, blue eyes, and very fair complexion—as to produce the impression of their being blood kin, which, however, is not the case. Mrs. Corban, from North Alabama, has borne one child to a former husband; the child has dark hair and eyes, resembling the father. Her four children are girls.

"The subject of this history was born May 12, 1868. The course of the pregnancy was normal. Labor, normal; presentation, vertex. Weight of child when three weeks old, ten pounds. The head and thorax are single, while the lower portion of the trunk is divided into the members of two distinct individuals. The spinal column divides at the third lumbar vertebra; there are two pelvic arches supporting the four lower extremities. An inch below the navel is the mark of an apparent failure in the development of a second umbilicus. The external genito-urinary organs are double and separate; the internal genito-urinary organs are presumably duplex. The rectum is double, with two anal orifices. The nates from below appear as those of two individuals, with a distinct cleft between them. The urine and fæces are commonly passed at the same time upon both sides. There are four distinct, fairly well-developed lower extremities. They exist in pairs on both sides of the median groove, which resembles the cleft of an ordinary pair of legs. The outer legs of both sides are the more natural of the four (though the foot of the right one is clubbed), but are widely separated by the two supernumerary ones, which are less developed, except at their junction with the body, from which point they taper to the feet and diminutive toes; the toes are turned inwards. One toe on the left inner foot is bifid. At birth these extra legs were folded flat upon the abdomen."

Lewis Whaley,[2] of Birmingham, Alabama, completes the history of the case. In the spring of 1887 he was called to see Mrs. B., who had been married about one year, on account of the following symptoms: pain in the left side, nausea, capricious appetite, headache and fever, amenorrhœa of two months' duration. She suffered from pain above the pubes, and thought an abscess was forming. Upon being informed that she was pregnant, she replied that she thought the physician was mistaken, but that she could have believed it more readily if the alleged pregnancy had been on the right side. Eight weeks later, after consultation with Drs. Haden and Aldridge, abortion was induced, under the twofold indication of contracted pelvis

---

[1] Journal of the American Medical Association, October 20, 1888, p. 545.

[2] Atlanta Medical and Surgical Journal, September, 1888; British Medical Journal, September 22, 1888, p. 676; Transactions of the State Medical Association of Alabama, 1888; The Medical Standard, October, 1888, p. 105; American Journal of Obstetrics, etc., December, 1888, p. 1265.

(antero-posterior diameter of outlet of left pelvis two inches, transverse one and one-half inches) and incoercible vomiting, by puncture of the membranes. She was delivered of a well-developed fœtus of three months and one-half; her recovery was rapid and complete.

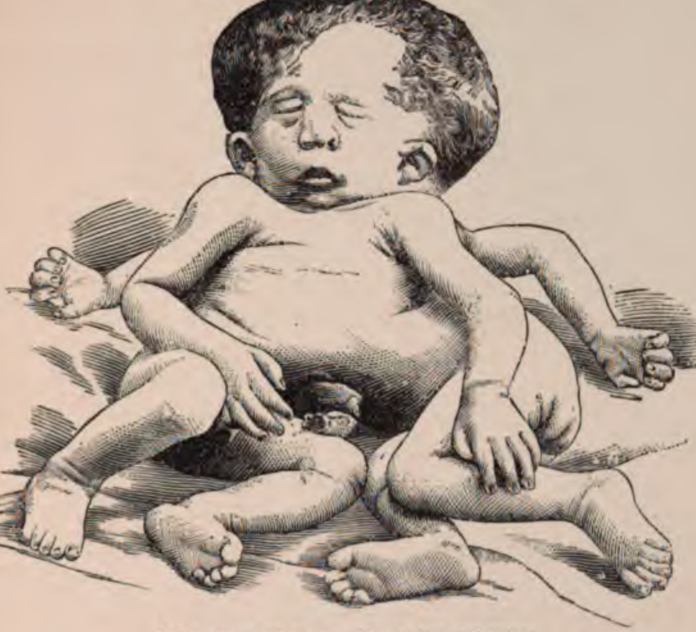
As described by Dr. Whaley, Mrs. B. is a refined woman, of some musical taste. Her very large hips are the chief thing noticeable about her. Her waist is also rather disproportionate to her height. She is a well-developed woman from the umbilicus up. About an inch from the navel is a second one partially developed. The lower extremities are present in pairs on either side of the median groove, which resembles the cleft of an ordinary pair of legs, except that there is no evidence of anus or genital organ. Between each pair of legs there is a complete, distinct set of genital organs, both external and internal, each supported by a pubic arch. Each set acts independently of the other, except at the menstrual period. There are apparently two sets of bowels (*sic*), and two ani; both are perfectly independent,—diarrhœa may be present on one side, constipation on the other. Menstruation began at the usual age, is normal, and occurs simultaneously from both sides. The two outer limbs, on which the woman walks, are well developed, though the foot of the right is in a state of equino-varus. The inner limbs are smaller, atrophied from disease, and, below the knee, very rudimentary. The accompanying cut is from a drawing by Dr. Whaley, and was made under the direction of Dr. Brooks H. Wells. (Figs. 11, 12.)

An interesting case of dipygus dibrachius tripus parasiticus has been recently described by J. Bechtinger,[1] Para, Brazil (Fig. 13). Bechtinger writes, "This person is twenty-five years of age, a native of Martinique (French West Indies), her father a Frenchman, her mother a quadroon. Both healthy, not remembering any deformity in their family or kindred, no constitutional disease,—syphilis, scrofula, nor allied maladies. The third leg is attached to a continuation of the processus coccygeus of the os sacrum, such as I have noticed among some Malay tribes in the interior of Sumatra (Dutch East Indies); however, not in such proportion even approximately. She is still living, but left her native country for Paris, where this photograph was taken, about one year ago. Besides the two well-developed mammæ in their natural position, a third one, that is double, is seen above the os pubis. The hair surrounding the lower segments of the abnormal mammæ covers the vulvæ, that are supplied with well-developed vaginæ. Both vaginæ are properly supplied with nerves, and normal sexual connection, with natural sensations, is possible in either vagina. The sexual appetite is very markedly developed. Being informed of the existence of a man in France who had two sets of genitals,—two penes, four testicles, and three legs,—she at once removed to Paris to make his acquaintance.

---

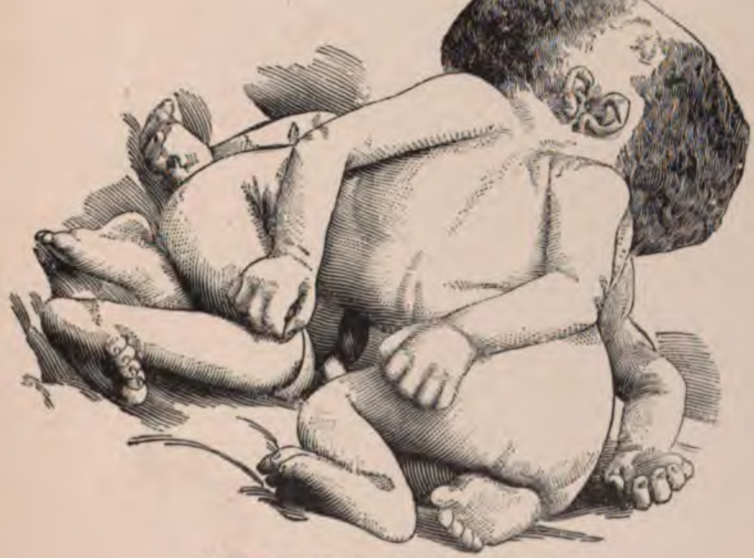
[1] The Medical Standard, October, 1888, p. 107; Annals of Gynæcology, May 1, 1888.

FIG. 14.



JANICEPS ASYMMETROS, FRONT VIEW (Sterley).

FIG. 15.



JANICEPS ASYMMETROS, SIDE VIEW (Sterley).

Syncephalus.—Under this genus are included those beings that represent the higher degrees of posterior duplicity. The typical form of this group is presented in the not uncommon double monster known as the Janus head. The thoraces are confluent above the single umbilicus, the left head and superior trunk being so united as to give to the anterior surface the appearance of a single individual. At the same time, the axes of the bodies are commonly not exactly parallel, but inclined to each other at an angle, and, in consequence, there is an arrest of development of the median portion of the anterior surface. In these forms—Janiceps asymmetros—there is a perfectly developed face only on one side; on the other there is present a rudiment, that shows varying degrees of fission,—agnathia, synotia, synophthalmia, and the like.

Figs. 14 and 15 are anterior and posterior views of a case of Janiceps asymmetros that occurred in the practice of Dr. J. B. Sterley, of Reading, Pennsylvania,—then living in St. Mary's, Elk County,—February 23, 1878. The specimen at present is in the Army Medical Museum.

The history of this case, as recorded by Parvin,[1] is: Mother, a German, eighteen years of age, one child before this pregnancy; the labor, thought to be premature by one month, protracted, the child living when it began, but dying during its course. Cephalic presentation, spontaneous delivery. Weight of monster, six pounds ten ounces.

Inspection of the rudimentary face, Figs. 14 and 15, shows synotia, synophthalmia, and agnathia. The monster was of the female sex. This case resembles closely a specimen of cephalothoracopagus in the Giessen collection, described by Vrolik.[2]

In rarer forms, the two bodies are exactly parallel to each other, so that each surface of the monster shows a fully-developed face, and the perfect type of the Janus head—Janiceps—is produced (Fig. 16).

In syncephalus, when the body of one of the twins is not developed, there results the form known as Janus parasiticus.

The characters of the varieties Iniops (ἰνίον, the "occiput," and ὤψ, the "eye," the "face") and Synote (σὺν, "with," and οὖς, ὠτός, an "ear") have been pointed out in connection with Dr. Sterley's case.

Craniopagus.—In craniopagi, the highest degree of posterior duplicity is observed. Two symmetrically developed forms are united at the cephalic extremity. According as the site of union is the occiput, the vertex, or the forehead, there result three varieties, respectively,—craniopagus occipitalis, parietalis, and frontalis. Each individual has a separate navel and umbilical cord. Förster collected seventeen examples of this monstrosity; one or two additional cases have been recorded within a recent period. Union at the vertex is most frequent, so that the two individuals lie in the same plane. The axes of their bodies may be inclined to each other at an angle, or they may be rotated so that the faces look in opposite directions. Homologous bones are not always united. Thus, the frontal bone of one child may coalesce with the occiput of the other. At the site of union there is either lacking a portion of the skin and bone, or the dura mater may also be involved. The brains, however, are commonly distinct and the beings are viable. Several have lived from six to ten years. (Figs. 17 and 18.)

---

[1] American System of Obstetrics, vol. i. p. 780.

[2] Perls, loc. cit., p. 654.

Craniopagi seldom occasion difficult labor, unless the axes of the bodies are inclined to each other at an angle. Vottem records a case in which a shoulder presented, that terminated after the performance of podalic version.

An interesting case of craniopagus frontalis is recorded in Münster's "Cosmographia" (1552). The twins, female, were born in 1495, and lived ten years. Upon the death of one of the children the bond of union was severed, but the other child perished within a short period. As the mother, during her pregnancy, was talking with another woman, a third individual is said to have stepped up behind them and to have brought the heads of the two in forcible contact.

TERATA ANAKATADIDYMA.—*Prosopo-thoracopagi* include joined twins in which the inferior portions of the two faces, the necks, and the thoraces are fused, while the cranial bones and cavities as well as the lower abdomen are distinct and separate. Examples of this double monster are very rare. (Fig. 19.)

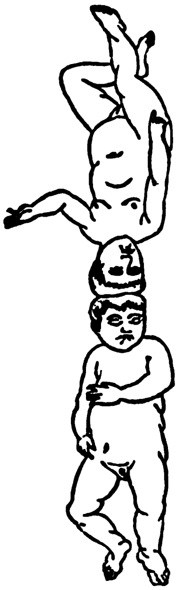
Prosopo-thoracopagus parasiticus.—Thoracopagi are double monsters that are united by the thorax and superior abdomen. They are of relatively frequent occurrence. Since they possess a single navel and umbilical cord, they are included under the class of omphalopagi. The degrees of union are manifold, and different forms accordingly arise.

Xiphopagi include those forms that are united at the processus xiphoideus by a cartilaginous bond. The best-known example of xiphopagus is presented in the case of the Siamese Twins Chang and Eng, who died at the age of sixty years. (Fig. 20.)

As informed by R. P. Harris, "The mother of these twins was a Chinese half-breed, short, and with a broad pelvis, and had borne several children previously. She stated on several occasions, in conversation with parties in Siam, that the twins were born reversed, the feet of one being followed by the head of the other, and that they were very small and feeble at birth and for several months afterwards. The twins confirmed this statement by affirming that they could, when little boys at play on the ground, turn themselves end for end upon the ensiform attachment, up to the age of ten or twelve, the attachment being then soft and pliable."

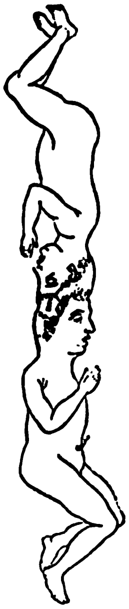
The bond of union in the case of the Siamese Twins was much smaller than is commonly observed; in position it was strictly ventral, and contained hepatic tissue that seemed to unite the two livers. Sir Astley Cooper, in view of this possibility, declined to separate the two individuals. In

Fig. 17.



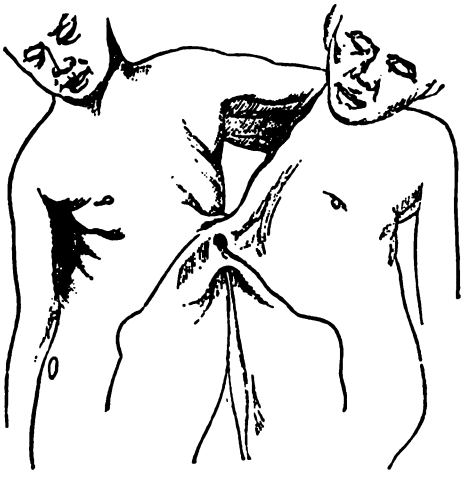
Craniopagus Parietalis (De Baer).

Fig. 18.



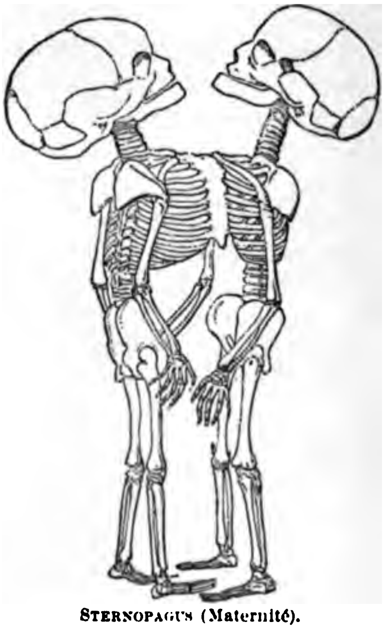
Craniopagus Parietalis.

Fig. 20.



The Siamese Twins (Xiphopagus). (From a plaster cast in the College of Physicians of Philadelphia.)

Fig. 21.



Sternopagus (Maternité).

two cases, in which the cavities of the abdomen did not communicate, the operation of division has been successfully performed. (Figs. 21, 22.)

Sternopagi have a common thoracic cavity, with a double or single sternum. Two of the upper extremities may be fused, when the variety is termed thoracopagus tribrachius; fusion of two lower extremities and the pelvis, thoracopagus tripus. In this, as in the other groups of double monsters, the parasitic form is also observed.

An example of thoracopagus parasiticus (sternopagus) is presented in the case of Laloo.[1] "The subject[2] is a lad from Oudh, aged seventeen, about five feet two inches in height, and of a very dark complexion. His expression is pleasing and intelligent, and his disposition very cheerful. There is no family history of any monstrosity. The mass, which appears to be of, at the most, very limited sensibility, is attached chiefly to the epigastric region. It consists of the structure forming the shoulder-girdle, including the integuments, which bear a pair of nipples; and of a second part, including the buttocks and lower extremities. The pubes is hairy, the penis well formed and its glans uncovered, urine occasionally passing from it. The anus appears to be imperforate. The arms are very long, like those of an American spider-monkey (Ateles); the buttocks form a projection rather bulkier than a cocoa-nut; the left foot hangs down nearly as low as the knee. Both extremities present numerous deformities. Next to the fact that there is a large parasitic fœtus dependent from the epigastrium, the most singular feature of the case is the complete separation of the shoulder-girdle from the lower parts of the parasite. The two parts appear to be separately united to the boy's trunk by freely movable joints; they are invested by a common integument and divided from each other by a deep groove." (Figs. 23, 24.)

About fifty cases of epigastrius have been recorded up to the present. The most famous case of all is that of Bartholin,—Lazaro Colloredo, of Genoa, born in 1716. This individual lived to manhood, with a highly-developed epigastrius, consisting of a head, trunk, arms, and one lower extremity. The face of the parasite had closed eyes and distinct ears and lips. The mouth bore teeth, saliva continually dribbled from it, and it did not take in any nourishment, yet it was said to breathe distinctly. A small beard grew from the parasite's face at puberty.

All double monsters confluent on their ventral surface, with the exception of a recently-described ischiopagus and a dipygus (Fig. 12), possess a common navel and a common umbilical cord. They are all accordingly included under the class of omphalopagi. The umbilical cord is commonly composed of two arteries and one vein; occasionally two veins are observed.

In the highest degree of duplication, as in the xiphopagi, the twins are viable and lead existences that are in a measure mutually independent. In the other forms, in which the bond of union is of greater extent,—thoracopagus, syncephalus, dicephalus,—important internal organs are confluent, and their development is disturbed so that the twins are either still-born or perish soon after birth. Only in isolated cases of ischiopagus, dipygus, dicephalus, has a birth-survival of a few weeks been observed.

---

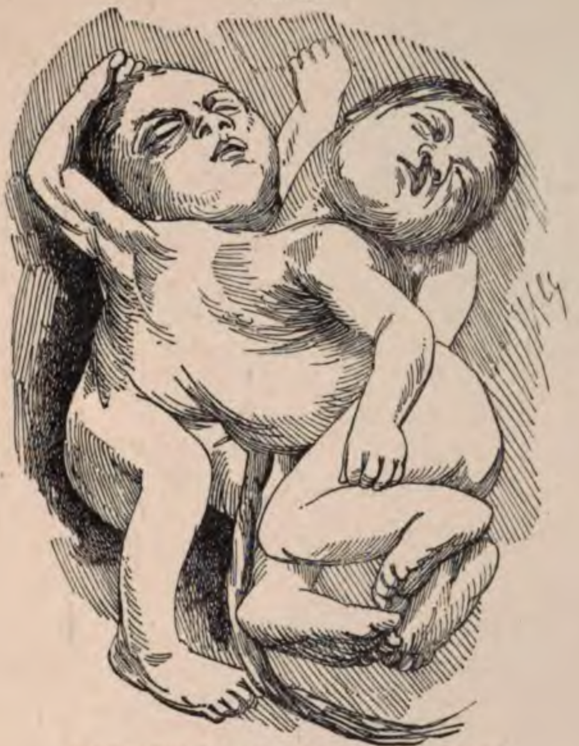
[1] Report to the Pathological Society of London, Messrs. Sutton and Shattuck, February 15, 1888.

[2] The British Medical Journal, February 11 and 25, 1888, pp. 312 and 436.

The development of the liver is found to be disturbed in the highest degree and in the largest number of cases. Thus, in thoracopagi, to which attention is restricted, the liver is always double. The jejunum and a portion of the ileum are single, while the remainder of the intestinal tract is double. In the thorax the lungs are double, while the heart shows various degrees of coalescence, from two separate hearts, each in its own pericardium, to a large, broad, single heart that shows signs of duplication in its interior.

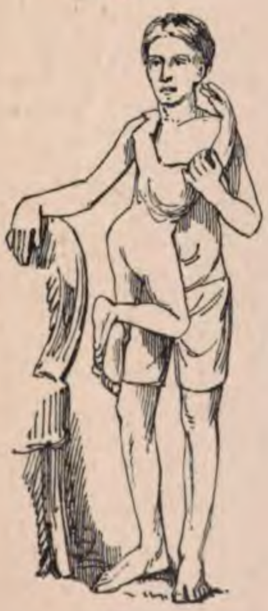
Literature.—FÖRSTER, Die Missbildungen des Menschen, Jena, 1865. PANUM, Untersuchungen über die Entstehung der Missbildungen, Berlin, 1860, und Virch. Arch., Bd. lxxii. AHLFELD, Arch. f. Gyn., Bd. ix., Die Missbildungen des Menschen, Leipzig, 1880 and 1882. MARCHAND, Eulenberg's Realencyclopaedie der ges. Heilkunde, art. Missbildungen. L. GERLACH, Sitzungsber. d. Phys.-Med. Soc. zu Erlangen, 1880, die Entstehungs-Weise der Doppelmissbildungen, Stuttgart, 1883. FOL, Recherches sur la Fécondation, etc., 1879. Lancereaux, Traité d'Anat. pathol., i., Paris, 1875–1877. Geoffroy St.-Hilaire, Hist. gén. et partic. des Anomalies de l'Organisation chez l'Homme et les Animaux, Paris, 1832–1837. DARESTE, Recherches sur la Production des Monstrosités, Compt.-rend. Acad. des Sciences, 1861, 1863, 1864, 1865, 1866. KORMANN, Ueber lebende Doppelmissbildungen der Neuzeit, Schmidt's Jahrb., cxliii., 1869.

FIG. 22.



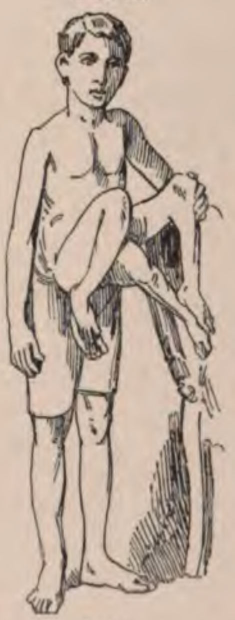
STERNOPAGUS (Herrgott). (Annales de Gynécologie.)

FIG. 23.



LALOO.

FIG. 24.



LALOO.

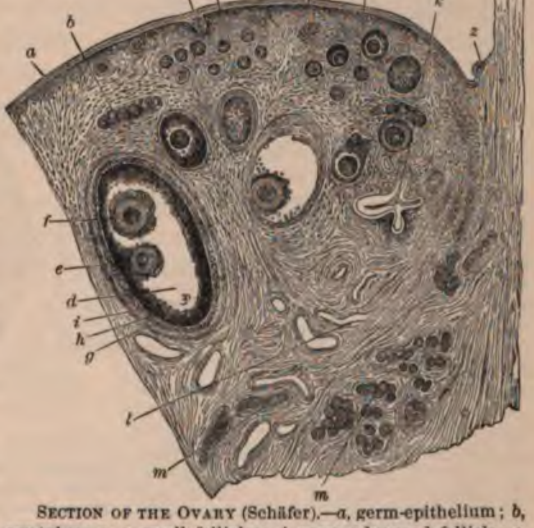
STERNOPAGUS PARASITICUS. (British Medical Journal, February 11 and 25, 1888.)

EMBRYOLOGY.[1]

By HORACE JAYNE, M.D.

MAN, in common with all Metazoa, is developed from an egg. This is formed and, for a time, nourished in the ovary. The ovary (Fig. 1) consists of a connective-tissue skeleton or stroma supporting the blood-vessels, lymphatics, and nerves. Within this framework are found the ova, contained in follicles or *Graafian vesicles*. The outer surface of the ovary is covered by a layer of columnar epithelial cells (*a*), and beneath this layer the stroma of the ovary is condensed to form a protective region, known as the albuginea, which does not contain any ova. Below this is found the *cortical layer*, which contains a large number of small Graafian vesicles with the diameter of about one-hundredth of an inch. These small follicles are especially numerous in the growing ovary (*c*, *c*). Below the cortical layer, the vesicles increase in size as they approach the highly-vascular centre of the organ, which is known as the *medullary substance;* here they reach the size of one-thirtieth of an inch (*d*). The ova appear at first as spherical cells lying between the columnar

FIG. 1.



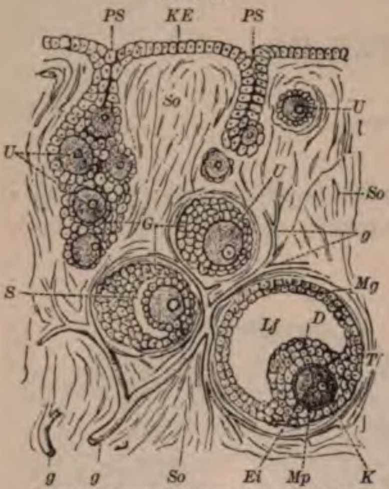
SECTION OF THE OVARY (Schäfer).—*a*, germ-epithelium; *b*, egg-tubes; *c*, *c*, small follicles; *d*, more advanced follicle; *e*, discus proligerus and ovum; *f*, second ovum in the same follicle (this occurs but rarely); *g*, outer tunic of the follicle; *h*, inner tunic; *i*, membrana granulosa; *k*, collapsed retrograded follicle; *l*, blood-vessels; *m*, *m*, longitudinal and transverse sections of tubes of the parovarium; *y*, involuted portion of the germ-epithelium of the surface; *z*, place of the transition from peritoneal to germinal or ovarian epithelium.

---

[1] The manuscript of this article was received too late for it to appear in the place originally assigned to it.

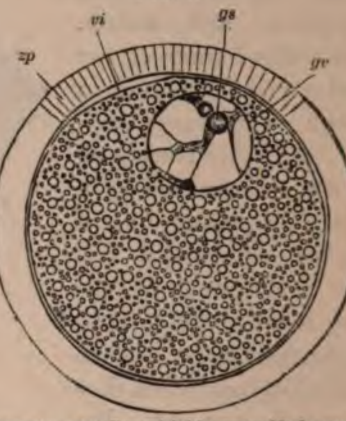
cells of the *germinal epithelium*, which covers the developing ovary (Fig. 2, *KE*). These spherical cells are carried down into the substance of the ovary in the lumen of tubular invaginations (*PS*) of the germinal epithelium, which thus forms the lining of the so-called ovarian tubes, which soon become closed and separated from the surface. Each of these spherical cells or *primordial ova* becomes invested by a capsule formed by the ingrowth of partitions into the tube from the stroma, and the entire capsule lined by the epithelium or *membrana granulosa* (*Mg*), derived, as we have seen, from the germinal epithelium, becomes the Graafian follicle (*Mg*). The follicle now enlarges and fluid begins to appear in the interior (*Lf*).

FIG. 2.



SECTION THROUGH A PORTION OF THE OVARY OF A MAMMAL: illustrating the development of the Graafian follicles (Wiedersheim).—*D*, discus proligerus; *Ei*, ripe ovum; *G*, follicular cells of germinal epithelium; *g*, *g*, blood-vessels; *K*, germinal vesicle (nucleus) and germinal spot (nucleolus); *KE*, germinal epithelium; *Lf*, liquor folliculi; *Mg*, membrana—or tunicata—granulosa or follicular epithelium; *Mp*, zona pellucida; *PS*, ingrowths from the germinal epithelium, ovarian tubes, by means of which some of the nests retain their connection with the epithelium; *S*, cavity which appears within the Graafian follicle; *So*, stroma of ovary; *Tf*, theca folliculi or capsule; *U*, primitive ova. When an ovum with its surrounding cells has become separated from a nest, it is known as a Graafian follicle. (From Haddon.)

FIG. 3.

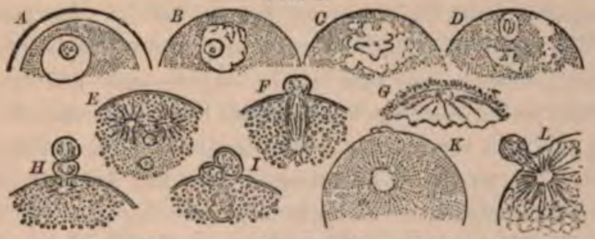


OVUM OF THE CAT: highly magnified; semidiagrammatic (after Schäfer).—*gs*, germinal spot; *gv*, germinal vesicle; *vi*, vitellus or protoplasm of ovum, filled with yolk-granules, round which a delicate membrane was seen; *zp*, zona pellucida (zona radiata); only a few radial pores are drawn.

The layer of lining cells or membrana granulosa is elevated at one part, to embrace and attach the ovum to the wall of the follicle. This growth of cells, which completely surrounds the ovum, is known as the *discus proligerus* (*D*). The ripening follicle, supported by a vascular layer from the ovary proper, the *theca folliculi* (*Tf*), sinks, at first, towards the centre of the ovary, but, when fully ripe and about to burst, rises to the surface, the intervening tissue becoming thinner and less vascular. The ovum in the follicle is nothing more than a typical undifferentiated cell. It possesses a thick, elastic cell-wall (Fig. 3, *zp*), called the *zona pellucida*, enclosing the protoplasmic cell-contents, known as the *vitellus* (*vi*), embedded in which is found the *nucleus* or *germinal vesicle* (*gv*), which in turn contains the *nucle-*

olus or *germinal spot* (*gs*). The cell-wall appears to be made up of at least two membranes: the external one, stout and perforated by innumerable fine pores, is the *zona radiata* or true zona pellucida (*zp*); the inner membrane is extremely delicate and lies in contact with the vitellus. The origin of these membranes is still a matter of much dispute. They are regarded by some as derivatives from the wall of the follicle, and by others as secretions from the ovum itself. The vitellus consists of protoplasm, contained in which can be seen so-called yolk-granules of albuminoid food-material. The amount of this yolk in the mammalian ovum is inconsiderable. In many of the lower animals, however, it is enormously developed, the protoplasm appearing only as a reticulum or supporting framework. The nucleus or germinal vesicle, which is about one five-hundredth to one six-hundred-and-twenty-fifth of an inch in diameter, resembles in structure the ovum itself. It contains in addition, however, a substance which, readily stained by certain reagents, has been called chromatin. In young developing ova this chromatin occurs as a long, contorted filament, but in the more mature the filament is condensed into a single or several spheres,—the nucleoli or germinal spots. Before fertilization the egg undergoes certain processes which are known as the *maturation of the ovum*. These changes begin by the nucleus abandoning its central position in the midst of the cell-contents (Fig. 4, *A*) and approaching one pole of the egg,—that pole where the upper layer of cells, or epiblast, will be developed. Here the nucleus apparently disappears or becomes diffused (*B*, *C*, *D*). At this point a swelling or bud (*F*) appears on the surface, formed partly of a portion of the nucleus and partly of the general cell-contents. A little later this bud is segmented off from the cell, but still lies under the cell-wall. The process is now repeated at the same point, and thus two particles (*H*) of cell-contents are cast out from the ovum, and are known from their position as the *polar cells*. These cells are not again used, and contribute nothing to the construction of the embryo. We do not know the meaning of these phenomena, but from studies on lower types of animals it would appear

FIG. 4.



Formation of Polar Cells in a Star-Fish (from Haddon, after Geddes, Fol, and Hertwig).—*A*, ripe ovum with eccentric germinal vesicle and spot; *B-D*, gradual metamorphosis of germinal vesicle and spot, as seen in the living egg, into two asters; *F*, formation of first polar cells and withdrawal of remaining part of nuclear spindle within ovum; *G*, surface view of living ovum and the first polar cell; *H*, completion of second polar cell; *I*, a later stage, showing the remaining internal half of the spindle in the form of two clear vesicles; *K*, ovum with two polar cells and radial striæ round female pronucleus, as seen in the living egg; *L*, expulsion of first polar cells.

that they are cast forth to make room for the male element, the spermatozoön. It has been shown that in certain lower forms the addition of the male element is not necessary to their subsequent development, which is thus termed asexual. The presence of a male element seems called for rather to add fresh energy to the cell-contents of the ovum than to supply something different from what is already there. When the protoplasm of the egg possesses sufficient vigor the development of the embryo may take place directly, but when the energy is small some of the weakened material appears to be cast out to make room for the new. The exclusion of the polar vesicles renders further development impossible unless the male element is supplied. The portion of the nucleus which is left after the formation of the polar cells again takes a central position in the ovum, and is known as the *female pronucleus.* The egg, when fully ripe and freed from the ovary by the rupture of its wall and the bursting of the follicle, is seized by the fimbriated extremity of the Fallopian tube, which has undergone erection for this purpose, and is fertilized by the spermatozoön.

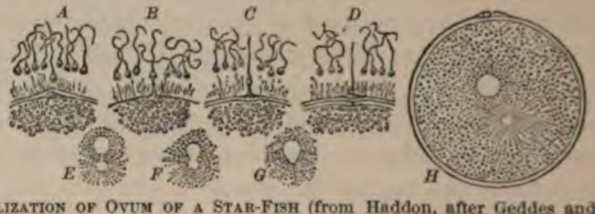
The spermatozoa are developed in the spermatic tubules of the testicles. These tubules are lined by a germ epithelium resting on a basement membrane containing flattened nuclei. This epithelial lining is now known to consist of four layers of cells which represent four generations of spermatozoa,—the undifferentiated cells lying in the outer or first layer, the fully-formed spermatozoa in the inner or fourth, and the intermediate stages between. We find three kinds of cells in the outer layer: (1) *supporting cells,* derived probably from the nuclei of the basement membrane, for they apparently never develop further, but, like the lining of the Graafian follicles, support and nourish the true sexual cell; (2) *growing cells;* and (3) *spore-cells.* The second layer consists of growing cells only, and the third of the young spermatozoa. The cycle of growth begins when the fully-ripe spermatozoa forming the inner layer are cast into the lumen of the tube. The spore-cells in the outer layer divide to form the growing cells and new spore-cells, which latter remain quiet until a new cycle sets in. The new growing cells push those already existing into the second layer, where they actively divide and are arranged in groups or clusters, and the groups of cells which formed the second layer now advance into the third layer as young spermatozoa, while the immature spermatozoa forming this third layer develop into the fully-ripe sexual elements of the inner layer. The supporting cells of the outer layer are at first stationary. When the clusters of growing cells have been formed in the second layer, however, they send prolongations up between them to meet and enclose the young spermatozoa lying in the third layer. As the spermatozoa increase in length the supporting cells again contract into the outer layer, so that the main portion of the spermatozoön, the part which will become the head, lies in this layer, while the long slender portion which develops into the cilium extends through to the inner layer. When the spermatozoa break loose, the supporting cells pass into a condition of rest until the next layer of young spermatozoa is ready for their support. In the development of the spermatozoa from the growing cells, we find that the nuclear membrane thickens at one side, the cell elongates, the nucleus occupying one pole, while a delicate thread is formed in the cell-protoplasm, connected at one end with the nucleus and projecting freely as a delicate lash from the opposite pole of the cell. The whole cell continues to increase in length, the nucleus at one pole, most of the rest of the cell-contents collected at the other in the form of a *globule*, the filament occupying the intermediate part and the cilium projecting freely beyond the globule. The nucleus thus forms the *head*, the general cell-protoplasm the *middle piece*, and by its growth outside the cell the cilium. Later the globule breaks free from the spermatozoön, and, like the polar vesicles of the ovum, appears to have no further function, but is found inert in the seminal fluid, as the *seminal granule*.

The spermatozoa of man are fifty micro-millimetres long, and consist of a head, a middle piece, and a delicate thread-like prolongation or tail. They are contained in the seminal fluid, a complex secretion from the cells of the testis proper, the glands of the vas deferens, the seminal vesicles, and Cowper's and the prostate gland. They possess the power of rapid movement for days, the movements appearing energetic at first and gradually becoming feebler. They are not affected by normal secretions of the female organs, but their movements are paralyzed by certain reagents and very acid or alkaline secretions. By virtue of their power of motion they are able to traverse the cavity of the uterus, enter the Fallopian tube, and make their way against the downward lashing motion of the cilia of the cells lining that canal. It is possible that they may be assisted by the peristaltic movements of the uterus and Fallopian tube.

The fertilization of the ovum may take place in the ovary, resulting in abdominal pregnancy. That it normally occurs in the Fallopian tubes is well settled; and if the ovum is thereafter unable to find its way to the uterus, tubal pregnancy ensues. It has been most positively stated by competent authority that fertilization in the uterus is impossible.

When a spermatozoön reaches the ovum (and it is almost certain that one only takes any direct part in the process of normal fertilization), it becomes attached by the head to the membrane (Fig. 5, *C*). In some cases it has been observed that the cell-contents of the ovum which have contracted away from the wall and more closely around the nucleus rise to meet the spermatozoön at the point of attachment (*A*). Whether there is an opening in the cell-wall, a so-called micropyle which is found in the eggs of some of the lower animals at this point, is not known. However this may be, one spermatozoön penetrates the membrane, the tail disappears, and the head, star-like in form and now called the *male pronucleus*, sinks to meet the female pronucleus (*E* and *H*) in its position near the centre. Here the two fuse together (*F*, *G*), forming the new spherical nucleus of the ovum enclosing a distinct nucleolus. This nucleus is to be distinguished as the *segmentation nucleus*, from the one possessed by the egg before fertilization, which alone should be termed the germinal vesicle. After fertilization the ovum divides or segments. This process appears to begin in the nucleus; it loses the spherical form and is converted into a spindle of fine fibres, at each pole of which the protoplasm of the cell is arranged in radiating lines. Since this spindle stains with difficulty, it has been termed the *achromatin* portion of the nucleus. That part of the nucleus which is known as *chromatin* collects around the centre of the spindle, probably first as a continuous looped fibre. The fibre breaks between the loops, giving a series of U-shaped rods arranged radially around the central circumference of the spindle, the points of the rods directed towards the periphery of the ovum. Each rod divides longitudinally to form two U-shaped loops, producing two stars, the *double star*, or *diaster*. The angles of the loops now become directed towards the opposite poles. The stars so formed travel along the spindle to the poles to form the *daughter stars*, which reverse the process just described, becoming wreaths and finally diffused as a delicate reticulum. The achromatin spindle disappears. When the daughter stars are formed, the cell-contents become constricted in a plane passing at right angles through the centre of the spindle, and the ovum has divided into two before the two new nuclei have reached a condition of rest. This process, which is known as *karyokinesis*, is common to many dividing tissue-cells.

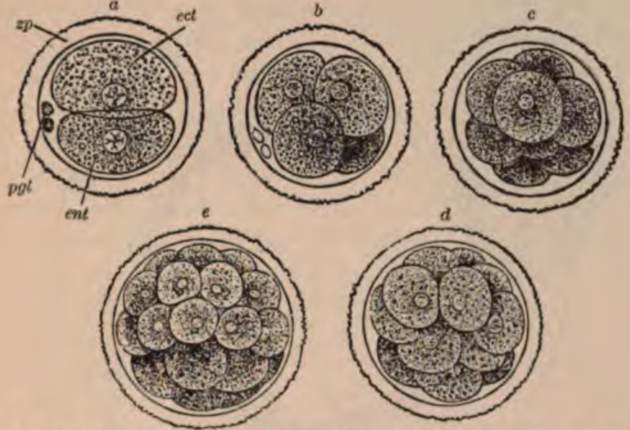
Fig. 5.



Fertilization of Ovum of a Star-Fish (from Haddon, after Geddes and Fol).—In *A–D* the spermatozoa are represented as embedded within the mucilaginous coat of the ovum. In *A* a small prominence is rising from the surface of the ovum towards the nearest spermatozoön, in *B* they have nearly met, and in *C* they have met. In *D* the spermatozoön has penetrated the ovum, and a vitelline membrane with a crater-like opening has been formed, which prevents the entrance of other spermatozoa. *H*, ovum showing polar cells and approach of the male and female pronuclei; the protoplasm is radially striated round the former. *E*, *F*, *G*, later stages in the coalescence of the two nuclei.

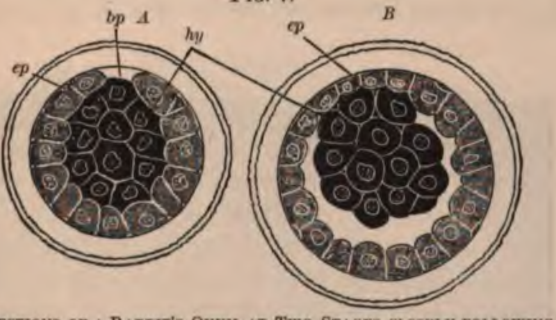
Each of the two cells thus produced (Fig. 6, *a*, *ent*, *ect*) now divides, the spindle being formed in the same plane as the former one, but at right angles to it. In this way four cells (*b*) are formed, each of which divides horizontally, the plane of segmentation intersecting the former plane at right angles (*c*). If the segmentation be perfectly regular, sixteen cells are now produced, then thirty-two, then sixty-four, and so on until a great number of small cells are found, the whole having a rounded form and known as the *morula* or *mulberry* stage (*e*). Segmentation is said to be completed in the human ovum by the end of the tenth day; and in the rabbit we know that by the end of segmentation the ovum has reached the uterus. On optical section this *blastosphere* is seen to be made up of an outer layer of smaller cells (Fig. 7, *ep*) surrounding a central group of larger, more granular cells (*hy*). The ovum now enlarges rapidly by the secretion within of fluid, and the outer layer of cells is forced away from those it surrounds, except at one spot, where the latter still adhere closely (Fig. 7, *B*). The blastosphere consists at this stage of one layer of cells, except at the points where there is also an inner lining (Fig. 8, *hy*). The double membrane thus formed is known as the *blastodermic membrane.*

FIG. 6.



FIRST STAGES OF SEGMENTATION OF A RABBIT'S OVUM: semidiagrammatic (from Quain).—*a*, two-cell stage; *b*, four-cell stage; *c*, eight-cell stage; *d*, *e*, later stages and the enclosure of the inner-layer cells; *ect*, outer-layer cells; *ent*, inner-layer cells; *pgl*, polar cells; *zp*, zona pellucida.

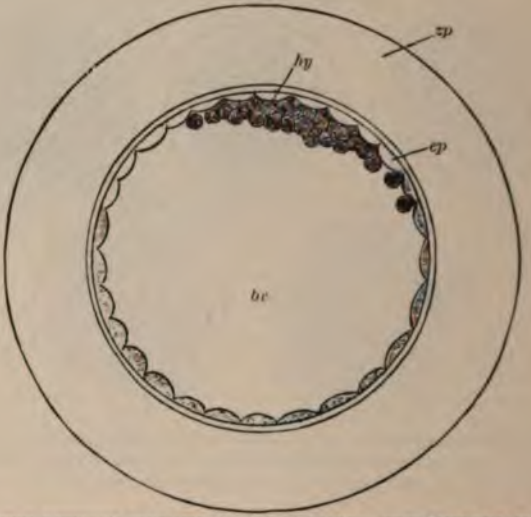
FIG. 7.



OPTICAL SECTIONS OF A RABBIT'S OVUM AT TWO STAGES CLOSELY FOLLOWING UPON SEGMENTATION (from Balfour, after E. van Beneden).—*ep*, epiblast; *hy*, primary hypoblast: the shading of the epiblast and hypoblast is diagrammatic.

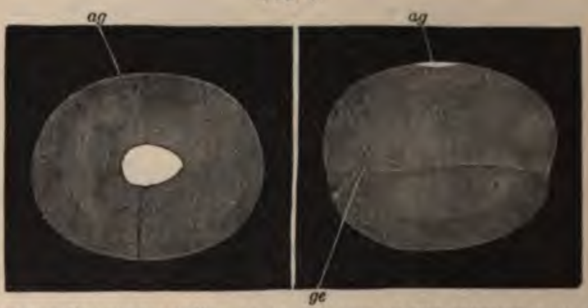
Its inner layer is known as the hypoblast; the outer is usually regarded as the *epiblast,* although it has been shown that in some ova this is merely a protective layer, the so-called *Deckschicht* of the German embryologist, which does not enter into the formation of the embryo, but thins away and disappears or becomes later fused with the enveloping membranes, the true epiblast developing from the inner cells. This inner layer of cells gradually spreads and extends downward, lining the outer layer, towards the lower pole of the egg. At the upper pole, where the hypoblast was first developed, there is seen a lens-shaped darker spot (Fig. 9, *ag*), for here the hypoblast consists of more than one layer: this is known as the *germinal disk* or *area germinativa*, and here alone the embryo develops.

FIG. 8.



RABBIT'S OVUM BETWEEN SEVENTY AND NINETY HOURS AFTER IMPREGNATION (from Balfour, after E. van Beneden).—*bv*, cavity of blastodermic vesicle (yolk-sac); *ep*, epiblast; *hy*, primitive hypoblast; *zp*, mucous envelope.

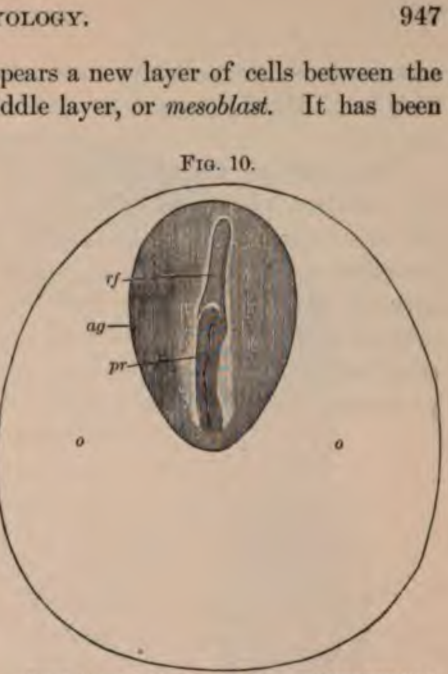
FIG. 9.



VIEWS OF THE BLASTODERMIC VESICLE OF A RABBIT ON THE SEVENTH DAY, without the zona: left-hand figure from above, right-hand figure from the side (from Balfour, after Kölliker).—*ag*, embryonic area; *ge*, boundary of the hypoblast.

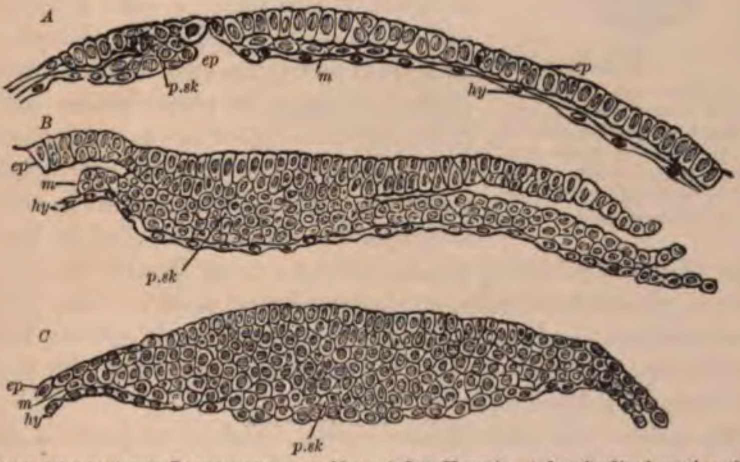
The first step in this process is marked by the formation at one end of the germinal disk, called the posterior, of a longitudinal thickening of the epiblast, in which is subsequently developed a groove. These are called respectively the *primitive trace* and the *primitive streak* (Fig. 10, *pr*). The anterior part of the area germinativa is translucent, and has been called the area pellucida, while the posterior part where the primitive trace is formed is opaque, because here there now appears a new layer of cells between the epiblast and the hypoblast,—the middle layer, or *mesoblast*. It has been shown, from the recent researches on development of the mole, that the mesoblast is developed as follows: the epiblast and hypoblast fuse in the region of the primitive streak, and from either side of the point where the two primary layers blend a mass of cells grows outward and also forward between the latter, so that the mesoblast may be said to arise on either side of the primitive trace or of lateral outgrowths of the fused epiblast and hypoblast (Fig. 11, *C*), and is therefore apparently derived from both. In front of the primitive trace it can be discovered split off on each side of the median line from the hypoblast (Fig. 11, *B*). After the formation of the mesoblast in the posterior part of the germinal area, there appear at the anterior end two parallel longitudinal thickenings of the epiblast, producing two closely-approximated ridges. These extend backwards and embrace the

FIG. 10.



EMBRYONIC AREA OF A SEVEN DAYS' EMBRYO RABBIT (Kölliker).—*o*, place of future area vasculosa; *rf*, medullary groove; *pr*, primitive streak; *ag*, embryonic area.

FIG. 11.

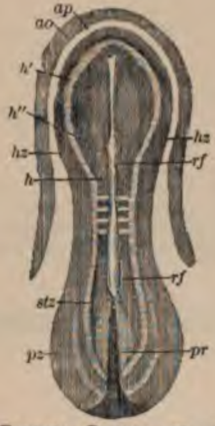


SECTIONS THROUGH THE BLASTODERM OF A MOLE (after Heape).—*A*, longitudinal section through the middle line of part of an embryonic area in which the primitive streak has commenced to form; the blastoderm is perforated in front of the primitive streak; *B*, transverse section through the middle of a well-developed primitive streak; the epiblast and mesoblast are fused, but the hypoblast is distinct; the mesoblast area extends beyond the embryonic area; *C*, same as *B*, but through the hind knob of the primitive streak. All the layers are fused in the embryonic area, but are distinct beyond. *ep*, epiblast; *hy*, hypoblast; *m*, mesoblast; *p.sk*, primitive streak.

anterior end of the primitive trace, which gradually disappears (Fig. 10, *rf*). This longitudinal groove in the epiblast is called the medullary groove (Fig. 10, *rf*).

The epiblastic ridges on each side of the groove grow upward to form the medullary plates, or *laminæ dorsales*, which, considerably later, arch over the groove and meet in the middle line to form a canal,—the *neural* or *medullary canal.* This tube, consisting of a layer several cells deep, becomes detached from the epiblast, and lies beneath and disconnected from the original epiblastic stratum which grows over it. The closure of the neural canal begins some little distance behind the head end of the embryo and progresses forward and backward. While the neural groove is developing, the axial mesoblast has been formed as two separate masses lying on each side of the median line, where epiblast and hypoblast are in contact (Fig. 12, *n*). A new structure has also arisen, in the shape of a rod of cells split off dorsally from the hypoblast and lying under the neural canal and between the lateral masses of the mesoblast. This is the *notochord*, and, although in the higher vertebrates it does not contribute to the actual formation of the axial skeleton, it is the axis around which the centra or bodies of the vertebræ are subsequently developed.

FIG. 12.



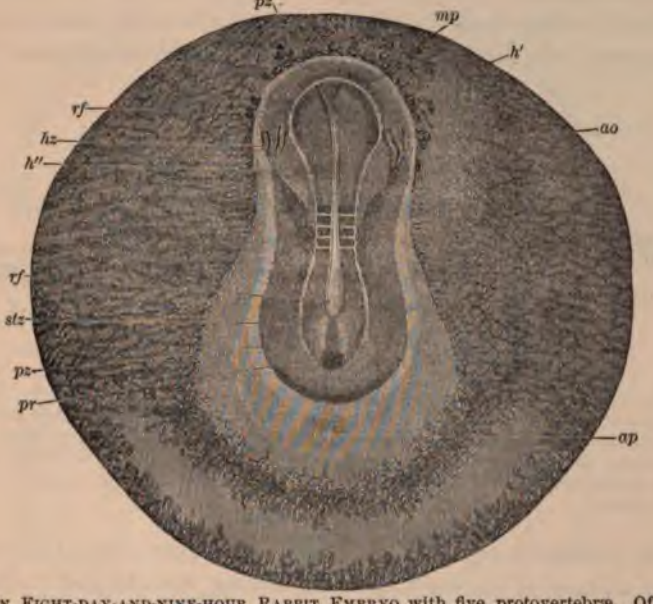
EMBRYO RABBIT OF EIGHT DAYS AND NINE HOURS, with five protovertebræ. × 18. (From Kölliker.)—*ao*, area opaca; *ap*, area pellucida; *mp*, medullary plate of head; *h'*, region of future fore-brain; *h''*, region of future mid-brain; *rf*, medullary groove; *hz*, heart; *pr*, primitive streak.

The lateral masses of the mesoblast are seen to consist of two parts: a plate lying on each side of the neural canal and notochord, which, later, splits up by transverse division into cubical blocks,—the so-called protovertebræ; and a lateral portion formed of two layers connected together near the protovertebræ but peripherally separated; the one layer applied to the under surface of the epiblast, the other to the upper surface of the hypoblast (Fig. 14), leaving a cavity between them which is known as the cœlum or body, or pleuroperitoneal cavity. The upper layer of the mesoblast is called the somatic layer, and with the epiblast forms the somatopleure; the lower layer is the splanchnic layer, and with the hypoblast forms the splanchnopleure.

The wall of the blastodermic vesicle, which is enclosed in the zona radiata, now consists of the embryo surrounded by a translucent ring,—the area pellucida (Fig. 13, *ap*),—and, beyond this, by a dark ring,—the area opaca,—in which the epiblast is thickened, the mesoblast is well developed, and the blood-vessels begin to appear. At the opposite pole of the vesicle the wall comprises only the two primary layers.

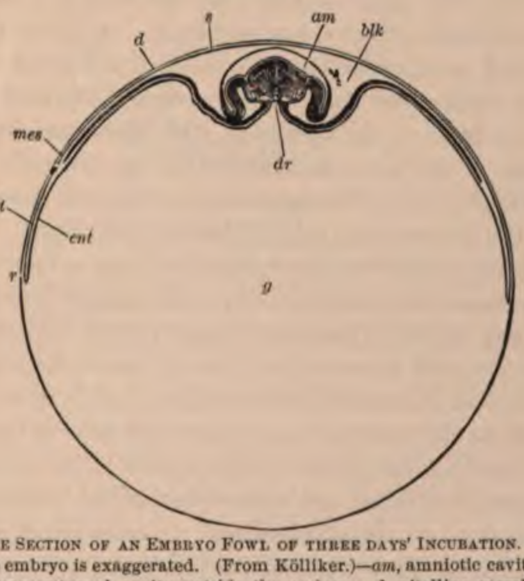
We have thus far been regarding the embryo as lying flat upon the surface of the blastodermic vesicle; but very early in the development, be-

FIG. 13.



AN EIGHT-DAY-AND-NINE-HOUR RABBIT EMBRYO with five protovertebræ. Of the blastodermic vesicle, only the area opaca is shown. × 14. (From Kölliker.)—*ao*, area opaca or vasculosa; *ap*, area pellucida; *mp*, medullary plate of head; *h'*, *h''*, region of future brain; *hz*, rudiments of heart; *pr*, primitive streak; *stz*, axial zone; *pz*, parietal zone.

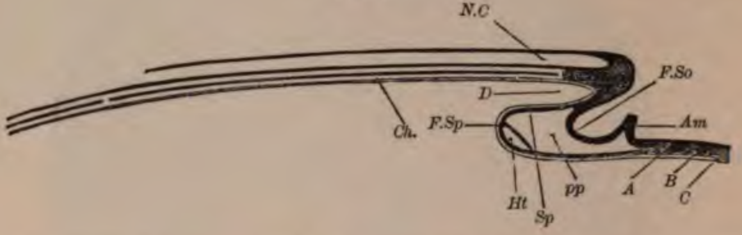
FIG. 14.



TRANSVERSE SECTION OF AN EMBRYO FOWL OF THREE DAYS' INCUBATION. The relative size of the embryo is exaggerated. (From Kölliker.)—*am*, amniotic cavity; *blk*, extension of pleuroperitoneal cavity outside the embryo; *d*, vitelline membrane; *dr*, intestinal groove; *ect*, epiblast; *ent*, hypoblast; *g*, yolk; *mes*, border of the splanchnic mesoblast (area vasculosa); *r*, edge of the blastoderm, here consisting only of epiblast and hypoblast; *s*, serous or subzonal membrane or false amnion.

fore, indeed, the neural groove is converted into a canal, it becomes folded off from the vesicle. This is brought about, at first at the anterior end, by the embryo growing more rapidly than the surrounding embryonic area, so that it projects over the now underlying blastodermic membrane. (Fig. 15.)

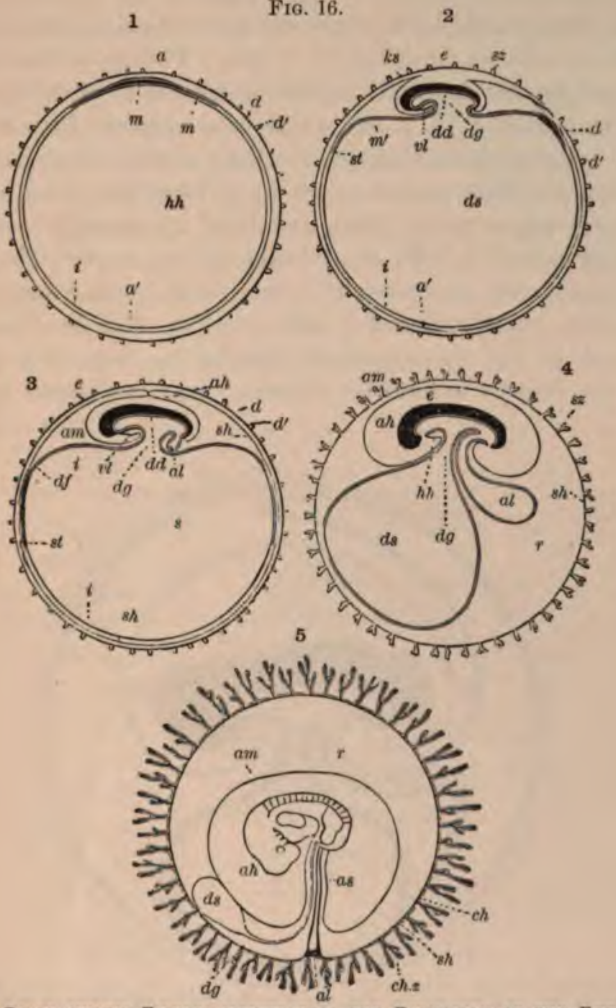
FIG. 15.



DIAGRAMMATIC LONGITUDINAL SECTION THROUGH THE AXIS OF AN EMBRYO (Balfour). The section is supposed to be made at a time when the head-fold has commenced but the tail-fold has not yet appeared.—*F.So*, fold of somatopleure; *F.Sp*, fold of splanchnopleure; *D*, fore-gut; *pp*, pleuroperitoneal cavity between somatopleure and splanchnopleure; *Am*, commencing (head) fold of amnion; *N.C*, neural canal; *Ch*, notochord; *A,B,C*, epi-, meso-, and hypoblast.

The same process is repeated posteriorly, and gives rise to the appearance of the membrane at the head and tail having been tucked under the embryo proper. Cavities are thus formed in the embryo in front and behind (Fig. 15, *D*) which are still in communication with the general cavity of the vesicle, and which will become the anterior and posterior ends of the intestinal canal. The elevation of the embryo above the general surface is further accomplished by the growing down of the sides or laminæ ventrales (Fig. 14). The first traces of the heart are seen as a tube on each side, formed by involution of the splanchnopleure. As the sides of the embryo grow down and together underneath, the two halves of the heart become united in the middle line and form a median undivided organ. On each side, the lower layer of the mesoblast with its ventral lining of hypoblast curves downward and meets its fellow of the opposite side to form the intestinal tract (Fig. 17, *H*). The region of the intestinal tract at the middle of the body of the embryo does not close below until later. The upper layers of the mesoderm covered by the epiblast do not, at first, meet underneath, so that the cœlum or body-cavity is in open communication with the space between the two layers of mesoblast which extend beyond the embryo laterally, anteriorly, and posteriorly. Pressure upon the embryo by the surrounding maternal structures has caused it to sink down into the blastodermic vesicle as the latter grows in size, and there is thus formed, at first in front, where the head of the embryo begins to be bent downward in the cephalic flexure, an external and upward fold of the blastodermic membrane. This fold is formed of the epiblast and its underlying somatic mesoblast. A similar fold then appears behind the tail, and as the embryo sinks deeper and deeper into the vesicle the two folds rise higher at the ends (Fig. 16, 2), while lateral folds arise on either side until they all meet above the embryo.

FIG. 16.



FIVE DIAGRAMMATIC FIGURES ILLUSTRATING THE FORMATION OF THE FŒTAL MEMBRANES OF A MAMMAL (from Haddon, after Kölliker).

In 1, 2, 3, 4, the embryo is represented in longitudinal section.

1. Oosperm with zona pellucida, blastodermic vesicle, and embryonic area. 2. Oosperm with commencing formation of umbilical vesicle and amnion. 3. Oosperm with amnion about to close and commencing allantois. 4. Oosperm with villous subzonal membrane, larger allantois, and mouth and anus. 5. Oosperm in which the vascular mesoblast of the allantois has extended round the inner surface of the subzonal membrane, and united with it to form the chorion; the cavity of the allantois is aborted. The yolk-sac (umbilical vesicle) has greatly diminished. The large amniotic cavity surrounds the umbilical cord. This figure represents an early human ovum.

a, epiblast of embryo; *a'*, epiblast of non-embryonic part of the blastodermic vesicle; *ah*, cavity of the amnion; *al*, allantois; *am*, amnion; *as*, amniotic sheath round the umbilical cord; *ch*, chorion; *ch.z*, villi of chorion; *d*, zona pellucida (radiata); *d'*, processes of zona; *dd*, embryonic hypoblast; *df*, area vasculosa; *dg*, stalk of yolk-sac; *ds*, yolk-sac (umbilical vesicle); *e*, embryo; *hh*, pericardial cavity; *i*, non-embryonic hypoblast; *kh*, cavity of the blastodermic vesicle, which practically is equivalent to the yolk-sac; *ks*, head-fold of amnion; *m*, embryonic, *m'*, non-embryonic, mesoblast; *r*, space between chorion and amnion containing albuminous fluid; *sh*, subzonal (serous) membrane; *st*, sinus terminalis; *sz*, subzonal villi; *vl*, ventral body-wall in the region of the heart.

The lower sheets of the fold, consisting of epiblast and mesoblast, fuse on the middle line just over the back of the embryo to form a sac surrounding the embryo above,—the *amnion* (Fig. 16, 2, *am*). The upper sheets of the fold also fuse and become detached from the amnion to form the *false amnion*,[1] or *serous envelope* (Fig. 16, 3, *sh*), which, as can be seen from the figures, is continuous below with the epiblast covering the whole blastodermic vesicle.

The amnion is thus covered externally by mesoblast, which subsequently is said to develop muscular fibres, and lined by epiblast, in more or less intimate contact, at first, with the outside epiblast of the embryo, while in the false amnion the mesoblast, if it here really reaches out so far from the body of the embryo, lines the epiblast. The other or splanchnic layer of the mesoblast and the hypoblast, after having been, as it were, tucked in under the embryo to form the intestinal tract, are spread out (Fig. 17) again over the contents of the vesicle, to form the yolk-sac (Fig. 16, *ds*). The whole ovum at this stage, therefore, consists of two sacs lying within the zona pellucida or vitelline membrane. The upper sac contains the embryo, the lower the yolk, and the body of the embryo forms the septum between the two sacs. The yolk-sac communicates with the intestinal canal within the embryo by means of its upper constricted part, the *omphalo-mesenteric* or *vitello-intestinal* duct, which will later be reduced to a tube by the complete ingrowth of the somatopleure in the form of laminæ

FIG. 17.

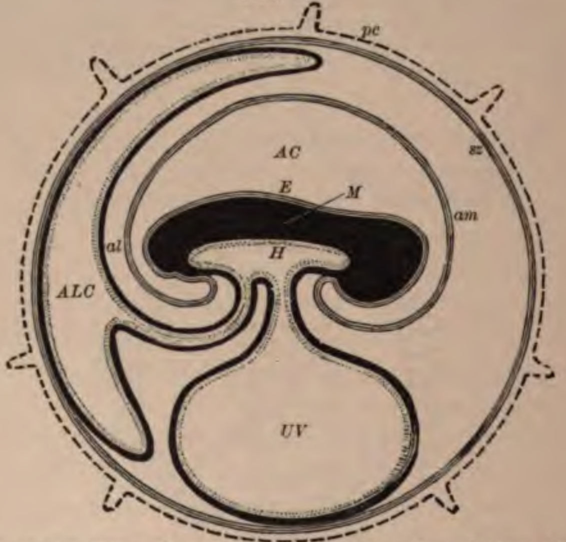


DIAGRAM OF THE FŒTAL MEMBRANES OF A MAMMAL (from Balfour, after Turner). Structures which either are, or have been at an earlier period of development, continuous with each other are represented by the same character of shading.—*pc*, zona with villi; *sz*, subzonal membrane; *E*, epiblast of embryo; *am*, amnion; *AC*, amniotic cavity; *M*, mesoblast of embryo; *H*, hypoblast of embryo; *UV*, umbilical vesicle; *al*, allantois; *ALC*, allantoic cavity.

---

[1] The false amnion of Kölliker and Von Baer is not the same as that of some later writers.

ventrales. The yolk-sac represents the blastodermic vesicle less that portion which has been included within the embryo to form the intestine by this folding-in process.

As each sac is covered by mesoblast, the space between the sacs is a direct continuation, outside, of the space which lies between the two layers within the embryo, the cœlum or body-cavity, but by the subsequent growing together of the laminæ ventrales the embryonic body-cavity is shut off from the non-embryonic. The amnion, at first small and in contact with the dorsum of the embryo, begins to grow rapidly (at the fourth or fifth week) by the accumulation within the amniotic fluid (Fig. 16, *ah*). This increases until about the sixth month, after which it diminishes. In the middle of pregnancy it weighs from two to three pounds; by the end of that period it is reduced to one pound. The nutritive function of the yolk-sac in higher mammals is not important. The yolk, which appears in the form of granules, is absorbed by the hypoblast cells and by blood-vessels which have already made their appearance in the peripheral splanchnic mesoblast, in that opaque portion which is called the area opaca or vasculosa. The vessels coming from the embryo traverse the walls of the omphalo-mesenteric duct, spread out on the surface of the yolk-sac, and terminate peripherally in a circular vessel, the *sinus terminalis* (Fig. 18, *S.T*) in the margin of the mesoblast covering the yolk-sac. From this area the blood is returned to the heart of the embryo by two long trunks (the omphalo-mesenteric veins (Fig. 18, *R.of*, *L.of*). The ovum, almost immediately upon its entrance into the uterus, becomes attached to the uterine wall. This union is at first superficial, the outer membrane enclosing the egg being covered with hollow finger-like processes or villi; but very soon a new and permanent structure arises from the embryo by which this connection is rendered more intimate. This organ is called the *allantois*.

FIG. 18.

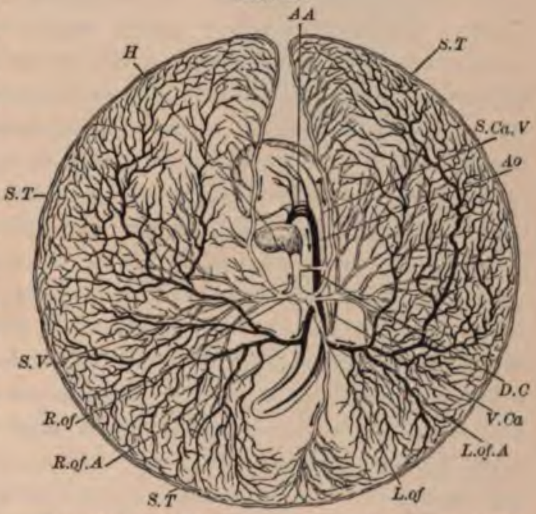
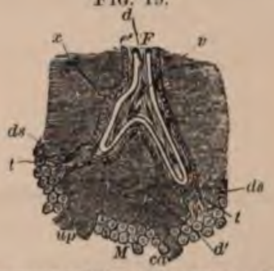


DIAGRAM OF THE CIRCULATION OF THE YOLK-SAC OF CHICK AT THE END OF THE THIRD DAY OF INCUBATION (Balfour).—*H*, heart; *AA*, the second, third, and fourth aortic arches; the first has become obliterated in its median portion, but is continued at its proximal end as the external carotid, and at its distal end as the internal carotid; *Ao*, dorsal aorta; *L.of.A*, left vitelline artery; *R.of..A*, right vitelline artery; *S.T*, sinus terminalis; *L.of*, left vitelline vein; *R.of*, right vitelline vein; *S.V*, sinus verosus; *D.C*, ductus Cuvieri; *S.Ca.V*, superior cardinal vein; *V.Ca*, inferior cardinal vein. The veins are marked in outline and the arteries are black. The whole blastoderma has been removed from the egg, and is supposed to be viewed from below. Hence the left is seen on the right, and *vice versa*.

The origin of the allantois in man appears to be peculiar, and its development is precocious and abbreviated. Whatever may be the details of its early appearance (and we are still in doubt, since few observations are recorded of such early ova), this much appears settled from the study of the lower forms: The allantois arises first as a bud, in the second week, from the caudal part of the primitive intestinal canal (Fig. 16, 3, *al*). This bud grows forward within the body-cavity as a hollow sac, and passes out from the embryo into the space between amnion and yolk-sacs (Fig. 16, 4, *al*). As it is derived from the intestinal canal, it consists of two layers, internally of hypoblast, externally of splanchnic mesoblast. We know that in many forms the cavity in the allantois persists for a long time, but it is almost certain that in man the hypoblast does not extend into the organ much beyond the embryo, and that the allantois, in the space between amnion and yolk-sac, consists of a nearly solid organ formed of mesoblastic tissue. The allantois grows rapidly in this space and reaches the vitelline membrane of the ovum, which, it will be remembered, now consists of the epiblastic false amnion, called also the subzonal membrane, within, and of the zona radiata without: there being no mesoblast, since the somatic layer thinned out in covering the amnion and the splanchnic layer has been split off from the primitive epiblast of the vesicle (now the false amnion) and covers the umbilical vesicle or yolk-sac. The appearance of the allantois antedates the complete folding off of the embryo from the blastodermic vesicle, and even the development of the amnion. The allantois increases in size (Fig. 17, *al*), and at the fourth week forms a complete lining to the subzonal membrane (Fig. 16, 5). The union of allantois with subzonal membrane forms the organ known as the *chorion* (Fig. 16, 5, *ch*). As we have seen, at a very early period in its development, in the first weeks, the ovum becomes covered with hollow villous outgrowths from the enveloping membrane. In the primitive attachment of the ovum to the uterine wall, the mucous membrane of the latter, which has undergone preparatory changes, grows around and encloses it in a distinct capsule. The mucous membrane is therefore divided into the part which covers the ovum, the *decidua reflexa*, and the part lining the general uterine cavity, the *decidua vera*. When the allantois lines the epiblastic layer or false amnion, it sends mesoblastic prolongations into the villi and develops a rich net-work of blood-vessels in them. This net-work is supplied by blood from the aorta of the embryo through the arteries arising in the mesoblastic tissue of the allantois, and returns the blood through two veins which join the omphalo-mesenteric or vitelline veins coming from the yolk-sac. As the part of the allantois which remains within the embryo, that part which is lined by hypoblast, will become later the urinary bladder, the arteries after birth are limited to the supply of that organ and are known as the hypogastric vessels. At first the chorion and decidua are everywhere vascular, but later the villi with their vessels disappear, and the decidua reflexa becomes gradually converted into a yellow membrane except at a disk-shaped patch where the capsule of the ovum becomes continuous with the uterine wall,—in other words, where the decidua reflexa joins the decidua vera. The decidua at this point is called the *decidua serotina*. Here the placenta is developed. It consists of a maternal part, the thickened mucous membrane of the uterus (the decidua serotina), and a fœtal part derived from the chorion, comprising the greatly developed mesoblastic allantois and the epithelial layer of the false amnion, or subzonal membrane. The zona radiata has been absorbed. The villi of the chorion become gradually enlarged to form tufts (Fig. 19), known as *cotyledons*, which project into great blood-sinuses formed in the decidua serotina; but, as the outermost epithelial layers of the fœtal and maternal tissues are interposed, the vessels are not in open communication, and interchange of materials takes place only by osmosis (Fig. 19). So intimate is the connection between the two parts of the placenta that upon birth the placenta as a whole, including portions of maternal tissue, leaves the uterine wall, and is therefore classed as deciduate. In many mammals this union is not so complete, and the line of separation is between the parts of the placenta, the fœtal portion only being cast off, the maternal remaining. Nor is the placenta always a compact structure, but in some animals forms a zone around the ovum and in others is diffused, the villi persisting all over the chorion. Or the cotyledons may be developed in patches, the decidua reflexa retaining its vascularity at these points. The amnion has now grown much faster than the embryo, and occupies all the space within the chorion, from which it is separated by a layer of gelatinous substance, and further covers as a sheath the stalks of both allantois and umbilical vesicle. The cord thus formed is known as the *umbilical cord*, and comprises, at first, splanchnic mesoblast containing the vessels from the umbilical sac and the vessels for the allantois, and the sheath from the amnion, which is made up within of somatic mesoblast, and without of epiblast. Later the vessels from the yolk-sac and one allantoic vein disappear, and the mesoblast derived from the amnion becomes converted into the jelly of Wharton, binding together the vein and the two arteries spirally wound around it. As the fœtus grows and the amniotic sac fills the entire cavity of the uterus, the space between the decidua reflexa and the decidua vera gradually disappears and these two layers become united.

FIG. 19.



STRUCTURE OF HUMAN PLACENTA.—*F*, fœtal, *M*, the maternal placenta; e', epithelium of maternal placenta; *d*, fœtal blood-vessels; d', maternal blood-vessels; *v*, villus; *ds*, *ds*, represents the decidua serotina of the placenta; *t*, *t*, trabeculæ of serotina passing to the fœtal villi; *ca*, curling artery; *up*, utero-placental vein; *x*, a prolongation of maternal tissue on the exterior of the villus outside the cellular layer e', which may represent either the endothelium of the maternal blood-vessel or delicate connective tissue belonging to the serotina, or both. The layer e' represents maternal cells derived from the serotina. The layer of fœtal epithelium cannot be seen on the villi of the fully-formed human placenta.

THE GENERAL THERAPEUTICS OF CHILDREN'S DISEASES.

By ROBERTS BARTHOLOW, M.D., LL.D.

PRELIMINARY.

Remedies, their Preparation and Classification.—Nothing can be more desirable than an improvement in the degree of certainty with which our therapeutical measures are employed. That greater precision in the use of drugs now obtains than formerly, is because of advances in pharmaceutical chemistry, whereby more powerful agents, whose physiological actions are accurately ascertained, can be substituted for the cruder preparations, formerly the only kind available for use. There are still greater changes in the armamentarium for children, that should be made. Whatever hold homœopathy has acquired is due for the most part to the sugar pellets, tasteless solutions, and other contrivances for rendering the administration of medicines a pleasure rather than a pain. The contests with children which the giving of nauseous medicines provokes add not a little to the hardships of parents. Our efforts should be directed to the preparation of efficient remedies that shall be, also, without disagreeable qualities. It is not by "elegant prescribing," so called, that this desirable result can be reached. In elegant prescribing, the power of a given combination is usually subordinated to the question of a pleasing taste. Attention to such details may make a good medical confectioner, but hardly a scientific medical practitioner. Power in action and facility in administration can be combined without descending to such trivialities.

Keeping in view the necessity of the case as above outlined, the conditions to be complied with are—

1. The use of concentrated preparations of medicaments.
2. Their arrangement in such form as to secure facility of administration and precision in action.

The use of remedies in the treatment of diseases of children and of adults, to have system and coherence, must be based on a true conception of their modes of acting. Classification becomes necessary if the facts ascertained are to be utilized with success. Although any classification must be defective because of our incomplete knowledge, we are now in a position to arrange remedies with some degree of scientific precision.

The obvious differences between those remedies which affect only the part that they are applied to, and those affecting the whole system, naturally divide them into two classes,—LOCAL REMEDIES: SYSTEMIC REMEDIES.

As remedies may increase or lessen function, only, and not, in the normal state of things, change its character, we have as regards systemic remedies *those that improve and those that retard nutrition.* As the function of nutrition is concerned with every tissue of the body, the classes just given are general in scope. The condition of the system as a whole organism is also affected by certain agencies from without, especially by organic substances and micro-organisms. Hence of general remedies we have three great classes:

1. *Those that improve nutrition and increase tissue-metamorphosis;*
2. *Those that retard nutrition and increase waste;*
3. *Antiseptics,—remedies destructive of micro-organisms.*

As the various functions may be affected individually by certain systemic medicines, we have classes thus limited to the function or functions of organs.

As the most widely connected, the diseases of the nervous system should occupy the first place. Here also the two modes of acting on function possible to drugs are perfectly well exhibited. One group increases, another lessens function: hence—

Excito-motor;
Depresso-motor;
Increasing action in the sensory area;
Lessening action in the sensory area.

In these several groups or classes we may readily include all remedies affecting the nervous system, and, through the nerves, other functions. But there yet remain for distribution unassigned remedies affecting some other functions. These are three: *the intestinal canal; the broncho-pulmonary mucous membrane;* and *the genito-urinary apparatus.*

Of the first, or the intestinal canal, are—

Emetics,
Cathartics,
Anthelmintics.

Of the broncho-pulmonary mucous membrane, are—

Inhalations,
  Vapors,
  Spray,
Douches, etc.

Of the genital and renal, are—

Diuretics,
Emmenagogues.

When we come to inquire whether there be any special mode in which to arrive at a safe conclusion regarding the utility of any remedy, it must be said that it can be based, only, on its physiological actions. Empiricism, or the information obtained by observation and experience, may serve until a more certain guide—the proper study of physiological actions—is had. Clinical experience must confirm the deductions of physiological examination, to make the evidence complete.

In the absence of more precise data, it can be said that all medicines acting on a tissue, an organ, or a system of the body, are influential, often curative, in the maladies affecting these several parts or organs. We can formulate a law, therefore, that has an enormous experience to support it: *In proportion as the remedy antagonizes the morbid action in a part is it curative;* or, stated conversely, *The more nearly the action of the remedy harmonizes or corresponds to the morbid action, the less it has of a curative action.*

We may illustrate these postulates by atropine and pilocarpine. Pilocarpine is the most powerful of sudorifics, or sweat-producing medicines, we possess; but in the treatment of morbid sweating it increases the action of the sweat-glands, or it opposes it feebly and inadequately, or it has no apparent effect. On the other hand, atropine, which most directly and powerfully restrains sweating, is, in states of disease, the most efficient of remedies to stop sweating. Many other examples could be quoted if it were necessary to do so.

Unfortunately, there are many remedies of whose curative power we can have no prevision except such deductions as may be derived from the results of observation and experience. Of such we are compelled to base our therapeutical conceptions on the old empirical formula: *A remedy that has cured one case must, also, cure analogous cases.*

There are many reasons, into which we need not enter, for doubting the general applicability of such a formula, but, in the absence of all other aids to a right conception, it may serve a temporary purpose.

Many approved methods of treatment which, in the case of adults, are much employed, cannot be in children because of difference in age, insusceptibility to impressions, tenderness of the tissues, and rapidity of the nutritive processes.

On the other hand, in consequence of the great activity of tissue-metamorphosis, some modes of procuring medicinal action not effective in the case of adults become highly so in children.

As an example of the greater activity of a tissue in children that increases the proportionate effects of medicinal application, the skin may be mentioned. Thus, digitalis moistened and enclosed in thin muslin and covered with oiled silk will, after some hours, begin to act in its most characteristic manner. Other medicaments that have active diffusion-power can be taken into the system in the same way, when the condition of the stomach and the nature of the remedy may make such a mode of entering the vessels feasible, in the maladies of early life.

The arrangement of the facts and principles of general therapeutics as applicable to children can be best presented under the head of the various modes in which medicaments are introduced into the body. The relation of the facts to each other, and to the scheme of classification already set forth, will be stated under each.

TREATMENT BY THE SKIN:

Enepidermatic; Epidermatic; Endermatic; Hypodermatic.

Inunctions.—In suitable cases inunction is an effective mode of treatment. Careful and persevering friction with oils and fats will improve the body-weight of children, sometimes in a remarkable degree. This practice is the more effective the thinner the skin and the more active the circulation. The absorption of fats is facilitated by a preliminary warm bath. This is best given at the bed-hour. The child should sit for a few minutes in the bath, the water at 90° to 100° F. according to circumstances. When taken out after one or two minutes, the skin should be wiped dry and gently rubbed with a rough towel. The inunction of the fat should be made immediately. The whole process should not occupy more than five to ten minutes, and usually five will suffice.

The fat should be animal fat, and not cacao butter, olive oil, cotton-seed oil, and similar vegetable productions. Benzoinated lard and benzoinated mutton suet are the best forms of fats for inunction, the addition of the benzoin being useful in improving the odor and preventing decomposition of the fat. By systematic application of fats, very great improvement in the nutrition of children, and with comparative facility, is made. Patience and thoroughness are necessary qualifications for those who undertake this plan of treatment; and, if the operator be gifted with these attributes, results worthy of the time and toil expended will be secured.

When the body is wasted, the skin dry and furfuraceous, the temperature at normal, or above, inunctions of fats contribute to the removal of the morbid state causing these signs.

Fat-inunctions are *antipyretic* to a sensible extent. In the *eruptive fevers* they act in two modes: allaying the irritation in the skin, and lowering the temperature. It is probable the latter is chiefly a result of the influence of the former over the combustion-process. For if the irritation of the skin is removed, it follows that the effect of this in exciting the circulation disappears. The heart acting less quickly, the vaso-motor tone is increased, and hence less blood passes through the tissues, and oxidation declines, in a corresponding ratio. In all fevers where decline in heat is necessary to safety, this procedure may be employed without risk.

In using fat-inunctions in fevers it is implied that the skin is kept in good condition by subsequent sponging with an alkaline solution,—sodium carbonate, especially.

Sponging with alkaline solutions is a highly useful measure in nervous diseases of irritation. Children in high fever, with twitching of muscles and tendons, partial or general convulsions threatened, are suitable subjects for this kind of medication. A saturated solution of sodium carbonate in rain-water or clean river- or spring-water should be sponged over the whole surface of the body, and as often as the case may require,—for a case of slow progress and long on hand, twice a day, but in acute cases with high fever, every two, three, or four hours, *pro re nata.* The cooling effect of the evaporation of water, due to a transferring of the body-heat into another mode of motion, is an antipyretic action, and is quite apart from the results of the alkaline reaction on the diffusion process.

Inunction of remedies in the form of ointments for specific effects is an old method that has been recently revived,—the Vienna school having been most active in bringing about the revival. In children the hereditary or acquired specific lesions are most readily and effectively treated in this way. The preparation most uniformly useful is the *oleate.* A small bolus of this, the size of a large pea, is put well up into the axilla, and the movements of the arm by the child rub it into the skin. Mercurial ointment is also rubbed in where the skin is thinnest. Extemporaneous formulæ of other mercurial salts are employed in the same way.

Some kinds of enlarged glands are effectively treated by topical inunctions. The ointment of the red iodide of mercury, which was official in the Pharmacopœia of 1870, is a suitable combination for topical use in such cases as goitre, enlarged spleen, etc. It is intended to be applied daily, until the skin begins to show signs of inflammation, when the application is suspended until it may be made with safety.

Baths.—The most important of applications to the skin to effect therapeutical results is by bathing. It must also be considered in its hygienic aspects, so far as these are utilized in the treatment of diseases.

In children, after the first dentition and subsequently, the daily morning sponge-bath should be a uniform practice. Children susceptible to colds and catarrhal processes in general are rendered less susceptible by this practice. It is also a tonic to the vascular and nervous systems, and this effect comes by an impression made on the peripheral nerves. Warm baths at or near the temperature of the body, and washings for cleanliness, cannot be substituted for the morning cold sponge-bath. The details consist in a tub large enough to stand in and catch the overflowing water; sufficient water at the temperature of the air in the room; the sponge or cloth sufficiently large to carry the necessary quantity of water; coarse towels for friction of the skin.

The whole body is gone over with the sponge; the coarse towel is used to dry and to make the necessary friction of the skin. The whole process requires no more than five minutes, and a glow of a very grateful character succeeds to the first chilliness of the surface. The clothing should then be put on, the toilet completed, and then to breakfast,—in these details assuming the state of fortune permitting such attention to the person.

Those having sufficient vigor of constitution—especially boys—can take a morning plunge-bath if the circumstances are propitious; but this mode of using water may be objectionable for these reasons: the heat of the body is more rapidly and considerably taken up; the shock to the nervous and vascular apparatus is greater; the reaction is more violent. Hence in children of weak, as in most of those of vigorous constitution also, the method by sponging is preferable.

In fevers and inflammations, the use of baths has attained to the dignity of a highly important therapeutical measure. The question of the propriety or need of the measure must be considered in connection with special therapeutics. Whether sponging of the surface with water or plunging into baths be the more useful expedient, is determined by the character of the case and the object to be accomplished, as the effects of these modes of application are not the same in kind or in degree. In sponging the surface the heat is reduced by evaporation of the water: in other words, the heat is converted into another mode of motion,—dissipated in the vapor. In a bath, the heat of the body is conveyed to the water by conduction, and the amount conveyed is considerable according to the distance between the heat-points of the water and the body. Thus it is that an expert swimmer fails at a distance from the shore. The heat of the body is so rapidly and largely removed that the muscles at length pass into a tetanic state, and are no longer obedient to the will.

The necessity for the use of baths for antipyretic effects is determined by the effect of heat-reduction on the course and severity of the fever. In general it may be said that the higher the fever, the more injurious the effects of the heat on the tissues and organs. Granular degeneration, not long since supposed to be due to the fever-heat, is now rather referred to the original cause: to the morbid influence or micro-organisms setting up the febrile movement. This fact admitted, the necessity for antipyretic baths is limited to the cases in which discomfort, restlessness, delirium, and so forth are caused by the heat itself. In such cases sponging the surface affords great relief; immersion in the bath is still more effective.

The mode of applying the bath in children, up to puberty, consists in immersion in water at or about 80° F. Seated in the tub, not covered above the lower border of the ribs, the water is gently put on the shoulders, arms, neck, and chest, so that complete immersion may presently be made without occasioning shock of any severity. The water of the bath should then be cooled gradually down to 60° F., or about that, by the addition of sufficient cold water, or some ice. If evidences of faintness or exhaustion appear, the child should be removed from the bath, given some stimulant if necessary, and placed between blankets if sweating be desired.

The general question of the utility of antipyretic baths in the treatment of fever remains undetermined. The most convincing series of figures, based on careful observations, are to be found in the last volume of

Guy's Hospital Reports (xlv. page 379, "Innominate Fever and Antipyretics," Dr. James F. Goodhart). In summing up, Dr. Goodhart says, "The antipyretics are pretty certain in their antipyretic action, but they do not cut short the fever; I cannot say they influence it for good in the majority of cases." What practical physician with the necessary opportunities for making clinical observations will not agree heartily with Dr. Goodhart's opinion? In children's febrile diseases, the treatment by antipyretics has proved to be specially useful and effective in appropriate cases; and of these, cold baths, easily, take the first place. There is, however, a growing belief that the lessening of fever in this way does not increase the chances of recovery, or promote improvement in the course and duration of a febrile malady. Further, there is reason to believe, indeed, that the effects of the antipyretic medicines must be added to the conditions imposed by the disease, and thus the morbid complexus becomes more complicated.

The immense superiority of the cold bath merely as an antipyretic is clear. It is true that so great an authority as Liebermeister[1] says that if restricted in his choice of antipyretics to one he would select *quinine.* If this dictum be assented to in the febrile diseases of adults, it is not true of those occurring in children. If the same restriction were imposed in the fevers of children, the cold bath would now be selected as the single and universal antipyretic.

In the febrile state now known as *hyperpyrexia,* the utility of the cold bath is unquestionable. Whenever in the course of an acute inflammatory or febrile disease the temperature rises above the level normal under the circumstances, the use of the cold bath is clearly indicated. On the other hand, if the disease pursues the regular course as to combustion and waste (which is, in typical cases, the regular course), then antipyretics are not necessary,—rather, indeed, they are hurtful. When the cold bath is not necessary in exacerbations of the fever, cold sponging without changes of posture or of clothing will prove grateful. If perfumes are pleasant to the senses, a little cologne can be added to the water; but a general bath of alcohol, diluted or undiluted, should never be used.

Medicinal Baths.—Several kinds, but few of real value, are applied to the skin to procure systemic effects. It is only by diffusion that such effects can be obtained, and this merely physical process is much hampered by the state of the epidermic layer.

Diffusion starts two movements,—one in the terminal nerves, the other in the blood-vessels. The double action may be illustrated by cocaine. When the outer skin—the epidermic layer—is taken off, the sensory nerves are acted on and deprived of sensibility for the time being; the general system is affected in a characteristic manner when the remedy is distributed by the blood, into which it diffuses.

---

[1] Handbuch der Pathologie und Therapie des Fiebers, Vogel, Leipzig, 1875, p. 634.

In the diseases of early life the skin is a less dense diffusion-membrane than it afterwards becomes, and hence "absorption" is more certain and more active; but no exactness is possible in such a mode of administration. The quantity admitted to the blood varies with the state of the skin, the fulness of the blood-veesels, and the character of the solutions. As the blood is an alkaline fluid, it is obvious that a medicinal solution having an acid reaction will diffuse into the blood more quickly than neutral or alkaline substances. Hence, when feasible, this fact should be utilized in preparing a solution.

The only solutions that need to be spoken of here are those of digitalis and quinine. The application of the former has been referred to by way of illustration, and here the additional details are given only in a general sense. When it is purposed to use digitalis or other similar vegetable remedies, the leaves, moistened with water and very slightly acidulated with vinegar, should be enclosed in a flat bag of thin muslin, but sufficiently flexible to adapt itself to the inequalities of the surface. As the action of digitalis is slow, impatience will spoil all, for if removed too soon no effect can be had.

Quinine in solution, slightly acidulated, certainly acts with considerable power. I am informed by a distinguished physician of Orange, New Jersey, in a private communication with which he has favored me, that quinine acts efficiently in children when rubbed into the skin. He makes use of ℈i to ℥i of lard, and has it thoroughly applied where the skin is thinnest. A slightly acidulated solution will probably be more efficient, and the quantity administered might be much larger.

The pads at one time so popular in this country and sold in enormous numbers must have had some effect, or the lavish advertisements in which they were kept before the people had not sufficed to maintain their position for so long a time. The Babcock pad had a strong odor of fenugreek-seed, and had a color like red cinchona bark. No doubt some quinine was contained in them also. After a few days' wear the skin on which the pad rested (the epigastric region) became red, and numerous papules appeared. The redness and moisture favored diffusion of the quinine. We do not doubt that physicians practising in malarial areas might utilize this expedient, for rarely children can take quinine without a struggle.

Endermatic.—A few words should now be said regarding applications to the true skin, the epidermis having been removed.

In very young children it is rarely that such an expedient can be practised, for the pain attending the removal of the epidermis is too great and persistent to justify the practice. In the older and more self-controlled children it may sometimes be employed. To raise a blister is the first step, and this is most easily and promptly done by moistening with stronger ammonia the inside of a watch-glass, which is then inverted over the part to be blistered. When the epidermis is removed, the medicament can then be put on the raw surface. Instead of water of ammonia in a watch-glass, pure carbolic acid can be brushed over the part to be blistered. As the anæsthetic effect of phenol is considerable, this mode of endermatic application may be used the more readily in children.

Hypodermatic Injections.—The real inventor of the hypodermatic method was Magendie, and so long ago as the first ten years of this century his demonstration was made, and consisted in placing the medicament, or a solution of it, under the skin of the animal, and then noting the time and the character of the effects produced. In this way he proved that diffusion into the blood took place, and that the action was the same in kind as, although different in degree from, that caused by the same medicine when it enters the system by the stomach. He made the first study of nux vomica, and the publication of his researches led to the use of the remedy in the maladies to which he thought it adapted, and his prescience has been confirmed by a vast experience.

In its present aspect, as introduced by Dr. Wood, of Edinburgh, the hypodermatic method consists in the use of a solution of a medicament which is thrown under the skin by means of a small syringe, the nozzle of which is a perforated needle armed with a cutting-point for puncturing the skin. The preparation of the solutions and the several methods and precautions required in injecting, the special dangers, etc., have been so copiously treated, and the information is so accessible, that further discussion of these points is not necessary.

The chief objections to the practice of hypodermatic medication in children are: the danger, the pain, the subsequent complications.

When the injection is most necessary, the little patient is not conscious,—for example, in uræmic convulsions, when pilocarpine is used. The dangers are idiosyncrasy, power and quantity of medicament. It is unsafe to use a powerful alkaloid without inquiry into the peculiar susceptibility which may exist in an adult as well as in a child. With suitable care, subcutaneous injections may be practised in the case of children not under three years of age. An inexperienced practitioner should not attempt this procedure except under experienced direction when the age is less.

The pain felt at the insertion of the needle may be blunted, or prevented, in several ways:

The needle should be small in size and sharp, having a properly-shaped cutting-point.

The skin may be benumbed by placing on it, where the injection is to be made, a pledget of absorbent cotton moistened with chloroform; by rubbing over the surface the cone used for counter-irritation and for the relief of pain (chlor. menthol, camphor, etc.); by applying the cathode of a mild galvanic current for several minutes to the skin. If the injection be practised at once, relief will be nearly complete.

The remedies used hypodermatically, chiefly, in the diseases of children, are the following:

Eserine.
Morphine and its salts.
Chloral hydrate.
Morphine and Atropine.
Pilocarpine.
Amyl nitrite.
Cocaine and its salts.
Apomorphine.
Hyoscyamine and its salts.
Quinine.
Duboisine and its salts.
Mercury.
Urethan.
Antipyrin.
Antifebrin.

The most eligible preparation for the hypodermatic method is the so-called "soluble hypodermic pellet,"—a flat, circular, pilular body, made in this form by compression with a die. The advantages of such a preparation are, convenience in preparing a solution, safety in dosage, and accuracy in amount of medicament.

The dose for administration subcutaneously is determined by age and body-weight. The rule given hereafter for apportioning the amount of a medicament for children is applicable to this method, with the exception that its power is about three times greater.

The maladies of early life in which the subcutaneous method is applied are the *painful*, the *spasmodic*, the *inflammatory*, and the *specific*. They may be conveniently grouped in this way for consideration here.

Painful Affections.—The kinds of disease meant here are those affecting the sensory apparatus of the system, apart from actual changes of structure in the nerve-substance, or in the parts to which nerves of sensation are distributed. The subcutaneous method should not be used in conditions of which pain is the chief element, unless the ordinary means of relief prove inadequate.

Such troubles as toothache and earache do not justify the use of such a powerful remedy. The more severe and exhausting neuralgic affections may justify or even require its administration. Cocaine is less dangerous than any other anodyne affording as much relief; and the curative action is greater; only, however, in the neuralgiæ of superficial nerves. It should be injected at or about the area the seat of pain.

In the convulsions of children the agent employed hypodermatically will depend on the cause: if uræmic, pilocarpine is the appropriate agent, for this so acts on the skin as to make it an efficient substitute for the kidneys. The depressing effects of this agent can be readily overcome by using its physiological antagonist, *atropine*. Therefore whenever symptoms of a threatening character arise from cocaine, its antagonist should be given without delay. As idiosyncrasy plays so large a part in the actions of pilocarpine when it causes toxic effects, the physician who purposes using it in any subject whose reactions to it are unknown should always be provided with the atropine; for should the emergency arise, delay may be fatal. In convulsions of an epileptiform character, or in uræmic convulsions not amenable to the action of pilocarpine, hypodermatic injections of morphine have proved remarkably successful in some cases. When convulsive seizures are due to inflammatory action of the meninges or of the brain-substance, or to aural troubles, or to "coarse lesions" of a subacute or chronic type, the morphine injections are to be placed first among the most effective remedies.

I must repeat my warning as to the dose and mode of administering such powerful medicines in the diseases of children. Less than three years is a contra-indication; for, although in exceptional conditions this limit may not be regarded, certainly an inexperienced physician should not take the responsibility.

The hypodermatic injection of ether is an expedient of exceptional value, as I have seen personally, when sudden and severe depression of the vital powers comes on,—as in the intestinal hemorrhage of typhoid fever, in pneumonia, both fibrinous and catarrhal, and in heart-failure from any cause. The injection is practised at any point where the subcutaneous tissue is sufficiently extensible.

Transfusion and Intravenous Injection.—But little is necessary to be said on this topic. Rarely is there need to transfuse blood, and more rarely to inject salines into the veins.

Transfusion may be required in cases of the bleeder disease (or hæmophilia), when bleeding can be arrested only by substituting blood capable of coagulation. For this purpose blood obtained from a healthy subject (boy or girl) is most suitable; but lamb's blood by direct transfusion will accomplish the object. In pernicious anæmia, if no other remedy can succeed, and the functions of other organs are in a good state, there are sound reasons for trying this expedient. Also when the blood is so far injured by the action of toxic agents, as in poisoning by phosphorus, carbonic oxide, chlorine, and other gases, the substitution of blood capable of functioning becomes imperative.[1]

The details of the operation of transfusion belong rather to surgery, and hence the reader is referred to the works in that department of medical literature.

BY THE GASTRO-INTESTINAL CANAL.

The usual route to introduce medicines into the system is by the stomach. The form of medicament and the manner of administration become especially important in the treatment of children. Attention to the preparation of such medicaments as will obviate the difficulties arising from the quantity, ill-taste, and generally nauseating character of our medicines, cannot be too strongly insisted on. Besides and beyond the therapeutical knowledge and skill which should be constant qualities, a wise physician possesses the mother-wit, in prescribing, to avoid the anguish of children and the distress of parents consequent on the struggles in giving nauseous drugs.

Of the liquids, fluid extracts, simple solutions, and concentrated tinctures are preferable. Much observation has made it clear that the pleasing mixtures in which these forms of medicaments are put by "medical confectioners" disappoint the expectations of their promoters. If the child can be readily induced to swallow the first mouthful or two, the next dose becomes a conflict, or prolonged persuasion is necessary, and at last the little one triumphs. On the other hand, a concentrated tincture or solution, or a fluid extract, can be given readily with a little address. Consideration must be given to the character of the case, to the physical and mental qualities of the child, to any idiosyncrasy known to exist, to the mental and moral stamina of the parents, and also to the nature of the medicament, the physiological effects to be produced, what dose is necessary, and at what interval the doses must be given to secure a constant impression. For example: aconite in inflammation, which for a child of three or five can be prepared by dropping into a little water, at the time required, for the first dose, two drops, and subsequently, every hour, or every two or three hours as the case may be, one drop.

---

[1] Berliner Klinische Wochenschrift, 1871, No. 21.

Large bottles of complicated mixtures or solutions are, as a rule, superfluous. It needs no diabolic instinct to determine the physician's perplexity and indecision by the number and variety of the mixtures that cumber the medicine-table or the mantel-piece. Such practice is wasteful, extravagant, costly, and should not be added to the other burdens occasioned by sickness. It is a method apt to engender unpleasant suspicions,—that the doctor and the druggist divide profits.

There are now so many fluid extracts from which to choose, that the physician can always avail himself of a concentrated preparation representing the full powers of the medicament. Besides these, alkaloids, neutral principles, glucosides, etc., are also available. As has been stated, these active principles are, as a rule, more certain, and more manageable when distinct physiological effects are to be produced. Some crude agents (*e.g.*, *pilocarpus*) are less exact, and more apt to fail under certain circumstances, than are their alkaloids (*pilocarpine*). All the alkaloids, or nearly all, and their salts, dissolve in so small a quantity of water that a drop or two of a given solution can contain a sufficient quantity to act as strongly as may be necessary.

The doses of medicaments for children have been stated in various formulæ; but mathematical devices can never take the place of discretion and knowledge. An arbitrary quantity of any medicament can never have more value than a mere suggestion. For the most part, to induce physiological actions within controllable limits is a necessary condition to effect curative results in certain states. Idiosyncrasy plays so large a part in the complex of disturbances caused by medicines, that the nature and limitations of such peculiarities must be ascertained before their therapeutical powers can be brought into action to the fullest extent. It is necessary, however, to have some guidance in fixing on the dose required at various ages as a preliminary step. The most scientific rule is that which determines the dose by comparison with the weight of the subject. Dr. E. H. Clark, of Boston, proposed the following:

The weight of the adult being fixed at one hundred and fifty pounds as the standard, this must be regarded as *unity*, or *one*. Accordingly, the dose for the child will have the same relation to one, or unity, as its weight has to one hundred and fifty pounds. If the weight of the child be divided by 150, the resulting fraction is the proper dose. Assuming the child's weight is 30, the proportion is $30 : 150 :: x : 1$. Whence we have $\frac{1}{5}$, or that the amount suitable for a child weighing thirty pounds is one-fifth of that suitable for a man weighing one hundred and fifty pounds. Reduced to actual example, it may be stated thus:

If the dose for an adult is twenty grains, the amount suitable for a child weighing thirty pounds is four grains.

Such a mode of arriving at the proper dose can be approximative only, yet it is fairly safe.

Dr. Cowling, of Louisville, Kentucky, has added to the list of formulæ by the following proposal: Take the next ensuing birthday, and divide this number by twenty-four. If the child's next birthday is four, and this is divided by twenty-four, the result is $\frac{4}{24} = \frac{1}{6}$. Consequently by this rule we have a number nearly the same as that arrived at by the Clark formula.

The method of Dr. Young, which has been long known, is to add twelve to the age of the child, and divide the product by the figure representing the age. Thus, if the age of the child is four, add twelve, which makes sixteen, and this divided by four gives $\frac{1}{4}$.

It were easier to carry the fractions in memory than to make the calculations. Any one will serve the purpose, if the resultant dose is not near the lethal one.

When we come to consider the numerous agents used to affect the digestive apparatus, it is clear that the functions are acted on in the most diverse manner; but they may be conveniently arranged in several groups:

Tonic Bitters;
Emetics;
Cathartics (including *Cholagogues*);
Anthelmintics.

At present our concern is with those remedies that improve appetite, that increase the disposition of the stomach to take food and to form peptones. Beyond this are the systemic states—as fever, inflammation, etc.—which are acted on by agencies that enter the system through the stomach.

The morbid state by the French writers called *apepsia* is one of the most common of the digestive disorders of early life. A condition characterized by slow and painful digestion, corresponding to nervous dyspepsia as it occurs in adults, is far from uncommon. In these disorders pepsin is said by its promoters to be curative; but I doubt the accuracy of their observations. There is a general distrust of reports made by physicians who so zealously defend some particular or proprietary preparation. There are numerous pharmaceutical combinations now "manufactured by the trade," containing pepsin and bismuth, pepsin and lime, pepsin and cod-liver oil, pepsin and lactic acid, etc. The number and variety of these formulæ must appeal to the average doctor and sell enormously; otherwise their reproduction, together with the additions constantly being made to the number, would cease to be profitable. It is generally believed, by those competent to form a correct opinion, that all wines of pepsin are without ferment-power, and hence that the alcoholic solutions are inert. From this conclusion the pepsin essence of Liebreich may be excepted.

In considering the question of a tonic bitter for children the quantity and disagreeable taste of the crude preparations of bitters put them aside. The most generally useful is the tincture of nux vomica, of which a minim or two will suffice, or the white fluid extract of hydrastis prepared by Lloyd of Cincinnati, which may be given in the same dose.

The routine administration of quinine when a tonic, so called, is required, has become an abuse of no inconsiderable proportions. Giving this for every trivial purpose has an ill effect on the nervous system, and may be a cause of chorea and other nervous diseases. During the formative period of the human organism it appears a not unreasonable presumption that permanent disability of nerve-matter—an excessive mobility—may be due to the impression made by quinine on the cells and fibres of the brain and cord. As Dr. Jacobi, of New York, has shown, children bear antipyretic doses of quinine sufficiently well, but such circumstances are of different character from the low state of the nutrition and the enfeeblement of constitution for which quinine is given as a supposed "tonic." No remedy is properly a tonic that does not supply normal material in which the organism is supposed to be deficient, or that does not increase the amount of food which may be received and properly utilized. Quinine is not a tonic from either point of view. As a rule, the so-called bitters have little real utility in the treatment of children's diseases.

The medical profession has been singularly neglectful of the mineral acids in treating children's diseases. Nitric acid is the first in value, and the diluted acid is the preparation most serviceable. It stimulates the secretions of the intestines, of the mucous glands, and, it is asserted, of the liver and pancreas; but how much these organs are really influenced by the acid, if at all, is not known.

In the treatment of the stomachal disorders of children, when acidity is a prominent symptom, diluted nitric acid should be given before the ingestion of food, and not after. One or two drops in some ice-water or cold water is a dose readily taken by children. In cases of colliquative diarrhœa attended with distinct acidity, the mineral acids—nitric especially—are very effective; and summer diarrhœa is of this character.

The recent discovery of *tyrotoxicon*,[1] a product of the pullulation of germs whose habitat is milk, has thrown much light on the influences that develop catarrhal affections of the intestines. The remarkable results that have been obtained by the administration of corrosive sublimate in some of these maladies would now seem to indicate that the success is due to the germicide power of the remedy. The increasing knowledge of this mode of setting up disease-processes enlarges the scope and improves the character and security of our therapeutical procedures. Hereafter this mode of treating diseases of the digestive tube must be the rule, and other modes the exception. Two points especially require attention: the first is to destroy the parasite; the second is to obviate the effects of the parasite itself, or the poison produced by it, on the organs and tissues of the body, which become altered to a greater or less degree. It is clear that antiseptics must be the most important of the remedies used to remove morbid states due to the action of micro-organisms in the gastro-intestinal canal. Those most promising in catarrhal and related affections are creasote, carbolic acid, thymol, resorcin, naphthol, corrosive sublimate, calomel, zinc, copper, and lead salts, iodol, iodoform, salol, etc.

Instead of treating merely the complex of symptoms, the real cause of the disturbance is to be acted on, and by agents whose powers have a constant relation to the work to be done. Taking into consideration the manner in which tyrotoxicon is formed, by special germs acting on milk mixed with changing organic matters, we may not only the more readily bring about decided curative results, but successful prophylaxis may be instituted. As it is always more desirable to prevent a disease than to effect a cure, especially when the cause is acting vigorously, it follows that such prophylactic measures as will prevent the admission of micro-organisms to the canal should have a place among the therapeutical measures.

The application of remedies to the treatment of obstructive disorders affecting the intestinal canal should be based on a proper conception of the conditions demanding action. Intussusception is a more common affection than fæcal impaction in children. On the other hand, the *appendix vermiformis* is more frequently the seat of inflammatory and ulcerative disorders in girls and young women. In intussusception the effects of those remedies which have a selective action on the muscular walls of the bowel should be utilized,—for example, nicotine, eserine, atropine, strychnine, picrotoxin, etc. More effective than these remedies, in some instances, are galvanism and faradism, and, as the application of either or both involves no present injury or future complication, one or both should be made use of before the inflammatory exudations and adhesions have occurred. The best mode of utilizing these powers is to place the negative electrode (a bulbous insulated sound) in the rectum, and the positive (a large, well-moistened sponge or absorbent cotton) on the abdomen, but at different points, to bring the several parts of the bowel within the circuit in turn. Galvanism may be expected to act most efficiently in cases of intussusception; faradism in impaction. It should also be known that a mild faradic current has in many instances reduced strangulated hernia when the taxis had failed. It is, in fact, the most efficient means of reducing hernia, and should be resorted to so that the taxis may be avoided.

---

[1] Ptomaines and Leucomaines, by Vaughan & Novy, Lea Brothers & Co., Philadelphia, 1888, p. 67, et seq.

Stimulants are well borne by children, and in the treatment of diarrhœal and choleraic diseases brandy (especially cognac) is usually a highly efficient remedy. Alcohol is antiseptic, and also tends to establish an inward diffusion, from the canal into the vessels, and thus checks the loss of the blood-serum.

The jaundice of children is usually catarrhal, and is an extension of the disease affecting the duodenum. Of all the remedies having power to increase the discharge of bile, phosphate and sulphate of sodium are the most efficient. In his recent original and excellent work on the liver, Harley has extolled the virtues of Glauber's salt,—sulphate of sodium. These salts may be given together, and are probably more efficient in combination than when depended on singly.

Mineral waters containing alkalies and saline constituents are also highly useful in the hepatic troubles of early life. Children are, however, not disposed to drink those, unless the taste be acquired. Of the Saratoga waters, Congress, Hathorn, and Geyser are the most grateful, and the taste for them is usually readily cultivated.

The circumstances under which hepatic medicines are required should be clearly defined in the mind of every practitioner. It is to be regretted that hepatic torpor (inactivity) is not differentiated sufficiently from irritation of the liver in which the bile is in excess. Again, when there is a mechanical obstruction to the passage of the bile to the duodenum, overflow into the blood takes place. So nicely balanced is the function that very little increased pressure in front suffices to cause the backward flow of the bile: so little pressure as a plug of mucus just filling the intestinal orifice of the ductus communis.

When increased pressure in front exists, it is useless to force the production of bile; for such attempts can only increase the michief. When the liver is in a condition due to irritation, and the state of the system is, in common language, that which is entitled "biliousness," calomel or gray powder is highly efficient. From one-sixteenth of a grain to one grain of calomel, according to the age of the child, is the amount most useful. Rarely indeed—probably never—is more than one grain necessary. It has been demonstrated by experiments on animals, and confirmed by observations on man, that calomel is not a stimulant but a sedative of the biliary function, and hence its remarkable utility in some cases of over-production of bile. Biliary fistulæ having occurred in a form to discharge all the bile externally, the other functions of the body being in a normal condition, it was found that when a full dose of calomel was given the quantity of bile produced was largely diminished. This result accords entirely with the experiments on dogs by the Edinburgh committee. These experimental facts render it clear that calomel allays hepatic irritation, and explain how it effected so much in certain kinds of diseases during the period when it occupied the first place in the esteem of the medical profession. It should be understood, also, that the color of the stools when the mercurial has had an effect is no doubt due to the influence exerted by it on the glands of the lower ileum, to whose secretion the characteristic color of the fæces is probably due. The change caused by mercury in the character of the evacuation would therefore seem to be a pathological state of this excretory matter. It has been said that children do not experience the toxic action of mercury as manifested in salivation; but it is not less true that other parts are affected quite severely, the spinach stools representing serious changes in the glands of the ileum. Notwithstanding the untoward effects of mercurials, they are powerful for good in some morbid states of the gastro-intestinal canal. Vomiting from local causes, from stomach-irritation especially, is relieved by the frequent administration of small doses—such as one-twentieth to one-twelfth of a grain—of calomel, and it acts more efficiently if subdivided by sugar of milk as minutely as possible. In the giving of small doses, frequency of administration is the rule: thus, one-twentieth of a grain every half-hour when vomiting persists.

Anthelmintics.—In considering this subject, the remedies should be discussed in relation to the form of intestinal parasite to be removed. The round worm and thread-worm are the forms that are most frequently encountered. Children of the most tender age have had tape-worm, and some instances of their congenital presence have been reported. The use of raw beef and other uncooked meats and meat-juices explains the occurrence of the tape-worm in children, but does not explain the congenital instances.

The anthelmintics most effective in the case of round worm are santonin, spigelia (pink-root), calomel (three to five grains), calomel and hydrocyanic acid, etc. After the parasites are disposed of, the catarrhal state of the mucous membrane and the general physical and mental depression require attention.

The most annoying of parasites in children, the *ascarides vermicularis*, have their habitat in the rectum and adjacent parts. Inefficient medication is the reason that the thread-worms so often recur and persist. Besides the immediate destruction of the worm and ova infesting the rectum, the ova deposited in the folds of the mucous membrane and skin, and neighboring parts, must be disposed of, for if one escapes it is the progenitor of millions soon to appear. Such remedies as quassia and aloes, in infusion, are effective in the destruction of those reached; but, as they pass up as far as the ileo-cæcal valve, inhabiting thus the whole of the large intestine, irrigation of the bowel becomes necessary. Externally parasiticide solutions should be thoroughly applied; but, as many of these are active poisons, discretion is necessary. Solutions of carbolic acid, corrosive sublimate, the salts of copper, zinc, and lead, are the most generally useful. Salicylic acid, resorcin, and other antiseptics and germicides of the same class will not cause toxic effects when applied to the skin, if judiciously used.

Tæniafuges are numerous. Recently chloroform has been much extolled, but such a remedy is not suitable for the treatment of children. As the tape-worm is composed largely of albuminous matter, it has been proposed to cause its digestion by the use of a digestive ferment, as pepsin, or the vegetable digestive agent, *papäin*, or *papayotin*. It is reported that tape-worm has been completely digested by the free use of this ferment. As, however, when the parasite is digested it disappears in the general mass of chyle and excretory matters, the result cannot be known in any other way than by the disappearance of the symptoms. The method well deserves attention.

Remedies to promote Nutrition.—As the remedies used to increase the vital resources enter the system by the stomach, it becomes necessary to say something in general terms as to the agencies required for this purpose. The use of bitters and digestive ferments to increase the activity of the primary assimilation has been discussed; but the remedial agents used to promote nutrition, to increase tissue-formation, and to add to the general resources, yet remain for discussion. The physical forces, heat, light, and electricity, and the natural stimulant and great vital restorative, oxygen, must have consideration hereafter.

First of the agents to promote constructive metamorphosis are those utilized by nature in building up the human organism. The salts of iron and lime, phosphates, and phosphites, are the chief agents employed for this purpose. The place held by iron is partly that of a necessary constituent, and partly that of an agent promoting the primary assimilation. Before the salts of iron can accomplish any improvement in the process of constructive metamorphosis, two conditions must be brought about: 1, the blood must be supplied by a due quantity of suitable aliment; 2, a proper distribution of the enriched blood must be effected.

A quarter of a century ago, Brown-Séquard asserted (and in this he echoed French opinion) that iron was reconstituent by virtue of its power to increase the activity of the primary assimilation, and not by contributing material necessary to the structure and functions of the blood-globules. It must be admitted that this view is correct, and that there is enough iron in the ordinary foods to supply the small deficiency in the blood. Another fact deserves consideration in this connection,—that the astringent salts of iron, sulphate, nitrate, chloride, etc., are more efficient as chalybeate tonics, and often agree better, than the salts formed with the vegetable acids.

Preliminary treatment is usually necessary to obtain the best results from iron. The state of the intestinal mucous membrane, the existence of constipation, a torpid state of the annexed organs, exert an untoward effect, and absorption and assimilation are thereby prevented or greatly retarded. Whether well or ill borne, effective or not effective, it is clear that iron is much abused when administered without preliminary treatment under the conditions named above. The combination of arsenic or of strychnine with iron contributes greatly to the curative results in cases of anæmia with special complications on the side of the nervous system. When the nutrition is low from the domination of cachexia, from prolonged suppuration, or from excess of waste over the reparative process, the combinations of phosphorous and phosphoric acids with lime and other salts are more useful than iron alone or than a mere phosphate; and this conception is given form in the official *Syrupus Hypophosphitum cum Ferro* of the Pharmacopœia of 1880.

Remedies for Inflammation and Fever.—In deciding on the use of remedies for the treatment of inflammation and its products, that are admitted to the system by the stomach, the stage of the process must be taken into account, so far as our present means of ascertaining the fact can be applied. Is it the stage of congestion? Is it the stage of exudation? Or do both processes continue in the same area?

The remedies that prove effective in the condition of congestion are of different powers and modes of acting from those that influence the exudation stage. The former are acted on by remedies that affect the calibre of the arterioles; but when exudation is going on, the remedies that liquefy and promote absorption are required.

Of the remedies used to influence the calibre of the arterioles, and at the same time to slow the heart, there are two kinds:

1. Those reducing congestion by acting physiologically on the vessels directly and by reflex action.

2. Antiseptics, which check inflammation by destroying or inhibiting organisms on whose presence and multiplication depends the congestion or inflammation.

In the first class are—

Aconite,
Digitalis,
Barium,
Veratrum viride,
Quinine,
Ergot.

These remedies are not equal in power and efficiency, nor do they agree in the rate at which they affect the functions concerned. They do agree in the manner in which they affect the organs of circulation, respiration, and calorification,—agree in kind, not in extent and power of action.

With these *physiological* remedies acting directly, so to speak, we include others accomplishing results by reflex influence. The following are the remedies in question:

Blood-letting (Arteriotomy; Venesection; Cupping; Leeching),
Saline Purgatives,
Counter-irritation,
Low Diet, etc.

The febrile state due to an inflammation of a tissue differs in many respects from that of an essential fever.

The stages of inflammation in a given tissue decide the character of the remedies to be used. Those affecting the amount of blood circulating in the area of inflammation have been mentioned above; but for the exudation stage the following are the most important:

The Alkalies,
Chloral,
Pilocarpine,
Picrotoxin,
Saline Laxatives,
Counter-irritants,
Ammonia,
Potash,
Soda,
Lithia,
Lime,
Renal Stimulants, or Diuretics.

The remedies for removing exudations act in various ways,—more variously, indeed, than those having the power to remove congestion. Only one result is to be reached, but that is arrived at by going several paths. Saline laxatives carry off sufficient serum to lessen the blood-pressure, and this in turn promotes absorption. Chloral has remarkable power to check the process of exudation, to liquefy the solid exudates, and to put them in a form most suitable for excretion. In such a complex as is furnished by croupous pneumonia, with cerebral disturbance, insomnia, and delirium, it is impossible to overestimate the value of this remedy. Pilocarpine has extraordinary powers for causing absorption and excretion of inflammatory exudates, and these powers are increased by combining picrotoxin with it, unless some contra-indication exist. It should not be understood that pilocarpine is given several times each day, but one dose each day, or on alternate days once, or once or twice in a week. It is adapted to the subacute, chronic states, rather than to acute cases. The manner of administering it is insisted on because it has a special office in this connection, and the desired result cannot be accomplished by frequent administration merely.

The use of antipyretics in the treatment of fever, and the nature of the functions which maintain the body-heat at the normal, are perplexing questions, until we cease to regard the heat-function of the body as something apart from heat-production as a physical process. We cannot get a truer insight into its real nature than by regarding animal heat as a mode of motion and correlative with the other physical forces.

Why should its manifestations in the human body be studied from a different stand-point from that pursued by the physicists? We learn from the highest authorities in physical science that heat must always be the product of burning,—of combustion, oxidation, chemical action, etc.,—and that there is a constant ratio between the amount of material consumed and the result in foot-pounds of force evolved. In the human body the same process must be in action to maintain a given amount of heat. The heat of the body in health is maintained at a given point (98.5° F.) in a manner that can be compared to the boiling-point of water, which does not exceed (at the sea-level) 212° F. The body-heat does not rise above 98.5° F. because there is a regulating mechanism,—the heat continuing to be produced above that point disappearing in the vapor of the perspiration, as the temperature of water is kept at the one rate by the dispersion of the water in steam. That variations in the amount of blood will change the rate of combustion going on, is true, and the conditions favoring heat-dissipation or heat-retention may also influence febrile temperature; but in these circumstances there is conformity to the physical laws governing heat as a mode of motion.

As it is clear that the temperature of the human organism is affected by the same conditions as heat in other places, the treatment of the febrile state should be conducted accordingly.

In fever the increased heat of the body represents increased combustion or oxidation, or the regulating function by which the normal state is maintained is in some way deranged. In heat-stroke or heat-fever, the tremendous rise of heat is accompanied by dry skin, so that the heat formed is retained, and not given off in vapor. In typhoid there is increased combustion, shown by the greater excretion of urea and carbonic acid. Again, in uræmia, and in jaundice, the temperature may be below normal because the state of the blood in the first-named lessens oxidation, and the lessened production of glycogen affects oxidation, because the supply is insufficient. Also, febrile heat can be lessened or increased by certain devices: lessened by measures to remove heat and thus prevent any accumulation; increased by preventing radiation or diffusion from the surface of the body.

These physical conceptions of the heat-function should govern the use of all antipyretic measures. If the cause of increased heat cannot be reached by the means employed, it may be asserted with confidence that antipyretics are useless. For example: if the more active combustion cannot be moderated, the fever-heat cannot be properly controlled.

Increased body-heat above the normal is a more common incident in children than in adults,—for the reason that the conditions necessary to combustion are more active: the circulation is more rapid and hence the materials are furnished more abundantly, and the nervous system is more quickly responsive to impressions. The need for antipyretics is therefore a more acute question in the febrile diseases of children than in those of adults. It is evident that a reformation of existing methods is coming near. The antiseptics that now play so large a part in the treatment of the specific fevers will be less employed hereafter as juster views obtain. It is now suspected that the granular degeneration and other changes which were supposed to be due to febrile heat are really the effects of the morbid materials or micro-organisms causing the disease. In this case antipyretics are less useful than they were supposed to be. It has been clearly shown also that in specific fevers and in the fever of inflammation the reduction of fever-heat does not modify nor shorten the course of the disease. In a paper of high practical value by one of the physicians of Guy's Hospital,[1] it has been shown in a collection of cases attended by fever and in specific fevers that antipyretics are of limited—in some instances of doubtful—value, in that they do not change the course of the fevers nor shorten their duration.

Cold baths are easily applied in the fevers of children, and are effective. In the essential fevers, the eruptive and specific fevers, it may be said that cold water is the most useful antipyretic. When the temperature exceeds 103° F. the cold bath may be utilized. Below that temperature, cold sponging or the use of fat-inunctions will suffice for the most part.

APPLICATIONS TO THE BRONCHO-PULMONARY MUCOUS MEMBRANE.

Methods.—As air is the material essential to the functions of the respiratory apparatus, so air may be made the vehicle for introducing medicaments. Air is also made to perform a curative part. Compressed air and rarefied air are used to act mechanically on the pulmonary tissues: the expansive force of the compressed air inspired, and the expansive force exerted by *suction* when breathing into rarefied air, are the mechanical forces employed; compressed air is utilized for increasing the relative proportion of oxygen, and thus takes the place of prepared oxygen breathed in certain doses, when the lungs are so affected either by permanent structural alterations or by functional disability that they cannot receive sufficient oxygen to carry on respiration.

Various machines have been constructed to compress air for therapeutical purposes. In places having a water-supply in reservoirs at a sufficient elevation, large metal cylinders are used, the air being compressed by the inflowing water. Such a cylinder may be employed also to procure rarefaction of air by the outflowing water. If supplied with suitable masks, flexible tubes, spray-tubes, etc., such an instrument can be utilized in many directions. Air-pumps whose action can be reversed, and provided with suitable reservoirs, masks, and tubes, are now made for every kind of service in respiratory therapeutics.

Pasteur demonstrated, not long since, that heat has a remarkable germicide power, and that the organism of alcoholic fermentation is inhibited or killed below the point at which heat becomes hurtful to wine in bottles,—at or about 160° F., and that in this way it is possible to sterilize wine. *Pasteurized* is the term used.

This fact has been turned to account in the treatment of phthisis. Heated air is now proposed as a substitute for air filled with germicide vapors. As this method is yet on trial, it will suffice to say of it now that it consists in the inhalation of heated air which is also washed by passing through solutions which remove organic impurities. The amount of heat used depends on the character of the case, on the condition and the idiosyncrasies of the patient, and also on the forbearance acquired by experience.

---

[1] Guy's Hospital Reports, vol. for 1888.

Mechanical appliances of the most complete kind have, however, but a limited scope, partly because of the form in which the agents are applied, and chiefly because of the limited time the action can go on. There is a growing conviction that the length of time during which the action of the medicament is being exerted is an important element in the curative effects. Hitherto the application, by spray, or vapor, or probang, has occupied but few minutes, rarely to exceed fifteen minutes, and hence the curative effects have been proportionally short in duration. In the application of some remedies their activity may render it necessary to use them briefly; on the other hand, many need time in which to develop their best powers. During the night the air of the bedroom, or during the day some special apartment, utilized for the purpose, can be filled with volatile materials, that can enter the ultimate air-sacs with the air breathed.

Although it has been proved that spray can pass the chink of the glottis, it is also certain that a small amount only can be made to enter the bronchi,—too little to be effective in diseases of the lungs. Young and timid children can rarely be induced to use spray effectively; but vapors in the air cannot be resisted. Irrespirable gases are exceptionally employed.

The vapors and gases most worthy of consideration as remedies are the following:

GASES.

Oxygen.
Ozone.
Compressed Air.
Rarefied Air.
Hot Air.
Carbonic Acid.
Sulphuretted Hydrogen.
Sulphurous Acid, etc.

VAPORS.

Pyridin.
Ethyl Iodide.
Ethyl Bromide.
Iodine.
Iodoform.
Bromine.
Creasote or Carbolic Acid
Eucalyptus.
Gaultheria.
Turpentine, etc.

Some of the vapors diffuse into the air at ordinary temperature, others require heat. In all cases, by the method of *protracted inhalation*, there must be enough of the medicament present to be recognized by the senses and to cause some irritation of the broncho-pulmonary mucous membrane. The effect of some of them on the system is prompt, but with the development of the action, is brief in duration; others, whether slow or quick in producing their effects, remain long in the system and keep up a sustained impression.

A method employed with success during an epidemic of *diphtheria* at the Children's Hospital at Rouen consisted in the volatilization of eucalyptus and turpentine on a common heating-stove. The leaves and stems of the eucalyptus were put in a vessel of water, and the turpentine added as required.

As the agents used by inhalation vary thus in the promptness with which the action begins, and in the duration of the effects, the length of time employed must also differ.

A small room or large closet can be readily filled by the vapors as required. A spirit- or gas-lamp or a small stove, a vessel containing some hot water, kept simmering if need be, and the medicament which is added to the hot water as required to keep the air of the apartment saturated, are the appliances needed for the purpose. Children young enough to enjoy play can carry on their sports as in an ordinary play-room. Older children can read or study. The air must be breathed, and hence the gases or vapors diffused through it must also enter the air-passages and in quantity to act on the micro-organisms.

EXCRETION AND DEPURATION.

The need of recognizing the many kinds and sources of metallic, organic, and organized poisons in these times is most imperative, and we believe that physicians are not sufficiently attentive to the indications and methods involved in the necessary therapeutic processes. The large group of affections in the treatment of which the remedies called alteratives have been, and continue to be, used is divisible into two classes: 1, those that increase the activity of the organs of excretion,—for example, diuretics, cathartics, cholagogues, and sudorifics; 2, those that act on the morbid material,—for example, mercury in specific deposits, iodides in aneurism, etc.

The definition of *alterative* has been and continues to be difficult. The vague wandering amid the clouds which formed the staple of the therapeutical conceptions formerly entertained, cannot now be accepted in the place of some tolerably distinct facts, and there are two in regard to which there can be no doubt or misconception. We observe under the action of certain remedies that deposits of a morbid kind are removed; but the tissues in which or on which these deposits are found are not necessarily changed in structure or disturbed in function by the action of the medicament. We conclude, therefore, that these remedies have power to remove morbid materials. On further investigation, it is ascertained that mineral substances deposited in and among the finest subdivisions of the tissues, and, indeed, chemically combined with the ultimate form of the organic structure,—with protoplasm,—are, also, by certain remedies sought out and put into a soluble form for excretion, and yet the tissue may remain unharmed and functionate as before. Formerly such therapeutic actions were called *alterative*, but now they are more appropriately held to be an increase of the destructive metamorphosis, or an increase of the process of waste. The products called "waste" are in part the materials produced in the functioning of all parts of the system, or thrown off as unused material, or the result of gland-activity. Such waste is physiological. Again, the matters that result from the action of medicines or morbid deposits are pathological products. In the complex materials that constitute the urine in health, we have an example of the waste that comes from the exercise of the normal functions; but in the urine we may find lead and other poisons, representing the waste caused by the action of remedies.

In our complex modern life there are so many ways in which various poisons can enter the human system that much disease often quite inexplicable in its origin, may arise in this way,—through the air or through food and water. When mysterious and complex morbid states arise in children, sources of morbid action of this kind should be taken into account. It is by maintaining activity of the organs of excretion that much may be effected in the way of curative results.

The part played by inherited syphilis is too little considered. The late Prof. Gross propounded the theory, in 1885, that inherited syphilis is the real source of most of the morbid action now afflicting mankind. Sir James Paget explains the high estimation in which mercury was held by the physicians of fifty years ago—who had no knowledge of visceral syphilis—that they found it amazingly successful in so many cases of inflammatory action, and consequent exudations, liquid and solid, that the results of its use were attributed to a general power to check inflammation and to remove its products, and did not suspect that it had an action of specificity.

It follows from the foregoing considerations that increasing the action of the excretory organs is an important function in the treatment of specific and constitutional maladies. Although these rules are more particularly applicable to patients more advanced in life, yet they cannot properly be disregarded in the treatment of children's diseases.

Besides maintaining action of the excretory organs under the conditions described above, it is necessary to take into consideration the action of certain remedies directed to the same end. I refer to the application of pilocarpine, which, causing such a copious discharge of sweat and saliva, greatly increases the absorption and excretion of morbid matters in consequence.

Although it is generally known that this agent, more than any other, has power to increase the flow of sweat, it is not so much utilized as a means to dispose of morbid products as it ought to be. Having had most satisfying results from this use of pilocarpine, I can properly urge the matter on the attention of the profession. The method proposed consists in the use of any remedy with the power to increase waste, as, for example, mercurial, which having been taken in the usual way for several days, pilocarpine is then given once a day, or on alternate days, or but once a week, to secure the extrusion of the morbific matters which had been softened, disintegrated, and thus prepared for excretion.

The pilocarpine should be given in such dose as will act efficiently, but there is no justification in the use of such a quantity as will cause after-depression in any considerable degree. It is idiosyncrasy, especially, that must be provided for! The initial dose must be a tentative one. When it is ascertained that the child is not unduly impressionable, then such doses should be prescribed as will cause decided action of the sweat-glands, and at such intervals as may be necessary. As a rule the best time for giving the remedy is at night. Provision should be made for ridding the child's person of the overflowing secretions. A warm night-gown should be in readiness to put on when the sweating has ended. If any coldness and depression remain, if the pulse become weak, and vomiting with approaching collapse is threatened, a minute dose of atropine—from $\frac{1}{400}$ to $\frac{1}{200}$ grain—will promptly arrest the symptoms. It should be a constant precaution to have some atropine at hand whenever a large dose of pilocarpine is given or the idiosyncrasies of the subject are unknown.

COUNTER-IRRITATION.

General principles only are to be considered here. The forms by means of which the irritation is effected must be stated as a guide for the application of principles to the actual practice. *Rubefaction*, *Vesication*, and the action called *Escharotic* are the general results of the impression made. Rubefaction is a making red of the skin,—red without changing its structure. Vesication consists in an action beyond the former, for the skin is not only reddened, but inflammation occurs, and the result is an exudation of serum, which constitutes "blistering." When an escharotic completes its action, a slough is formed, and, being cast off, an open ulcer is left.

In children any irritation beyond rubefaction is rarely necessary; in infants, never, for the skin is then too tender and the nervous system too responsive to impressions. It is, indeed, doubtful whether, at any age, any counter-irritation more powerful than that caused by a mustard plaster is ever necessary or beneficial. To understand the nature of the action and to define the results the physiological process must be explained.

When an irritant—a mustard plaster, for example—that causes rubefaction is applied, a local impression occurs, and wider systemic effects follow. By the local action we find dilated and fuller vessels and the end-organs of the sensory nerves are excited, pain is transmitted to the cord, and to the brain, where it is translated into consciousness, and from the cord an excitation proceeds to the vaso-motor system. As a result of the reflex, the peripheral vessels contract, and in this we have an explanation of what utility soever counter-irritation has in inflammation. The results may be stated in two separate postulates:

Mild irritation increases the vaso-motor tonicity;

Severe and destructive counter-irritation causes vaso-motor depression, and ultimately vaso-motor paresis.

If these formulæ become the guide to the use of external irritants, the method and results of such treatment will always have a sound basis.

INDEX TO VOLUME I.

A.

- Abdomen, the, 29.
  - examination of the, 84.
  - in enteric fever, 455, 467, 489.
  - prominent, in rachitis, 85.
    - its significance, 86.
- Abdominal distention, its significance, 86.
    - in enteric fever, 453, 468, 489.
  - dropsy, 86.
  - obstruction, 970.
  - organs in infancy, 3, 4, 33–37.
  - phthisis, 88.
  - pregnancy, 943.
  - ring, 31.
- Abnormal conditions at birth, 244.
  - position of the limbs after certain cases of breech-presentation at birth, 269.
- Abnormalities resulting from maternal impressions, 191–216.
- Abscesses in and after enteric fever, 474, 477.
- Absence of the cry and of tears, 108.
- Absorption by the skin, 963.
- Acid, lactic, bacillus of, 153.
  - perspiration in rheumatism, 797.
  - sarcolactic, as a cause of the sense of fatigue in growing children, 55.
- Acids, mineral, 969.
- Aconite in fevers, 435.
- Acute consumption, 404.
  - phlegmonous periostitis, 126.
  - rheumatism, etc. *See* Rheumatism, Acute, etc.
  - tuberculosis, 404.
    - sometimes mistaken for enteric fever, 480.
- Adams, Dr. S. S., on diet after weaning, 337.
- Addison's disease, 106.
- Affusion, cold, in enteric fever, 494.
- Agar-agar flesh-peptone, 182.
- Age as a factor in disease, 350.
  - in its relation to height and weight, 89, 90.
- Agnathia, 935.
- Air in medication, 977.
- Albuminuria of diphtheria, 603, 626, 628, 672.
  - in enteric fever, 473.
  - in scarlet fever, 564, 570, 578.
  - temporary, 433, 473.
- Alcohol, ethylic, produced by a bacillus, 153.
- Alcoholic bath condemned, 962.
  - friction after relapsing fever, 513.
  - solutions in practical bacteriology, 161.
  - stimulants, in enteric fever, 485, 496.
    - to be avoided in the dysmenorrhœa of puberty, 400.
  - (*See* also Stimulants, under the names of different diseases in this index.)
- Alcoholism as a cause of tuberculosis, 403.
  - juvenile, 411.
- Alimentary canal, its development, 34.
  - its peculiarities in infancy, 4, 34, 41.
- Alkaline solutions in practical bacteriology, 161.
- Alkaloids, poisonous animal. *See* Ptomaines.
- Allantois, 954.
- Alteratives, 979.
- Amenorrhœa, 399.
- Ammoniacal fermentation, bacillus of, 152.
- Amnion, 952.
  - false, 952.
- Amniotic fluid, 234.
- Amputations, intra-uterine, 224.
- Anadidymous monsters, 932.
- Anaërobic bacteria, 181.
- Anakatadidymous monsters, 936.
- Analysis of foods, 311, 313.
  - of milk, 275, 279, 280, 282, 283, 284, 288, 289, 290, 306, 307, 308, 309, 310.
- Anasarca of the fœtus, 231.
- Anatomy of children, 3–8, 11–50.
  - of joined twins, 938.
  - pathological. *See* Pathology, under the head of the principal diseases named in this index.
- Angina of scarlet fever, 568.
- Anglo-Swiss condensed milk, 315.
- Aniline dyes for the demonstration of bacteria, 161.
- Animal culture-media, 186.
- Ankle, 50.
- Ankylosis in the uterus, 231.
- Ante-natal fractures, 231.
- Anthelmintics, 972.
- Anthrax, the microbes of, 153, 154.
- Antifebrin, 435.
  - in dengue, 898.
- Antipyretics, 434, 962, 975.
  - in typhoid, 492.
- Antipyrin, 435.
  - in cerebro-spinal fever, 553.
  - in dengue, 898.
  - in malarial neuralgia, 852.
  - in scarlet fever, 576.
- Antiseptic medicines, 970.
- Antisyphilitic remedies, 225.
- Aorta, 6, 7, 26, 64.
- Apepsia, 968.
- Aphasia, 117.
  - from typhoid, 472.
  - in cerebro-spinal fever, 529.
- Aphthous stomatitis, 128.
- Apoplectic condition at birth, 247.
  - causes of, 247, 248.
  - treatment of, 248.
- Apoplexy, pulmonary, in diphtheria, 616.
- Apparatus for bacteriological investigations, 171.
  - for sterilizing food, 299, 371.
- Apparent still-birth, 247.
- Appendages, fœtal, diseases of the, 233.
- Appendix vermiformis, 970.
- Appetite, medicines which affect, 968.
- Arch of the palate, 20.
- Area germinativa, 946.
  - opaca, 948, 953.
  - pellucida, 948.
  - vasculosa, 953.
- Arms in infancy, 47.
  - lifting children by the, 47.
- Arnold's nerve, 16.
- Arseniated mineral waters, their use in chlorotic and strumous disorders, 406.
- Arsenic in malaria, 851.
- Arterial pressure in infancy, 5, 6.
- Arteries, hypogastric, their peculiarities, 255.
    - and umbilical, their closure, 252–257.

Arteries in rachitis, 5.
  in relation with the tonsils, 20.
Artery, pulmonary, 7, 26, 27, 64, 253.
  the superior vesical, its peculiar development, 256, 257.
Arthritis, rheumatic, 795.
  rheumatoid, 822. *See* Rheumatoid Arthritis.
Artificial feeding, 291.
  foods, 311, 319.
  immunity from infectious diseases, 188.
  respiration for the newly-born, 249.
Ascarides, 129, 972.
Asphyxia at birth, 247, 250.
Asses' milk, 290.
Asthenia, infantile, 245.
Asymmetric janiceps, 935.
Atavism, 111.
Ataxia, hereditary, 111.
Atelectasis pulmonum in pertussis, 709.
Athetosis, 116.
Atomizer in diphtheria, 664, 671.
Auscultation, 82, 123.
  of the back, 83.
  of the head, 95.
    in rachitic children, 5.
Authorities, lists of, on diphtheria, 583, 584.
Autumnal fever, 443.
Averages in height and weight, 53, 88, 89, 90, 91.
Axial mesoblast, 948.
Axilla, temperature of the, 100.

B.

Bacilli, multiplication of, 157.
Bacillus, 153.
  how to stain for observation, 160–169.
  of ammoniacal fermentation, 152.
  of anthrax, 153, 154.
  of blue milk, 153.
  of butyric acid fermentation, 154.
  of cholera, 900, 910.
  of cholera nostras, 186.
  of dengue, 833.
  of diphtheria, 152, 154, 587.
  of enteric fever, 154, 164, 446, 451.
  of ethyl alcohol, 153.
  of glanders, 168.
  of Klebs and Lœffler, 587.
  of lactic acid, 153.
  of leprosy, 153, 167.
  of malignant œdema, 154.
  of splenic fever, 153.
  of syphilis, 167.
  of tuberculosis, 153, 165.
  of yellow milk, 152.
  the genus, 153.
  (*See* also Bacteria, and the names of various diseases.)
Back, examinations of, 97.
  topography of, 28, 36.
Bacteria, classification of, 148.
  counting of, 186.
Bacteria, culture of, 158, 169, 186.
  definition of, 150.
  examination of, 158.
  fæcal, 69, 301.
  in alimentary canal, 302–305.
  in cerebro-spinal fever, 517.
  in enteric fever, 154, 164, 445, 451.
  in fevers, 422.
  in foods, 301.
  in pertussis, 706.
  in typhoid, 154, 164, 445, 451.
  isolation of, 169.
  morphology of, 156.
  multiplication of, 157.
  of diphtheria, 154.
  of fowl-cholera, 154.
  of pébrine, 152.
  of small-pox, 724.
  of yellow milk, 152.
  pathological properties of, 188.
  preparation of, for the microscope, 159.
  propagation of, 158.
  proper, described, 156.
  ptomaines developed by, 189, 190, 422, 451, 586, 588, 900.
  staining of, 160, 162, 168.
  transmission of, to the fœtus, 225.
  (*See* also Bacillus, and the names of the various diseases.)
Bacteriaceæ, 153.
Bacteriology, 147.
  apparatus and instruments used in studying, 171.
Bacterium (the genus), 152.
  multiplication of the, 157.
Bard, Dr. S., his observations on diphtheria, 583.
Barley, Robinson's, 310.
Barley-water, 311.
Bartholow, Dr. Roberts, on the general therapeutics of children's diseases, 956.
Bath, hot, in apparent still-birth, 248.
  in typhoid, 494.
  medicated, 962.
Bathing, 357, 366, 960.
  in fevers, 436, 961.
  sea-, 367.
  the new-born child, 241.
Beading of the ribs, its significance, 86, 94.
Bed, the, 358.
Bed-sores, prevention of, 352.
  in typhoid, 491.
  in typhus, 502.
Beef-broth, 338.
  tea, 338.
Beggiatoa (the genus), 153.
Belly. *See* Abdomen.
Benzoate of sodium in diphtheria, 669.
Biedert's cream mixture, 311, 321.
Bilateral hemiplegia, 115.
Bile as an antiferment and germicide, 303, 304.
  in children, 68.
Bilious temperament, 106.
  typhoid of Lebert, 509.
Biliousness, 971.
Bill of fare, a child's, 340, 344.
Biniodide of mercury in scarlatina, 574.
Birth, abnormal conditions at, 244.
  debility at, 245.
Birth-marks, 193, 218.
  (*See* also Nævus.)
Black measles, 678.
  vomit, 867.
Blackader, Dr. A. D., on variola, 722.
Blackleg, the microbe of, 154.
Bladder, 38.
  and uterus, their relative position in the infant, 18, 42.
  stone in the, 130.
Blastodermic membrane, 945, 947.
Blastosphere, 945.
Bleeder-disease. *See* Hæmophilia.
Blindness following cerebro-spinal fever, 538.
Blisters, 352, 981.
Blood in diphtheria, the, 612, 650.
  in infancy, the, 5–7, 64–66.
  tumor, 97.
Blood-poisoning in diphtheria, 602, 603, 612, 651.
Blood-poisons taken up by the fœtus, 219.
Blood-pressure, effects of, upon the fœtus, 219.
  in early infancy, 5, 6.
Blood-serum for culture-purposes, 175.
Blood-transfusion, 966.
Blood-vessels in early infancy, 5–7.
Blue milk, bacillus of, 152.
Blush, the febrile, 429.
Body, section of the, 20.
Boiled milk, 271, 299, 301.
Bones, formation of, 45.
  fractured in the uterus, 231.
  injuries of, at birth, 263–269.
  of the head, injuries of, 263.
  syphilitic affections of, in the fœtus, 223.
  the, in typhoid, 475.
  Wormian, 12.
Bottle-feeding, 354.
Bottles, nursing, 371.
Bovine virus, 747.
Bowels, 35.
  constipation of the, 341, 433, 467, 488.
  in enteric fever, 467, 488, 489.
  (*See* also Intestine.)
Boys, average height and weight of, 89, 90.
Brain, development of, 13.
  in infancy, 3, 12–14.
Bread-jelly, 338.
Break-bone fever, 878.
  (*See* Dengue.)
Breast of the wet-nurse, 334.

Breast-milk, 275, 279, 280, 282, 283, 284, 289, 290, 303, 306, 308, 310.
Breathing. *See* Respiration.
Brefeld's methods in bacteriological investigation, 171.
Bright's disease, 129.
Bromides in cerebro-spinal fever, 552.
Bronchial phthisis, 124.
    its diagnosis, 124.
Bronchitis, 124.
    and teething, 383.
    capillary, in whooping-cough, 708.
Broncho-pneumonia, 124.
Bronzing, its significance, 106.
Broths, 338.
    as culture-media in bacteriological study, 177.
Browne, Sir Crichton, his report on overpressure in schools, 410.
Bruit skodique, 123.
Bursæ of the knee, 49.
Busey, Dr. S. C., on scarlet fever, 555.
Butyric acid fermentation, bacillus of, 154.
Byers, Dr. J. W., on the influence of race and nationality upon disease, 132.

C.

Cachexia, malarial, 845.
Cadaver, the, after enteric fever, 452.
Cæcum, 35.
Calcification of the ligamentum arteriosum, 254.
    of the teeth, 374, 375, 376.
Calculus, vesical, 130.
Callahan, Fla., yellow fever at, 861.
Calomel as a remedy, 971.
    in diphtheria, 666.
Campbell, C. F., on the comparative death-rates in Northern and Southern States, 134.
Canada balsam for mounting bacteriological preparations, 163.
Canal, inguinal, 31, 32.
    medullary or neural, 948.
    of Nuck, 32, 33.
Cancer, 111.
    of the kidney, 130.
Capacity of the stomach, 295.
Capillary bronchitis in whooping-cough, 709.
Capsules, suprarenal, 38.
Caput succedaneum, 251, 258, 259, 260, 261.
    causes of, 259.
    diagnosis of, 261.
    position of, 260.
    treatment of, 261.
Cardiac diagnosis, 122, 125.
    system, 122.
Care of the child at birth, 236–251.
    in abnormal conditions, 244.
Carnrick's food, 311, 315, 318, 320, 322.
Carpal bones, 48.
Carpo-pedal spasms, 121.
Carrageen as an article of food and as a remedial agent, 405.
Cartilage, cricoid, 21.
Catamenial milk, 281.
    period. *See* Menstruation.
Cataphora, 398.
Causes of fever, 423.
    (*See* also Causation or Etiology, under the names of diseases in this index.)
Cells, mastoid, 15.
    of the spermatic tubules, 942.
    polar, 941.
Cellular tissue, induration of, 109.
Cephalhæmatoma, 97, 261.
    blood in, 4.
    causes of, 261, 262.
    spurious, 251, 258, 259, 260, 261.
    treatment of, 262.
    (*See* Caput Succedaneum.)
Cephalothoracopagus, 935.
Cerebellum at birth, 14.
Cerebral paralysis of the newly-born, 266, 267.
    symptoms in fever, 428, 431.
Cerebro-spinal fever, 514.
    age of patients, 523.
    anatomical characters of, 540.
    antipyrin in, 553.
    bacteria in, 517.
    blindness following, 537.
    blood-changes in, 541.
    bromides in, 552.
    causes of, 517, 521.
    cerebral effects of, 514, 541, 542, 543, 545.
    chloral in, 553.
    choreic movements in, 531.
    coma of, 546.
    commencement of, 525.
    complications of, 522, 532, 535, 546.
    conditions favoring, 518, 521.
    contagiousness of, 518.
    convulsions in, 530.
    cutaneous surface in, 535.
    deafness following, 537.
    deaths from, 540.
    definition of, 514.
    diagnosis of, 524, 539, 546.
    diet in, 554.
    digestive system in, 532.
    drowsiness in, 531.
    ear in, 538.
    eclampsia in, 531.
    endemic, 516, 539, 540.
    epidemic quality of, 515, 519, 520, 524.
    ergot in, 553.
    eruption of, 536.
    etiology of, 517, 521.
    external applications in, 551.
    exudation of, 517, 542, 543.
    eye in, 530, 537.
    geographic range of, 137, 515.
    headache in, 553.
    history of, 514.
Cerebro-spinal fever, hygiene of, 518.
    hyperæsthesia in, 528.
    in the lower animals, 520.
    influence of race and climate in, 137.
    influence of sex in, 522.
    joints in, 536.
    kidneys in, 544, 545, 546.
    lotions in, 551.
    lower animals, in the, 520.
    maculæ of, 536, 545.
    medication in, 551, 552.
    meningitis in, 514, 541, 543, 547.
    mental state in, 528.
    microbe of, 517.
    mortality from, 540.
    muscular contractions in, 528.
    names of, 514.
    naturalized, 539.
    nature of, 539.
    nephritis in, 544, 545, 546.
    nervous system in, 527.
    nomenclature of, 514.
    non-contagiousness of, 518.
    nursing in, 554.
    nutrition in, 554.
    onset of, 525.
    opium in, 553.
    origin of, 514, 517.
    pain in, 525, 527.
    paralysis in, 529.
    pathological anatomy of, 540.
    predisposing causes of, 521.
    prevention of, 547, 552.
    prognosis in, 544.
    pulse in, 533.
    pupil in, 537.
    respiratory system in, 535, 546.
    retraction of the head in, 529.
    seasons of the year in, 518.
    secondary, 522.
    sex as a factor in, 522.
    skin in, 535.
    special remedies in, 552.
        senses in, 537.
    spots in, 536, 545.
    strabismus in, 530, 537.
    stupor of, 531.
    symptoms of, 524, 525, 527, 539.
    synonymes of, 514.
    temperature in, 533.
    treatment of, 547, 548, 552.
    urinary organs in, 536.
    use of lotions in, 551.
    venesection in, 549.
    vomiting in, 526.
Cerebro-spinal meningitis, 514.
    (*See* Cerebro-Spinal Fever.)
Cerebrum and cerebellum, their relative size, 14.
Chalybeate mineral waters in chronic strumous and tubercular diseases of the young, 406.
Chamber, moist, in bacteriological observation, 172.
Chamberland's filter, 189.
Charbon, bacillus of, 153, 154.

Chart of temperatures for enteric fever, 460, 462, 476.
    for typhus fever, 498.
    for yellow fever, 854, 856, 864, 871.
  of weekly gains in weight, 272.
Cheadle, Dr. W. B., on rheumatism, 785.
Cheap foods not to be tolerated, 293.
Cheeks in infancy, 17.
Chest, examination of the, 86, 87.
  its size at birth, 55.
Chest-diameter, its ratio to head-diameter, 55, 94.
Chest-diseases, 74, 87.
Cheyne-Stokes breathing, 118, 123.
  rhythm, 67.
Chicken jelly, 342.
Chicken-pox, 754.
  (*See* Varicella.)
Child in health, the, 236.
  the sick, 349.
  the spoiled, 348.
Childhood, death-rate in, 134, 135.
Children, 79-131.
  the term defined, 497.
Children's clinic, 9.
  hospitals, 347.
Chills and fever. *See* Malaria.
Chinese measles, 141.
Chinking percussion, 123.
Chloral in cerebro-spinal fever, 553.
Chloranæmia, 7, 400.
  (*See* Chlorosis.)
Chlorate of potassium in diphtheria, 661.
Chloride of iron in diphtheria, 659.
Chlorosis, 7, 400.
  causes of, 400.
  symptoms of, 400.
  treatment of, 401.
Cholera, Asiatic, 900.
  bacillus of, 155, 900, 905, 910-914.
  bowels in, 904.
  cleanliness in, 902, 904.
  dejections in, 902, 905, 915.
  diagnosis of, 917.
  disinfection in, 902, 905, 907.
  dry, 915.
  effects on the fœtus, 229.
  enteroclysis in, 917, 918.
  fœtal, 229.
  food in, 903.
  foudroyante, 916.
  hygiene during, 902, 904.
  hypodermoclysis in, 917, 920.
  infantum, geographic range of, 142.
  infectiosa, 900.
  infectious quality of, 900.
  microbe, 155, 900, 905, 910-914.
  milk a vehicle of, 901.
  mortality from, 921.
  nostras, bacillus of, 186.
  of fowls, bacterium of, 151.
Cholera, premonitory signs of, 914, 915.
  prevention of, 902, 904, 906.
  prognosis in, 921.
  prophylaxis against, 902, 905-909.
  ptomaine of, 900.
  quarantine against, 905, 907-909.
  rice-water discharges in, 910, 913.
  sicca, 915.
  stages of, 914-916, 921.
  symptomatology of, 914.
  tannic acid enteroclysters in, 918.
  temperature in, 916.
  toxica, 917.
  treatment of, 917.
  walking attacks of, 906.
Cholera infantum, its range, etc., 142.
Cholerine, 916.
Chondrus crispus, its value as a food and as a remedial agent, 405.
Chorea, 113, 115, 123.
  in rheumatism, 803.
  its heredity, 113.
Choreic movements in cerebro-spinal fever, 531.
Circulation, fœtal, 27, 28, 64, 253, 256.
  infantile, 64.
Circulatory system in typhoid, 463.
Circumcision, 40.
Class, its influence on disease, 135.
Classification of bacteria, 148, 149, 150, 151-156.
  of remedies, 957.
Clavicle in infancy, 46.
  fracture of, 268.
Cleanliness, 356.
Cleft palate, 20.
  as a consequence of maternal impressions, 203.
Climate, 133.
  change of, as a remedial measure for tubercular disease, 406.
  in treatment of chronic strumous and tubercular disorders of puberty, 405.
  its effects on puberty, 390.
    on typhoid, 443.
Climatic influences, 133.
Clinical instruction, 9, 10.
Clostridium (the genus), 154.
Closure of ductus arteriosus, 252.
  of hypogastric and umbilical arteries, 252.
Clothing in sickness, 358.
  (*See* also Dress.)
Coagulum, 308.
Coccaceæ, 151.
Cocci, 156.
Cohn's classification of bacteria, 148, 149.
Cold pack in typhoid, 493.
  water treatment of scarlatina, 576.
  wet pack, 437.
Colden, Dr. Cadwallader, his observations on diphtheria, 582.
Colicky pains in young infants, 242.
Collapse in enteric fever, 464.
  of lung, 125.
  of lungs in pertussis, 709.
  temperature of, 103.
Collar-bone, 46.
Collie, Dr. Alex., on typhus, 497.
Colloredo, Lazaro, a double monster, 937.
Colon, 35.
  rupture of, in the fœtus, 232.
Colored races, cerebro-spinal fever in the, 137, 138.
    cholera infantum in the, 143.
    diphtheria in the, 141.
    measles in the, 140.
    scarlet fever in the, 139.
    small-pox in the, 144.
    syphilis in the, 145.
    whooping-cough in the, 137.
Colors, recognition of, 62.
  used in preparing bacteria for observation, 160.
Colostrum, 276.
Coma in cerebro-spinal fever, 531.
  (*See* also Stupor.)
Comma bacillus, 155, 186, 900.
Complexion, 106.
Complications of enteric fever, 476.
  (*See* also under the names of the various diseases described in this volume.)
Compressed air, its therapeutic uses, 977.
Compresses in typhoid, 493.
  use of, 436.
Compulsory education, evils of, 409.
Condensed milk, 315, 339.
Condition, social, as affecting disease, 135.
Confluent small-pox, 729, 731.
Congenital hernia, 33.
  movements, 56.
Congestive asphyxia at birth, 247.
Conjunctivitis and teething, 382.
Consanguineous marriages, 111.
Constipation, 341.
  in enteric fever, 467, 488.
  in fever, 433.
Continued fever, 438.
Contracted pupil, 118.
Contractions, muscular, 22.
Convalescence from enteric fever, 495.
Convolutions of the brain, 14.
Convulsions and teething, 382.
  causes of, 385.
  in cerebro-spinal fever, 530.
  in fevers, 428.
  in tubercular meningitis, 428.
  malarial, 835.
  nodding, 121.
  salaam, 121.
Convulsive diseases, 119, 120.
Cooling apparatus in bacteriological work, 172.

- Co-ordinating power, 61.
- Corban, Mrs. (a monstrosity), 933.
- Cord, cutting the, 236–238.
  - dressing the, 241.
  - ligation of the, 237.
  - umbilical, 237.
- Corrosive sublimate in diphtheria, 664.
- Cortical layer of the ovary, 939.
- Cost of infant-foods not to be considered, 293.
- Cotyledons of the placenta, 955.
- Cough, 122.
  - absence of, in chest-diseases, 122.
- Counter-irritation, 981.
- Counting of bacteria, 186.
- Cover-glass preparations, 162.
- Cow-pox. *See* Vaccination.
- Coxalgia, 48.
- Cracked-pot sound, 123.
- Cradle, the, 243.
- Cranial injuries, 263.
- Craniopagus, 935.
- Craniotabes, its significance, 95.
- Cranium, the hydrocephalic, 12.
  - the infantile, 12.
- Cravings, maternal, their possible ill effects on offspring, 214.
- Cream, 323.
  - mixture, Biedert's, 311.
  - mixtures, 321.
- Creeping, 61.
- Crenothrix (a genus of bacteria), 155.
- Cricoid cartilage, 21.
- Crookshank's classification of the bacteria, 151–156.
- Croup, 122.
- Crucifixion attitude, 121.
- Cry, 107.
  - absence of, 108.
  - hydrencephalic, 108.
  - significance of the, 107.
- Cultivations of bacteria from animal tissues, 186.
- Culture at puberty, 407, 412.
  - of bacteria, 169.
- Culture-media, animal, 186.
    - bacteriological, 177.
    - fluid, 177.
    - opaque, 184.
    - solid, 178.
  - oven, 176.
  - plates, 183, 184.
  - tubes, 184.
- Curtin, Dr. R. G., on relapsing fever, 504.
- Curvatures of spinal column, abnormal, 97, 98.
  - normal, 43, 44.
- Curve of temperature, daily, 102.
- Cutaneous disorders and teething, 385.
  - reflexes in enteric fever, 473.
  - surface. *See* Skin.

D.

- Dabney, Dr. W. C., on maternal impressions, 191.
- Daily curve of temperature, 102.
- D'Aquin, Dr., his observations on dengue, 888, *note*.
- Deafness from cerebro-spinal fever, 538.
- Death-rate of children, 135.
- Death-rates as influenced by climates, 134.
- Deaths from diphtheria, 650.
  (*See* also Mortality, under the names of various diseases in this index.)
- Debility in the newly-born, 245.
- Decidua, 955.
- Deciduous teeth, 375.
- Deckschicht, 945.
- Defecation, a regular habit of, 369.
- Defective nutrition, its effect on the fœtus, 219.
- Defects due to maternal impressions, 191, 207.
- Definition of an infectious disease, 451.
  - of the term "children," 497.
  (*See* also under the names of different diseases in this index.)
- Deformity of the head, temporary, 251.
  - unilateral, 97.
- Degenerative changes associated with fever, 424.
- Dejections in enteric fever, 482.
- Delay in walking, 92.
- Delayed dentition, 92, 376.
  - menstruation, 390.
  - walking, 92.
- Delirium in enteric fever, 471, 487.
  - its significance, 431.
- Dengue, 878.
  - antifebrin in, 898.
  - antipyrin in, 898.
  - bacteria of, 883.
  - causes of, 881.
  - clinical history of, 886.
  - complications of, 890, 891.
  - conditions favoring, 881.
  - convalescence from, 899.
  - course of, 891.
  - defined, 878.
  - derivation of the name, 879.
  - differences from malaria, 893.
    - from yellow fever, 893.
  - differential diagnosis of, 893.
  - duration of, 891.
  - epidemics of, 879.
  - eruption of, 888, 899.
  - etiology of, 888.
  - etymology of the word, 879.
  - geography of, 879, 880, 881.
  - headache in, 889.
  - history of, 879, 885.
  - incubation of, 887.
  - invasion of, 887.
  - liniments in, 899.
  - malaria compared with, 894.
  - McLaughlin's discoveries concerning, 882–886.
  - micrococci in, 883.
  - names of, 878, 879.
  - pathological anatomy and pathology of, 895.
- Dengue, prognosis in, 892.
  - pulse in, 888.
  - relapses in, 892.
  - sequels of, 890.
  - stages of, 887–890.
  - symptomatology of, 886–890.
  - synonymes of, 878, 879.
  - temperatures in, 887, 888.
  - treatment of, 896.
  - urine in, 889.
  - yellow fever and, 893–895.
- Dental groove, 18.
- Dentition, 373.
  - alleged disorders of, 350, 379.
  - anomalies of, 376.
  - dangers of, 75.
  - delayed, 92, 376.
  - history, 69.
  - in rachitis, 382.
  - irregular, 377.
  - multiple, 377.
  - precocious, 376.
  - retarded, 92, 376.
  - second, disorders of, 385.
  - symptomatology of, 379.
- Depressions of cranial bones, 263.
- Depurative medication, 979.
- Development, its diagnostic import, 87.
  - of the abdominal organs, 33, 34.
  - of the teeth, 373.
- Diabetes, 130.
  - saccharine, 114.
- Diagnosis, cardiac, 122, 125.
  - general principles of, 73.
  - methods of, 79.
  (*See* also under the names of different diseases in this index.)
- Diapers, 369.
- Diaphragm, the, 29.
- Diarrhœa during dentition, 383.
  - from improper food, 341.
  - in typhoid, 467, 489.
  - of cholera, 915.
- Dicephalus, 927.
- Dicranus, 926.
- Dicrotic pulse, 105.
- Diet after weaning, 337.
  - in diphtheria, 657.
  - in fever, 433.
  - in typhoid, 484.
- Differences between infantile and adult life, 3–5.
- Differential diagnosis. *See* Diagnosis under the names of different diseases in this index.
- Differentiation of sex, 41.
- Difficult labor as a cause of infantile paralysis, idiocy, etc., 266, 267.
- Digestion in infancy, 67, 68.
  - of milk, 68.
- Digestive functions in fever, 429.
  - system, its peculiarities during the infantile period, 3, 67, 126.
- Diphtheria, 580.
  - age of patients, 595.
  - albuminuria in, 603, 626, 672.

- Diphtheria, anatomical characters of, 589, 608, 625, 639.
  - animals as affected by, 592.
  - antiseptics in, 663.
  - apoplexy, pulmonary, in, 616.
  - atomizer in, 664, 671.
  - authorities on, 583, 584, 631.
  - bacillus of, 154, 585–591.
  - bacteria of, 152, 585.
  - benign type of, 612, 618.
  - blood in, 602, 612, 620, 650, 651.
  - brain in, 613.
  - calomel in, 666.
  - causes of, 585.
  - chlorate of potassium in, 661.
  - chloride of iron in, 659.
  - clinical history of, 618–621.
  - communication of, 592.
  - conditions favoring, 591.
  - congestion of lungs in, 650.
  - constitutional disease, 602.
  - corrosive sublimate in, 664.
  - death from, 650.
  - diagnosis of, 128, 605.
  - diet in, 657.
  - disinfectants in, 651–656.
  - ear in, 623.
  - emphysema from, 615.
  - epidemic nature of, 581.
  - epiglottis in, 614.
  - eruptions in, 625.
  - etiology of, 585.
  - eye in, 622.
  - fauces in, 606, 613, 619.
  - follicular tonsillitis in, 606.
  - food in, 657.
  - fowls affected by, 592.
  - gangrenous pharyngitis in, 608, 621.
  - genito-urinary organs in, 624.
  - geographical range of, etc., 140, 581, 584.
  - germicides in, 663.
  - glands in, 616.
  - heart-failure in, 616, 637–643, 650.
  - hemorrhage in, 621.
  - herpetic pharyngitis in, 608.
  - history of, 580.
  - hygiene of, 656.
  - incubation of, 598.
  - inflammation of, 609, 610.
  - influence of age in, 595.
  - intestines in, 612, 617, 623.
  - in the newly-born, 596.
  - iron as a remedy for, 659.
  - isolation of patients, 651–655.
  - kidneys in, 603, 618, 626, 628.
  - laryngo-tracheal, 606.
  - lists of authorities on, 583, 584.
  - literature of, 582, 583, 631.
  - liver in, 617.
  - local remedies in, 670.
  - localities of, 584.
  - lower animals affected by, 592.
  - lungs in, 614, 616, 650.
  - lymphatic glands in, 616.
- Diphtheria, membrane of, 601, 605, 610.
  - mercurials in, 664, 666.
  - mild type of, 612, 618.
  - milk as a vehicle of, 594.
  - mortality from, 650.
  - mouth in, 617, 623.
  - mucous membranes in, 610.
  - nares in, 605, 611, 621.
  - nature of, 600.
  - nursing in, 355.
  - œdema pulmonum in, 616, 650.
  - Oertel's views on, 589, 613.
  - œsophagus in, 623.
  - ordinary type of, 619.
  - papayotin in, 671.
  - paralysis of. *See* Diphtheritic Paralysis.
  - pathogeny of, 585.
  - pathology of, 589, 608.
  - pepsin in, 671.
  - pharyngitis in, 607, 608.
  - pilocarpine in, 669.
  - pleurisy in, 615.
  - pneumonia in, 615.
  - potassium chloride in, 661.
  - prevention of, 651.
  - primary, 600.
  - prognosis of, 649–651.
  - propagation of, 591.
  - pseudo-membranes of, 601, 610, 611, 621.
  - ptomaines of, 649.
  - pulmonary apoplexy in, 616.
    - œdema in, 616, 650.
  - pultaceous pharyngitis in, 607.
  - quinine in, 659.
  - range, etc., of, 141.
  - scarlatinal, 564, 568, 569, 574, 600, 607.
  - secondary form of, 600.
  - septic poisoning in, 650.
  - severe type of, 619.
  - sewer-gas as a cause or vehicle of, 591.
  - skin in, 625.
  - sodium benzoate in, 669.
  - solvents in, 670.
  - spinal cord in, 613.
  - spleen in, 617.
  - stimulants in, 658.
  - stomach in, 611, 617, 623.
  - strychnine in, 673.
  - sulphur-fumigation in, 652.
  - Taylor, Dr. W. M., his opinions concerning, 590.
  - temperature in, 620.
  - tongue in, 617, 620.
  - tonsils in, 606, 613.
  - toxæmia from, 603.
  - transmission of, in milk, 594.
  - treatment of, 602, 651, 656, 672.
  - turpentine in, 655, 667.
  - ulcero-membranous pharyngitis in, 608.
  - uræmia in, 650.
  - urinary organs in, 603, 618, 624, 626.
  - uvula in, 613, 614.
  - vesicular emphysema in, 615.
  - virus of, 585, 589.
- Diphtheria, vomiting in, 620.
  - vulvitis in, 625.
  - wounds as the seat of, 624.
- Diphtheritic inflammation, 672.
  - nephritis, 603, 618, 626–629, 672.
  - paralysis, 629, 635, 672.
    - authorities on, 631.
    - cardiac, 637.
    - causes of, 643.
    - clinical history of, 631.
    - commencement of, 631.
    - dysphagia in, 636.
    - etiology of, 643.
    - facial, 636.
    - heart-failure in, 637–643.
    - history of, 629.
    - knee-jerk absent in, 633.
    - multiple, 635.
    - mutations of, 635, 647.
    - palatal, 635.
    - pathology of, 639, 644, 645.
    - strychnine in, 673.
    - tendon reflexes absent, 633.
    - treatment of, 672.
  - stomatitis complicating measles, 679.
  - toxæmia, 603.
  - vulvitis, 625.
- Diphtheroid symptoms in small-pox, 725, 736.
- Diprosopus, 926.
- Dipygus, 932.
- Discrete small-pox, 729.
- Discus proligerus, 940.
- Diseases of the fœtal appendages, 233.
  - of the fœtus, 217.
- Disinfectants in diphtheria, 651–656.
- Disinfection, 355, 365.
- Disk, germinal, 946.
  - proligerous, 940.
- Dislocations of cranial bones, 263.
  - of the hip at birth, 269.
  - spontaneous, 475.
- Disorders of dentition, 379.
  - of female puberty, 396, 399, 402, 415.
- Distortions of the head at birth, 251, 258.
- Diverticulum, Meckel's, 30.
- Dorning, Dr. John, on dentition, 378.
- Dorsal laminæ, 948.
- Double monsters, 922.
  - (*See* Joined Twins.)
- Dress, hygiene of, 386, 414.
  - of children, 367–370.
  - of young women, 414.
- Drinking-water, 372.
- Drooling, 380.
- Drop cultures in bacteriological investigation, 178.
- Dropsy, 108.
  - abdominal, 86.
  - of the fœtus, 231.
  - scarlatinous, 571.
- Drouin twins (a monstrosity), 930.
- Drowsiness in cerebro-spinal fever, 531.

- Drowsiness in enteric fever, 487.
- Dry cholera, 915.
- Dry-heat sterilizer, 173.
- Dryness of climate, its influence on enteric fever, 443.
- Duct, omphalo-mesenteric, 952.
  - vitello-intestinal, 952.
- Ducts, Muellerian, 41.
- Ductus arteriosus, closure of, 252–257.
    - structure of, 252.
  - venosus, 28.
- Dysmenorrhœa, 399.

E.

- Ear, defects of, due to maternal impressions, 207.
  - in childhood, 15.
  - in diphtheria, 623.
  - in malaria, 842.
  - in scarlatina, 570.
  - in typhoid, 475.
- Earache and teething, 382.
- Early puberty, 93, 392.
    - and premature decay, 134, 392.
- Eclampsia, 343.
  - in cerebro-spinal fever, 531.
  - nutans, 121.
- Eczema, how distinguished from small-pox, 738.
- Eczema rubrum and erysipelas, 780.
- Education, compulsory, 409, 410.
  - medical, with respect to pediatrics, 9, 10.
  - of youth, 409.
- Edwards, Dr. W. A., on rubella, 684.
- Effects of fever, 424.
- Eklund's compilations on the death-rate of children, 135.
- Elbow, the, 47.
- Electric reactions in enteric fever, 472.
- Electricity in abdominal obstructions, 970.
- Embolism as a cause of enlarged spleen, 85.
  - in rheumatism, 805.
- Embryo, 30.
- Embryology, 939.
- Embryonic fowl, 949.
- Emphysema, subcutaneous, 109.
  - vesicular, in diphtheria, 615.
- Encephalocele, how diagnosticated from spurious kephalohæmatoma, 261.
- Endermatic medication, 963.
- Endocarditis, rheumatic, 797.
- Endometrium, diseases of the, their effects upon the fœtus, 219.
- Enemata, 353.
  - of cold or iced water in enteric fever, 495.
- Enlarged head, 95.
  - pupil, its significance, 118.
- Enteric fever, 441.
  - abscesses in and after, 474, 477.
  - age as cause of, 444.
  - albuminuria in, 473.
- Enteric fever, alcoholic stimulants in, 485, 496.
  - analysis of symptoms of, 462.
  - anatomical lesions in, 452, 475.
    - groups of, 452.
    - of central nervous system, 458.
  - antipyretics in, 492.
  - aphasia from, 472.
  - bacilli of, 446.
    - entrance of, into the organism, 450.
    - experiments of cultivation of spores in, 446.
    - not developed from other micro-organisms, 447.
    - production of a chemical poison by, 451.
    - tendency to collect in particular organs, 449.
  - bacteria in, 445.
  - bath in, 488, 494, 495.
  - bed-sores, 474, 491.
  - belly in, 467.
  - bones in, 475.
  - bowels in, 453, 467.
  - cadaver after, 452.
  - causes of, 442.
    - drinking-water as one of the, 447.
    - exciting, 445.
    - predisposing, 442.
  - charts of temperature in, 460, 462.
  - chest-troubles in, 491.
  - cicatrization of ulcers in, 454.
  - circulatory system in, 463.
  - climatic influences in, 443.
  - clinical course of, in childhood, 459.
  - coils for the head in, 493.
  - cold affusion in, 494.
    - pack in, 493.
    - sponging in, 492.
  - collapse in, 464.
  - complications of, 458, 464, 465, 469, 471, 474, 476, 485.
  - compresses in, 493.
  - constant lesion of, 453.
  - constipation in, 467, 488.
  - constitutional symptoms in, 451.
  - consumption, acute, and, 480.
  - contagiousness of, 449.
  - convalescence from, 463, 464, 495.
  - course of, 458–463.
  - cutaneous reflexes in, 473.
  - defervescence in, 462.
  - definition of, 441.
  - dejections in, 482.
  - delirium in, 459, 471, 487.
  - derivation of name, 441.
  - diagnosis of, 478.
  - diarrhœa in, 467, 489, 496.
  - dietetics of, 357, 483.
  - differentiated from acute consumption, 480.
    - from remittent, 479.
- Enteric fever differentiated from trichinosis, 481.
  - digestion in, 452, 466.
  - direct causes of, 442.
  - disinfectants in, 482.
  - dislocations in, 475.
  - drinks in, 484.
  - duration of, 441, 463.
  - ear in, 475.
  - effects of, upon the fœtus, 229.
  - electric reactions in, 472.
  - enemata in, cold, 495.
  - epidemics of, 447, 449, 482.
  - epistaxis in, 466.
  - eruption of, 460, 474.
  - etiological treatment of, 495.
  - etiology of, 442.
  - exciting causes of, 445.
  - expectancy in, 486.
  - eyes in, 470.
  - fastigium of, 463.
  - floccitation in, 471.
  - fœtal, 229.
  - geography of, 443.
  - glands in, 453, 455.
  - hair after, 474.
  - headache in, 486.
  - heart in, 456, 464, 491.
  - hemorrhage in, 466, 468, 490.
  - history of, 442.
  - house epidemics of, 449.
  - hypostatic pneumonia in, 465.
  - ice in, 493.
  - incubation of, 449.
  - inoculation of, 446.
  - intestinal hemorrhage in, 468, 490.
    - perforation in, 468.
    - symptoms of, how produced, 451.
  - invagination of intestine in, 453.
  - kidneys in, 456.
  - knee-jerk in, 472.
  - larynx in, 457, 466.
  - lesions of, 452.
    - bones in, 475.
  - liver in, 455.
  - lobar pneumonia as complication of, 458, 465.
  - lungs in, 457, 465.
  - lysis of, 462.
  - maculæ of, 460, 474.
  - management of, 483.
  - meningitis in, 471, 488.
    - simulating, 119.
  - menstruation in, 473.
  - mesenteric glands in, 455.
  - mode of life as cause, 445.
  - moisture of soil as affecting, 444.
  - mouth in, 488.
  - muscles in, 456, 457.
  - names of, 441, 443.
  - nervous system in, 470.
  - neuralgia in, 472.
  - nomenclature of, 441, 443.
  - nosebleed in, 466.
  - nurse's duties in, 484.
  - œdema of the limbs in or after, 464.
  - onset of, 459, 460.
  - osseous system in, 475.

- Enteric fever, pain in, 468, 470, 472, 475.
  - pathological anatomy of, 451.
  - pathology of, 450.
  - perforation of bowel in, 454, 468, 491.
  - peritonitis in, 469, 491.
  - Peyer's patches in, 453, 454.
  - pneumonia in, 465.
  - privy-vaults in, 482.
  - prodromes of, 459.
  - prognosis in, 481.
  - prophylaxis against, 481.
  - ptomaine of, 451.
  - pulse in, 464.
  - rash in, 460, 473.
  - reflexes in, 472, 473.
  - relapses of, 461.
  - remittents differentiated from, 479.
  - respiratory system in, 465.
  - seasons of the year in, 444.
  - sequels of, 476.
  - sex in, 444.
  - simulated by other diseases, 479.
  - skin in, 473.
  - sleeplessness in, 487.
  - soil-moisture as predisposing towards, 444.
  - sordes in, 488.
  - specific treatment of, 495.
  - spleen in, 455, 470.
  - sponging in, 492.
  - spots of, 460, 473.
  - stages of, in childhood, 458.
    - of intestinal ulceration in, 453, 454.
  - stimulants in, 485, 496.
  - stupor of, 487.
  - subsequent attacks of, 461.
  - sudden death from, 464.
  - symptoms of, 458, 462.
  - synonymes of, 441, 443.
  - taches bleuâtres in, 474.
  - temperature in, 459, 460, 462, 476.
    - during convalescence, 463.
    - height of, in childhood, 462, 463.
    - of room, 484.
  - thoracic troubles in, 491.
  - tongue in, 466, 470, 488.
  - trachea in, 457.
  - treatment of, 481, 485, 495.
  - tremor in, 487.
  - trichinosis, how distinguished from, 481.
  - tubercular meningitis, how distinguished from, 119.
  - tympany in, 453, 468, 489.
    - cause of, 453, 468.
    - form of, 454.
  - type of, in childhood, 451, 458, 460, 462.
  - ulceration of the bowels in, 453, 454.
    - of larynx and epiglottis in, 457.
    - of vocal cords, 466.
  - unrecognized, 462.
  - urine in, 460, 473.
  - use of disinfectants in, 482.
  - voluntary muscles in, 457.
  - vomiting in, 466.
- Enteritis simulating typhoid fever, 480.
- Enteroclyses in cholera, 918.
- Enumeration of bacteria, 186.
- Enuresis, 130.
- Envelope, the serous, 952.
- Epiblast, 945.
- Epiblastic ridges, 948.
- Epidemics. *See* under the names of contagious diseases.
- Epigastrius, 937.
- Epilepsy, diagnosis of, 120.
  - hysterical, 397.
  - hystero-, 121.
  - in the wet-nurse, 333.
- Epispadias, defined, 40.
  - in typhoid, 466.
- Epistaxis in enteric fever, 466.
  - in relapsing fever, 507.
- Epithelial pearls, 3.
- Epithelium, the germinal, 940.
- Ergot in cerebro-spinal fever, 553.
- Erotic literature, evil effects of, 413.
- Erotomania, 413.
- Eruption in typhoid, 474.
  - of the teeth, 375, 377.
- Eruptions, cutaneous, 385. (*See* Skin Diseases.)
- Eruptive diseases, nursing in, 355.
- Erysipelas, 771.
  - age in, 773.
  - ambulatory, 780.
  - anatomical characters of, 778.
  - anodyne applications in, 782.
  - antiphlogistics in, 782.
  - antiseptics in, 783.
  - astringents in, 782.
  - bacteria of, 772.
  - carbolic acid in, 783.
  - causes of, 771.
  - cerebral symptoms, 776.
  - chill before, 774.
  - commencement of, 775.
  - complications of, 778, 781.
  - contagion of, 773.
  - counter-irritation for, 782.
  - course of, 777.
  - definition of, 771.
  - derivation of the word, 771.
  - diagnosis of, 779.
  - digestive organs in, 776.
  - eczema confounded with, 780.
  - epidemics of, 773.
  - etiology of, 771.
  - etymology of the name, 771.
  - exclusion of other diseases by, 772.
  - external applications in, 781.
  - fever of, 777.
  - history of, 771.
  - in fœtal life, 228.
  - in the new-born, 777.
  - integument in, 775.
  - iodine in, 783.
  - kidneys in, 776.
  - local treatment of, 781.
  - lymphangitis, how diagnosticated from, 780.
  - micrococci of, 772.
  - mucous membranes in, 776, 780.
  - nomenclature of, 771.
  - nourishment in, 784.
  - pathological anatomy, 778.
  - prevention of, 781.
  - prodromes of, 774.
  - prognosis of, 780.
  - prophylaxis of, 781.
  - pseudo-, 780.
  - respiratory organs in, 779.
  - sarcoma banished by, 772.
  - sclerema, how distinguished from, 780.
  - season of the year as affecting, 773.
  - seat of, at commencement, 775.
  - sequelæ of, 778.
  - sex in, 773.
  - skin in, 775.
  - spleen in, 779.
  - stimulants in, 784.
  - symptoms of, 774, 776, 777.
  - synonymes of, 771.
  - treatment of, 781.
  - turpentine in, 783.
  - vaccination as a cause, 751, 774.
  - varieties of, 771.
- Erythema distinguished from erysipelas, 780.
- Escharotics, 981.
- Escherich's studies on the digestion of milk, 68.
- Esmarch tubes, 183.
- Ethyl alcohol, bacillus of, 153.
- Etiology, 497.
  - (*See* under names of special diseases.)
- Eustachian tube in childhood, 16.
  - in tonsillar disease, 20.
- Evacuations in sickness, 353, 365.
- Examination of back, 97.
  - of head, 93.
  - of mouth, 127.
  - of sick children, 16, 73, 75, 79, 93, 97.
  - of throat, 128.
  - of wet-nurses, 330–336.
- Excretions, the regulation of the, 979.
- Exercise, 372.
  - for chlorotic girls, 401.
- Expectoration, infantile, 74.
- Expense not a matter to be considered in selecting artificial food for infants, 293.
- Experiments on sterilized milk, 298.
- Extremities, lower, 48.
  - upper, 46.
- Eye, 14, 60.
  - and orbit, 14.
  - fœtal, 14.
  - in diphtheria, 622.
  - in disease, 116.
  - in malaria, 842.
  - movements of, 60.
- Eyes, defects of, due to maternal impressions, 285.
  - half-open, 107.

F.

Fæces, 68, 94.
Face in disease, 16, 17, 105, 106.
  in infancy, 16.
  in yellow fever, 866.
  wounds of the, 262.
Facial paralysis, 263.
    diphtheritic, 636.
  reflex movements, 59, 60.
  signs of disease, 16, 17, 105, 106.
Fall fever, 443.
  (*See* Enteric Fever.)
Family history, 110.
Fashionable dress, its ill effects, 414, 415.
Fat, its use in inunctions, 959.
Father, influence of, upon the health of his offspring, 220.
Fatigue in children, causes of, 55.
Fauces in diphtheria, 613.
Febricula, 438.
Febrile blush, the significance of, 429.
Feeding, 270.
  artificial, 288–329.
  in sickness, 356.
  intervals between, 273.
  rules for, 273.
  through the nose, 357.
Feeding-bottle, 354.
Feeding-cup, Haven's, 371.
Feeding-tubes, 294, 296, 297, 298.
Fellows's syrup of the hypophosphites, 405.
Female dress, errors in regard to, 414, 415.
Female puberty, disorders of, 396, 399, 402, 415.
  hygiene of, 412.
Femur, development of the, 49.
  fracture of the, 269.
Fermented drinks for children, 345.
  for wet-nurses, 336.
Fertilization, 943.
Fever, 417.
  (*See* also Pyrexia.)
  antipyretics in, 434.
  bacteria in, 422.
  bathing in, 436.
  blush of, 429.
  causes of, 421.
  cerebral symptoms in, 431.
  cerebro-spinal, 514.
    (*See* Cerebro-spinal Fever.)
  cold applications in, 436.
  compresses in, 436.
  constipation in, 433.
  continued, 438.
  convulsions in, 428.
  definition and nature, 417.
  delirium in, 431.
  diet in, 433.
  digestive functions in, 429.
  effects of, 424, 433.
  enteric, 441.
    (*See* Enteric Fever.)
  heat-production and heat-loss in, 417, 418.
  hectic, 427.
  lips in, 429, 430.
  nature of, 417.
Fever, necrosial, 110.
  of diphtheria, 777.
  pack in, 432, 437.
  paludal. *See* Malaria.
  poisons of, 422, 423.
  pulse in, 429.
  rapid growth associated with, 54.
  relapsing. *See* Relapsing Fever.
  remedies for, 974.
  remittent, 478.
  respiration in, 431.
  scarlet. *See* Scarlet Fever.
  senses in, 431.
  significance of, 425.
  simple continued, 438.
  skin in, 419, 429.
  sponging in, 436.
  spotted. *See* Cerebro-spinal Fever.
  stages of, 427.
  stimulants in, 437.
  symptoms, 427.
  thermic, 439.
  tongue in, 429.
  treatment of, 433, 974.
  types of, 427.
  typhoid. *See* Enteric Fever.
  typhus. *See* Typhus Fever.
  urine in, 432.
  vomiting in, 429.
Fevers and miasmatic diseases, 417.
Fever-type of typhoid in children, 451, 458, 460, 462.
Fibrous nodules in rheumatism, 801.
Fibula, development of the, 49.
Filter, Chamberland's, 189.
Finlayson, Dr. James, on diagnosis, 73.
Fireplace, the, 362.
Fission, monstrosities by, 924.
Fissure of Rolando, 13, 14.
  of Sylvius, 13.
Fistulæ at navel, 30.
Fits, maniacal, 121.
  (*See* also Convulsions.)
Fleck-typhus, 497.
Flesh-peptone, agar-agar, 182.
  gelatin, 180.
Flexure, sigmoid, 35.
Floccitation in typhoid fever, 471.
  its significance, 431.
Fluid culture-media, bacterial, 177.
Flushing of the face, febrile, 429.
Fœtal appendages, diseases of the, 233.
  circulation, 27, 28, 64, 253, 256.
  diseases, infectious, 217.
    non-infectious, 230.
  heart, 26.
  membranes, 951.
  syphilis, 221–225.
    diagnosis of, 222.
    liver in, 224.
    origin of, 221.
    osteochondritis in, 223.
    pathology of, 223.
    prognosis of, 225.
    signs of, 223.
Fœtal syphilis, spleen in, 224.
    treatment of, 225.
  tissues, 254, 255.
  traumatism, 232.
Fœtus, diseases of the, 217, 230.
  invasion of, by typhoid microbes, 449.
  papyraceus, 923.
Fontanels, 12.
  auscultation of, 95.
  closure of, 55.
  hydrocephalic murmurs over, 95.
  rachitical murmurs over, 5, 95.
  size of, 55.
Food of sick children, 345.
  the care of, 370.
Foods, 4.
  artificial, 310, 311, 315, 318, 321, 340.
  household preparation of, 326–329.
  infant's, so called, 340.
  patent, 310, 311, 315, 318, 321.
Foot, development of the, 50.
Foramen of Magendie, 13.
  of Winslow, 34.
Forchheimer, Dr. F., on malaria, 825.
Foreign bodies in trachea, 24.
Fore-milk, its inferior quality as compared with other milk, 289, 290.
Forster on the source of bodily heat, 418.
Fowl, the embryonic, 949.
Fowls, transmission of diphtheria by, 592.
Fractures. *See* Injuries.
  of cranial bones, 263.
  of the clavicle, 268.
  of the femur, 269.
  of the humerus, 267.
  in utero, 231.
Frænum linguæ, 19.
  in whooping-cough, 711.
Freckles as a diagnostic sign, 106.
French measles, 684.
Friedreich's disease, 111.
Frog-spawn fungus, 152.
Frolowski's investigations on the infantile stomach, 273, 295.
Fruit as a cause of digestive troubles, 345.
Funicular process, 33.
Furrows of the face in disease, 17, 105.
Fusion, monstrosities by, 923.

G.

Gain in weight, normal, 5, 52, 271–273.
Gall-bladder, 36.
Galloping consumption, 404.
    mistaken for enteric fever, 480.
Gangrenous pharyngitis, 608.
Gardner, J. J., on the complications of small-pox, 734–736.
Garments, infantile, 367, 370, 414.

Gases, therapeutic use of, 978.
Gastric capacity, 295.
Gastro-intestinal medication, 966.
Gelatin flesh-peptone, 179, 180.
  how kept solid at high temperatures, 177.
General development, its diagnostic import, 87.
  rules of feeding, 273.
Generation, development of the organs of, 41.
Genital precocity, 93, 390, 410.
Genito-urinary system, 129.
Geography of diseases, 132.
  (*See* also under the names of various diseases in this index.)
German measles, 684.
Germinal disk, 946.
  epithelium, 940.
  vesicle, 940.
Germinative area, 946.
Germs of disease, organic. *See* Bacteria.
  of the teeth, 18.
Gingivitis, 383.
Girls, average weight of, 89.
Glæogenes, 148.
Glanders, bacillus of, 153, 168.
Glands, lymphatic, 22.
Glandular swellings, 110.
  system, 4.
Glycogen, 54.
Gonorrhœa in the wet-nurse, 333.
Gout, inherited, 114.
Graafian vesicles, 939.
Gram's method of staining bacilli, 168.
Gravel, 130.
Green-sickness. *See* Chlorosis.
Green-stick fracture, 45, 47.
Grinding of the teeth, 129, 381.
  in typhoid, 471.
Groin, 31.
Groove, dental, 18.
Grosvenor's system of dress for children, 368.
Growing-pains, 54, 110, 113.
Growth of the child, 5, 7, 51, 52.
  its relation to weight, 53, 54, 88, 89, 90.
Guitéras, Dr. John, on yellow fever, 853.
Gums, 18.
  in teething, 382.
  lancing the, 388.

H.

Hæmatoma, 97.
Hæmatoxylin solutions, 162.
Hæmophilia, 112, 966.
Hair, care of the, 367.
  after enteric fever, 474.
Half-open eyes, 107.
Hand-fed infant compared with the breast-fed, 55.
Hand-feeding, 270, 354.
Hanging drop cultures, 178.
Hardness of water, 372.
Hare-lip, 20.
  and cleft palate, 203.
  as a consequence of maternal impressions, 203.
Haven's feeding-cup, 371.
Hay-bacillus, 153.
Head, anatomy of, 11.
  auscultation of the, 5, 95.
  defects of, due to maternal impressions, 209.
  distortions of, in labor, 251, 258.
  enlarged, 95.
  examinations of, 93.
  injuries of the, in labor, 258–267.
  percussion of the, 95, 96.
  rachitic, 12.
  sections of the, 14, 16.
  size of, at birth, 55.
  smallness of the, 96.
  unilateral deformity of the, 97.
Headache in enteric fever, 486.
Head-diameter, its ratio to chest-diameter, 55, 94.
Health an inheritance, 244.
  resorts, 406.
Hearing, development of, 63.
Heart, embryonic, 950.
  fœtal and infantile, 5–7, 26–29.
  its normal weight as compared with that of the lungs, 5.
  the, in the newly-born, 5.
Heart-diseases, diagnosis of, 125.
Heart-failure in diphtheria, 637.
Heat, dissipation of, 419.
  of fevers, its origin, 418.
  (*See* Pyrexia.)
  of the body, its source, 418.
Heat-stroke, 439.
Heated air for phthisis, 977.
Hectic fever, 427.
Height, ratios of weight to, 53, 54, 88–90.
  and weight, 89, 90.
Heightened temperature, 425.
Helen and Judith (pygopagous twins), 931.
Hemiplegia, 115.
Hemorrhage in typhoid, 490.
  in yellow fever, 869.
  meningeal, 266, 267.
Hemorrhagic small-pox, 729, 732.
Hendon disease, the, 557.
Henoch's observations on the duration of typhoid fever, 463.
Hepatic remedies, 971.
Hereditary ataxia, 111.
Heredity, 110.
  in disease, 220.
Hernia, 32, 33.
Herpetic pharyngitis, 608.
Hesselbach's triangle, 33.
Hiccough, 59.
Hide-bound, 109.
High temperature, its effect on the fœtus, 218.
Highmore, antrum of, 16.
Hip-joint, 48.
  disease, 48.
Hippocrates, his observations on the disorders of female puberty, 396.
Hirschfelder, Dr. J. O., on erysipelas, 771.
Hirst, Dr. B. C., on diseases of the fœtus, 217.
History, family, 110.
Hoffmann, E., his observations on arterial pressure in very young animals, 5.
Hooping-cough. *See* Pertussis.
Hot-bath for apparent still-birth, 248.
Human milk, 275, 279, 280, 282, 283, 284, 289, 290, 303, 306, 308, 310, 335.
Humerus, 47.
  fracture of the, during labor, 267.
Hunger-pest. *See* Relapsing Fever.
Hutchinson teeth, 379.
Hyaline degeneration of the fœtal tissues after birth, 254.
Hydramnion, 234.
Hydrencephaloid diseases, 119.
Hydrocephalic skull, 12, 95.
Hydrocephalus as a sequel of cerebro-spinal fever, 554.
  diagnosis of, 95.
Hydronephrosis, 130.
Hygiene, 360.
  of dress, 367–370, 414.
  of female puberty, 412.
  of food, 370–372.
  (*See* also Food.)
  of puberty, 407.
Hyperæsthesia, 109.
Hyperpyrexia, 421, 432.
Hypoblast, 945.
Hypodermatic medication, 964.
Hypodermoclyses in cholera, 919.
Hypogastric artery, closure of, 252.
  peculiarities of, 255.
Hypophosphites of iron, lime, and potash, their remedial value, 405.
Hypospadias, defined, 40.
Hysteria, 121.
  causes of, 414.
  the voice in, 396.
Hysterical epilepsy, 397.
  insanity, 397.
  paralysis, 398.
  trance, 397.
Hystero-epilepsy, 121.

I.

Ice in typhoid, 493.
Ice-bags, 437.
Ice-water, 372.
Idiocy, 121.
  as a supposed result of injury at birth, 267.
  microcephalic, 96.
Illness, general signs of, 350.
Imbecility, 121.
Imitative movements, 57.
Immunity from infectious diseases, artificial, 188.
Imperial Granum, 310, 311, 318, 319.
Impressions, channels of, their influence on the child, 213.
  character of, most liable to produce defects, 214.

- Impressions, duration of, in cases followed by defects, 213.
  - expectation not always a forerunner of defects after, 212.
  - maternal, 191, 218.
  - proportion of cases followed by defects in the child, 211.
  - value of expectations and statements of the mother regarding, 212.
- Impulsive movements, 57.
- Induration of cellular tissue, 109.
- Infant-feeding, 270.
- Infant-foods, 340.
- Infantile development, 3.
- Infectious disease defined, 451.
  - artificial immunity from, 188.
- Infectious diseases, fœtal, 225.
  - nursing in, 355.
- Infiltration, sero-sanguineous, 258.
- Inflammation, croupous, 609.
  - diphtheritic, 609.
  - remedies for, 974.
- Influenza resembling enteric fever, 480.
- Inheritance of good or bad health, 244.
- Iniodymus, 926.
- Iniops, 935.
- Initial weight of infants, 273.
- Injections, hypodermatic, 964.
  - in cholera, 918.
  - intravenous, 966.
  - of iced water in enteric fever, 495.
- Injuries, ante-natal, 231.
  - intra-cranial, 266.
  - of the arms, 267.
  - of the bones of the head, 263.
  - of the lower limbs, 269.
  - of the neck, 265.
  - of the trunk in labor, 267.
- Inoculation with ptomaines as a preventive of infection, 188–190.
  - with small-pox, 744.
- Insanity, hysterical, 397.
- Insolation, 439.
- Instinctive movements, 56.
- Instruction in pediatrics, 9, 10.
- Instruments for bacteriological investigations, 171.
- Intermittent fever, 825, 833. (*See* Malaria.)
  - pulse, 105.
- Intervals of feeding, 273.
- Intestinal obstruction, 127.
  - perforation, ante-natal, 232.
    - in enteric fever, 491.
  - troubles wrongly ascribed to teething, 384.
- Intestine, 35.
- Intolerance of light, its significance, 118.
- Intoxication, ill effects of, upon the fœtus, 218, 220, 221.
- Intra-cranial injuries, 266.
- Intra-uterine amputations, 232.
  - life, 13.
- Introduction, 1.
- Inunctions, 959.
- Involuntary movements, 58.
- Iodide of potassium after cerebro-spinal fever, 554.
- Iodine in tuberculosis, 405.
- Irish moss, its value as a food-medicine, 405.
- Iron as a remedy, 973.
  - chloride of, in diphtheria, 659.
- Ischiopagus, 931.
- Isolation of bacteria, 169, 183.
- Itching at anus, 129.
  - at nose, 129.

J.

- Jaborandi (pilocarpine) as a depurative agent, 980.
  - in diphtheria, 669.
  - in scarlatinal nephritis, 579.
- Jacobi, Dr. Abraham, introductory chapter, 1.
- Jadelot's lines, 17, 107.
- Jaggard, Dr. W. W., on joined twins, 922.
- Janiceps, 925, 935.
- Jaundice, its significance, 106.
  - of yellow fever, 866.
  - remedies for, 971.
- Jaw, the lower, 19.
- Jaws and teeth, 17.
- Jayne, Dr. Horace, on embryology, 939.
- Jeffries, Dr. J. A., his papers on the sterilization of food, 301, 305.
  - his views on sugar in the digestive tract, 325.
- Jelly, bread, 338.
  - chicken, 342.
  - of Wharton, 955.
- Jenner, Dr. Edward, his illustrious discovery, 744, 745.
- Jennings, Dr. C. G., on varicella, 754.
- Johnson, Dr. James, his observations on the dangers of puberty, 408.
- Joined twins, 922.
  - anadidymous, 932.
  - anakatadidymous, 936.
  - anatomy of, 927, 938.
  - by fission, 924.
  - by fusion, 923.
  - by radiation, 924.
  - causation of, 924.
  - circulatory system of, 928.
  - classification of, 925.
  - craniopagous, 935.
  - dicephalous, 927, 929.
  - dicranous, 926.
  - digestive system in, 929.
  - diprosopous, 926.
  - dipygous, 932.
  - epigastrious, 937.
  - genesis of, 922.
  - genito-urinary system in, 929.
  - ischiopagous, 931.
  - katadidymous, 925, 926, 932.
  - literature of, 938.
  - monocranous, 926.
- Joined twins, nervous system in, 929.
  - nomenclature of, 925.
  - omphalopagous, 926, 937.
  - prosopo-thoracopagous, 936.
  - pygopagous, 931.
  - radiation, 924.
  - respiratory system of, 928.
  - skeleton of, 927.
  - sternopagous, 937.
  - syncephalous, 932.
  - thoracopagous, 937.
  - xiphopagous, 936.

K.

- Karyokinesis, 944.
- Katadidymous monsters, 925, 926, 932.
- Kephalohæmatoma, 261. (*See* Cephalhæmatoma.)
  - spurium, 258. (*See* Caput Succedaneum.)
- Key West, Florida, death-rate at, 855, 856.
- Kidney-disease, 130.
- Kidneys, 37.
  - in diphtheria, 603, 618, 626–629, 672.
  - in enteric fever, 456.
  - in pertussis, 711.
  - in scarlet fever, 122, 564, 570, 578.
  - passage of microbes through the, 228.
- Kindergarten, 410.
- Klebs-Lœffler bacillus, 587.
- Klebs's method with bacteria, 170.
- Knapp's observations on the eye-complications of cerebro-spinal fever, 538.
- Knee-jerk in diphtheritic paralysis, 633.
  - in typhoid, 472.
- Knee-joint, 49.
- Knock-knee, 49.
- Koch, Prof. R., his discoveries, 147.
  - his methods with bacteria, 169, 170.

L.

- Labial line, 17, 107.
- Lactation, 270.
- Laloo (a sternopagous double monster), 937.
- Laminæ dorsales, 948.
- Lancing the gums, 388.
- Lanugo, 72.
- Large intestine, 35.
- Larrey's observations on the effects of maternal impressions, 192.
- Larynx, anatomy of the, 21.
- Leeching, 352, 353.
- Leiter's head-coils, 493.
- Lemon-juice in malaria, 852.
- Lens, Zeiss's apochromatic, 158.
- Leprosy, bacillus of, 153, 167.
- Leptothricheæ, 155.
- Lethargy, 398.
- Leuconostoc, 152.
- Lifting children by the arms, 47.

- Ligamentum arteriosum, 253.
- Ligation of the cord, 237.
- Light, artificial, in the nursery, 364.
  - intolerance of, its significance, 118.
  - perception of, 62.
- Limbs, development of, 44.
- Linea alba, 29.
- Lines, Jadelot's, 17, 107.
- Lips in fever, 429.
  - in infancy, 17.
- Literature of teratology, 938.
- Liver, 36.
  - in enteric fever, 455.
  - its peculiarities in infancy, 68.
  - its peculiarities in monsters, 938.
- Liver-pad, 963.
- Lobar pneumonia in enteric fever, 458.
- Lœffler's method with bacteria, 168.
- Lœfland's sterilized milk, 317.
- Longings, maternal, their possible ill effects, 214.
- Lordosis, 98.
- Love-sickness, 413.
- Lower extremities as affected by maternal impressions, 207.
  - extremity, the, 48.
- Lungs, 25.
  - in diphtheria, 614.
  - in double monsters, 938.
  - in enteric fever, 457.
- Lustgarten's method in bacterial study, 167.
- Luxations. *See* Dislocations.
  - in utero, 231.
- Lymphangitis, how distinguishable from erysipelas, 780.
- Lymphatic glands in diphtheria, 616.
    - of the neck, 23.
  - vessels of the head and neck, 40.

M.

- MacAlister on the origin of bodily heat, 418.
- MacLaughlin, J. W., his investigations regarding dengue, 882–886.
- Maculæ of cerebro-spinal fever, 536, 545.
  - of enteric fever, 460, 474.
  - of typhus, 499, 500.
- Madden, Dr. T. M., on puberty, its pathology and hygiene, 380.
- Malaria, 825.
  - age as affecting, 832.
  - alimentary tract in, 839.
  - anatomical characters of, 832.
  - arsenic in, 851.
  - bacteria of, 479, 826–831.
  - benign type of, 834.
  - blood-corpuscles in, 829, 833.
  - brain-complications in, 836.
  - bronchial tubes in, 833, 839.
- Malaria, cachexia of, 845.
  - causation of, 826.
  - cerebro-spinal nerves in, 837.
  - change of residence for, 851.
  - changes in its geographic range, 825, 831.
  - chill from, 835.
  - chronic, 845.
  - circulatory system in, 840.
  - complications in, 836, 846.
  - definition, 826.
  - dengue compared with, 894.
  - diabetic symptoms in, 841.
  - diarrhœa from, 840.
  - effect of the seasons in, 830.
  - etiology of, 826.
  - fœtal, 228.
  - glycosuria in, 841.
  - how distinguished from dengue, 894.
  - infant-mortality from, 832.
  - intermittent, 833.
  - intestinal complications, 840.
  - lemon-juice for, 852.
  - medication for, 848.
  - mental disturbances of, 837.
  - microbes in, 826.
  - miscarriage due to, 831.
  - moisture as a factor in, 831.
  - morbidity from, 831.
  - nervous system in, 836.
  - neuralgia from, 837, 844.
  - night-air in, 831.
  - organs of special sense in, 842.
  - pathological anatomy of, 832.
  - pathology of, 826.
  - peculiarities of, in children, 835.
  - pernicious intermittent, 834.
  - plasmodium of, 828.
  - pneumonia, its relation to, 839.
  - poison of, 826, 830.
  - prognosis, 847.
  - prophylaxis, 847.
  - quartan, 833.
  - quinine in, 848.
  - quotidian type, 833.
  - remittent type, 842.
    - continuous, 844.
    - forms of, 843.
    - thermometric range of, 844.
  - respiratory apparatus in, 838.
  - season of the year in, 830.
  - skin in, 842.
  - spleen in, 840, 846, 851.
  - stages of, 835.
  - symptomatology of, 833.
  - tertian type, 833.
  - thermometry of, 844.
  - treatment of, 847.
  - typhoidal forms of, 842.
  - urinary organs in, 841.
  - vaso-motor disturbances in, 838.
- Malarial cachexia, 845.
  - disease in the fœtus, 228.
- Malformations of the teeth, 378.
- Malignant disease of the kidney, 130.
- Malpighi, pyramids of, 37.
- Malposition of teeth, 378.
- Mammals, ovary of, 940.
- Management of sickness, 354.
  - of teething, 386.
- Maniacal fits, 121.
- Marie and Rose (joined twins), 930.
- Marks, mother's, 17, 193, 211, 218.
  - (*See* Maternal Impressions; *also* Nævus.)
- Mastoid cells and antrum, 15.
- Masturbation, 93, 131.
- Matas, Dr. Rudolph, on dengue, 878.
- Maternal impressions, 191, 216, 217, 218.
  - as affecting the ears, 207.
    - eyes, 208.
    - hair, 210.
    - lower extremities, 207.
    - skin, 210.
    - trunk, 209.
    - upper extremities, 205.
  - as producing defects, deformities, or marks, 196–216.
- Maturation of the ovum, 941.
- Maxillary bones, 17.
- McClellan, Dr. George, on the anatomy of children, 11.
- Meals, 344.
  - time of, 343.
- Measles, 675.
  - anatomical characters of, 676.
  - atypical courses of, 678.
  - black, 678.
  - bronchitis in, 680.
  - causation of, 675.
  - Chinese, 141.
  - complications of, 677, 679.
  - conjunctivitis in, 679.
  - contagion of, 675.
  - convalescence from, 682.
  - convulsions in, 681.
  - decline, stage of, 677.
  - definition of, 675.
  - diagnosis of, 678.
  - diet in, 355.
  - diphtheritic symptoms in, 679, 680.
  - disinfection in, 355.
  - distinction from rubella and from scarlet fever, 699.
    - from small-pox, 738.
  - epidemic characters of, 680.
  - eruptive stage of, 677.
  - etiology of, 675.
  - eye-troubles in, 679.
  - fœtal, 228.
  - geographical range, etc., 140.
  - German or French, 684.
  - hemorrhagic, 678.
  - history of, 675.
  - hot drinks in, 681.
  - hygienic conditions in, 681.
  - in fœtal life, 228.
  - intestinal troubles in, 680.
  - invasion of, 676.
  - malignant types of, 678.
  - mouth in, 679.

Measles, names of, 675.
  nursing in, 355.
  pathological anatomy of, 676.
  pneumonia in, 680.
  prevention of, 681.
  prognosis of, 680.
  stage of decline in, 677.
  stimulants in, 682.
  stomatitis in, 679.
  symptomatology of, 676.
  synonymes of, 675.
  treatment of, 681.
Meckel's diverticulum, 30.
Meconium, 69.
Media aortæ, its relations with the ligamentum arteriosum, 253, 254.
Media for the culture of bacteria, 177.
Medullary plates, 948.
  substance, the ovarian, 940.
Meigs's mixture, 322.
Mellin's food, 310, 311, 319.
Membrana granulosa, 940.
Membrane, subzonal, 954.
Membranes of the brain, 12.
  fœtal, 951.
Meningeal hemorrhage, 266, 267.
Meningitis, 109.
  cerebro-spinal, 116, 137, 547.
    (*See* also Cerebro-spinal Fever.)
  in typhoid fever, 471.
  its alleged frequency at Havana, 857.
  rheumatic, 805.
  simulating typhoid, 480.
  sporadic, its discrimination from cerebro-spinal fever, 547.
  tubercular, 119.
  (*See* Tubercular Meningitis.)
Menstrual disorders of puberty, 399.
Menstruation, commencement of, 391.
  delayed, 390.
  in typhoid, 473.
  premature, 390.
  return of, 281.
Mental defects due to maternal impressions, 191.
  training of youth, 409, 413, 414.
Mercurials in diphtheria, 664.
  in fœtal syphilis, 225.
Mercuric iodide in scarlatina, 574.
Merismopedia (the genus), 152.
Mesoblast, 947.
Metabolism a source of heat, 418.
Metacarpal bones, 48.
Metatarsal bones, 50.
Method in bacterial study, Klebs's, 170.
  Koch's, 169, 170.
  Lœffler's, 168.
Methods of staining in bacterian study, 160, 162, 168.
Miasmatic diseases, 417.
  (*See* also Malaria.)
Microbes. *See* Bacteria.
Microcephalic idiocy, 96.
Micrococcus (the genus), 151.
  diphtheriticus, 585.
Microkinesis, 56.
Micro-organisms. *See* Bacteria.
Microscopy of the bacteria, 158.
Micturition, 70, 130.
Miliary tubercle, 404.
Milk, abnormal, 276.
  analyses of, 275, 279, 280, 282, 283, 284, 289, 290, 306, 307, 310.
  as a food after weaning, 337, 338.
  as a vehicle of infection, 557, 594, 901.
  asses', 290.
  bacillus of blue, 153.
  bacterium of yellow, 152.
  boiled, 299, 301.
  catamenial, 281.
  chemical analysis of, 288, 307, 360.
  condensed, 315, 339.
  cow's, 290, 306, 308, 309, 310, 339.
  digestion of, 68.
  human, its analyses, etc., 275, 279, 280, 282, 283, 284, 289, 290, 303, 306, 308, 310.
    its characters, 335.
  of the wet-nurse, 279, 280, 282, 283, 284, 335, 336.
  one cow's, 338.
  peptonized, 317, 340.
  secretion of, 277.
  sterilized, 317.
  variations of, 280.
  yellow, its bacterium, 152.
Milk-sugar, 325.
Milk-teeth, 374.
Milzbrand, bacillus of, 153.
Mineral acids, 969.
  waters in infantile hepatic troubles, 971.
    in strumous and tubercular disease, 406.
Miscarriages due to malaria, 831.
Mitchell, Dr. A., on the results of maternal impressions, 193.
Mixed foods, 322, 327.
Moist chamber, its use in bacteriological investigations, 172.
Moisture in the air, its therapeutic uses, 354.
Mole, blastoderm of the, 947.
Money, Dr. Angel, on the physiology of infancy, 51.
  his observations on certain complications in enteric fever, 472.
Monocranus, 926.
Monoplegia, 115.
Monsters, anadidymous, 932.
  double, 922.
Monstrosity, moral, 217.
Monthly nurse, 240.
Moral abnormity as a result of maternal impressions, 217.
Moral qualities requisite in a wet-nurse, 330.
Moral training of youth, 408, 409.
Morbid anatomy. *See* under names of individual diseases.
Morbilli. *See* Measles.
Morphology of bacteria, 156.
Morula, 944.
Mother's marks, 17, 193, 211, 218.
Mountain health-resorts, evil as well as good results from visiting, 407.
Mounting of bacteriological preparations, 163.
Mouth, examinations of, 127.
  in enteric fever, 488.
Movements classified, 57.
  congenital, reflex, instinctive, and volitional, 56.
  of the newly-born, 57.
Muguet, 128.
Multiple dentition, 377.
  paralysis, diphtheritic, 635.
Multiplication of bacilli and of the bacterium, 157.
Mumps, 767.
  (*See* Parotitis.)
Murmurs over rachitic fontanels, 5.
Muscles, heat-producing function of the, 418.
Muscular contractions, 22.
  paralysis, pseudo-hypertrophic, 98, 112, 118.
  sense, 62.
Music, excessive devotion to, 413.

N.

Nævus, facial, 17.
Napkins, infants', 369.
Nares in diphtheria, 611, 621.
Nasal line, 17, 107.
Nationality, its influence on disease, 132.
Neck, anatomy of the, 21–24.
  injuries during labor, 265.
Necrosial fever, 110.
Negroes. *See* Race and Nationality.
Nematogenes, 148.
Nephritis, diphtheritic, 603, 618, 626–629, 672.
  in cerebro-spinal fever, 544, 545, 546.
  scarlatinous, 564, 570, 571, 578.
    pleural effusions in, 122.
Nerves, their course as a guide in diagnosis, 31.
Nerve-sedatives, their use in the disorders of female puberty, 416.
Nervous disorders of females at puberty, treatment of, 415.
  system in disease, 115.
    in typhoid, 470.
    its peculiarities in infancy, 3, 51.
    its relations to thermogenesis and thermolysis, 420.
Nestlé's food, 311, 315.
Neural canal, 948.
Neuralgia after typhoid, 472.
  from malaria, 837.
Neurotic diseases, 113.
Newly-born child, care of the, 236, 238, 244.

Newly-born child, movements of the, 57.
  senses of the, 62.
Nightingale, the two-headed, 930.
Night-light, 364.
Night-terrors, 121, 343.
Nocturnal terrors, 121.
Nodding convulsions, 121.
Nodules, rheumatic, 800, 801.
Non-infectious fœtal diseases, 230.
Normal gain in weight, 273.
Nose in diphtheria, 611, 621.
  in infancy, 16.
Nosebleed in enteric fever, 466.
  in relapsing fever, 507.
Notch of Rivinus, 15.
Notochord, 948.
Nourishment. *See* Food.
Novel-reading, excessive devotion to, 413.
Nuck, canal of, 33.
Nucleolus, 940.
Nucleus, 940.
Numbering of bacteria, 186.
Nurse, wet-, 286, 330.
Nurse's duties at childbirth, 240.
  in cerebro-spinal fever, 554.
  in general, 347–359.
Nursery, the, 351, 361.
  hygiene, 360.
Nursing of sick children, 345, 347-359.
Nursing-bottles, 371.
Nutrition of the fœtus, defective, 219.
Nutritive remedies, 973.
Nycturia, 130.

O.

Oatmeal bath, 358.
Obstruction of the intestine, diagnosis of, 127.
Occipito-atlantoid disease, 98.
Occupation in its relations to female health at puberty, 415.
Oculo-zygomatic line, 17, 107.
O'Dwyer, his discoveries in the treatment of diphtheria, 584.
Œdema, 109.
  of the extremities after enteric fever, 464.
  pulmonum in diphtheria, 616, 650.
Oertel's views on diphtheria, 589, 613.
Œsophagus, the, 24.
  in diphtheria, 623.
Ointments, 960.
Oleates, 960.
Omphalo-mesenteric duct, 952.
  veins, 953.
Omphalopagus, 926, 937.
One cow's milk, 338.
One-sided paralysis, 115.
Oosperm, 591.
Opaque area, 948, 953.
  culture-media, 184.
Opium in cerebro-spinal fever, 553.
  in rheumatism, 819.
Opodymus, 926.
Optic nerve, 118.
Orbit of the eye, 14.
Oscillation of the pupil, 118.
Osseous system in typhoid, 475.
Osteoblasts, 45.
Osteochondritis, syphilitic, 223, 224.
Otalgia during teething, 382.
Otitis after scarlatina, 570.
Out-door exercise, 372.
Ova, the primordial, 940.
Ovarian tumor in a child, 93.
Ovaries, 42, 939.
Ovary, cortical layer of, 939.
  follicles of, 940.
  germinal epithelium of, 840.
  mammalian, 940.
  medullary substance of, 939, 940.
  membrana granulosa of, 940.
  section of, 939, 940.
  structure of, 939.
  vesicles of, 939.
Overwork in schools, 410.
Ovum, 940.
  of the cat, 940.
  of the mammal, 940.
  of the rabbit, 945–949.
  the fertilization of the, 943, 944.
  the maturation of the, 941.
Oxyuris vermicularis, 129.

P.

Pack, 437.
  dangers of, 432.
Pain as elicited by diagnostic manipulations, its significance, 84.
  in enteric fever, 468, 470, 472, 475.
  in general, 109.
Painful affections, the relief of, 965.
Pains, growing, 54, 110, 113.
  in general, 109.
Palatal paralysis, diphtheritic, 635.
Palate, cleft, 20.
  in infancy, 20.
Pallor, its significance, 106.
Palpation in diagnosis, 84.
Pancreas, 37.
Pancreatic secretion, 67.
Papain, or papayotin, 973.
  in diphtheria, 671.
Papyraceous fœtus described, 923.
Paralysis, 116, 117.
  cerebral, in the newly-born, 266, 267.
  facial, 263.
  hysterical, 398.
  in cerebro-spinal fever, 529.
  in enteric fever, 472.
  infantile, 115.
  one-sided, 115.
  of the arm in the newly-born, 268.
  pseudo-hypertrophic, 98, 112, 117.
Parish, Dr. W. H., on wet-nurses, 330.
Parotitis, 767.
  abscess in, 770.
  causes of, 768.
  defined, 767.
  diagnosis of, 769.
  epidemic, 768.
  etiology of, 768.
  idiopathic, 768.
  local effects of, 768.
  metastasis in, 767, 769.
  names of, 767.
  pathological anatomy of, 768.
  prognosis of, 770.
  symptomatology of, 768.
  synonymes of, 767.
  treatment of, 770.
  varieties of, 767.
Parvin, Dr. Theophilus, on injuries of the new-born child, 258.
Passage of bacteria into the fœtal circulation, 225–230.
Pasteur, Dr. Wm., on fever, 417.
Pasteur, Louis, his illustrious discoveries, 147.
Pasteurization, 977.
Patent foods, 311, 315, 318, 321, 340.
Paternal influence in producing diseased offspring, 220.
Pathogenetic qualities of bacteria, 188.
Pathology. *See* the names of various diseases in this index.
Pearls, epithelial, 3.
Pediatrics as a specialty, 1, 2.
  the study of, 9.
Peligot and Reiset on milk, 289, 290.
Peliosis rheumatica, 802.
Pellucid area, 948.
Pelvis, 38.
Penis, 40.
Penrose, Dr. R. A. F., on the care of newly-born children, 236.
Pepsin, 968.
  for tape-worm, 973.
  in diphtheria, 671.
Peptonized milk, 317, 340.
Peptonum siccum in bacteriological culture-media, 177.
Percussion of the head, 95.
Perforation of the intestine in enteric fever, 454, 468, 491.
  in the fœtus, 232.
Pericarditis, 125.
  from periostitis, 126.
  pyæmic, 126.
  rheumatic, 126, 798.
Perichondritis of the arytenoid cartilages in or after enteric fever, 466.
Pericranium, 11.
Perineum, 43.
Periosteum, 45, 110.
Periostitis, 110.
Peritoneum, 33.
Peritonitis in enteric fever, 469, 491.
  tubercular, 85, 86, 127.
Perityphlitis, its seat, 35.
Permanent teeth, 375.
Pernicious intermittent, 834.

Pertussis, atomizer in, 719.
  bacteria in, 706.
  belladonna in, 720.
  brain and membranes, 708.
  bronchi in, 708, 709.
  capillary bronchitis in, 709.
  causes of, 703, 713.
  collapse of lungs in, 709.
  complications of, 708, 711, 721.
  contagion of, 704, 706, 713.
  convulsive stage of, 715, 720.
  cough in, 711.
  curative measures in, 719.
  definition of, 703.
  diagnosis of, 715.
  diet in, 721.
  disinfection in, 717.
  emphysema in, 709, 712.
  etiology of, 703, 713.
  eyes in, 708.
  frænum linguæ in, 711.
  geography of, 136.
  glands in, 710.
  heart in, 710.
  history of, 703.
  hygiene of, 717.
  in public institutions, 718.
  isolation of, 716.
  kidneys in, 711.
  kink of, 711.
  lungs in, 708, 712, 713.
  mortality from, 705.
  mucous membrane in, 708.
  names of, 703.
  nose in, 708.
  palliative treatment of, 719.
  pathology of, 706, 713.
  pneumonia in, 710.
  prevention of, 715.
  prodromes of, 713.
  prognosis in, 715.
  prophylaxis of, 715.
  pulmonary collapse in, 709.
  quarantine of, 716.
  race and nationality in, 136.
  sanitation in, 717.
  stages of, 713, 719, 720.
  stomach in, 708.
  symptoms of, 713.
  synonymes, 703.
  tracheo-bronchial glands in, 710.
  treatment of, 715, 719.
  virus of, 704, 706.
  whoop of, 712.
Pestilential diseases, their origin, 581.
Petechiæ of typhus, 499.
Petechial fever, 497.
Peyer's patches, 453, 454.
Phalanges, 48, 50.
Pharyngitis, gangrenous, 608.
  herpetic, 608.
  in diphtheria, 607, 608.
  pultaceous, 607.
  scarlatinous, 607.
  ulcero-membranous, 608.
Pharynx, anatomy of, 20.
Phimosis, 131.
Phlegmonous inflammation, how distinguishable from erysipelas, 780.
Photophobia, 118.
Phragmidiotherix (a genus of bacteria), 157.
Phthisis, 123.
  abdominal, 88.
  therapeutics of, 977.
  wasting in, 88.
Physical signs of disease, 17, 74, 107.
    in the wet-nurse, 333.
  training of youth, 409.
Physiognomy, 17, 105.
Physiology of infancy, 51.
Pigeon-breast, 24, 86, 230.
Pigmentation, its significance, 106.
Pilocarpine as a depurative, 980.
  in diphtheria, 669.
  in scarlatinal nephritis, 579.
Pin-worms, 129, 972.
Pitting in small-pox, 740.
Placenta, 28, 955.
  diseases of, 233.
  separation of the, 236.
Placental cotyledons, 955.
Plant, Dr. W. T., on vaccination, 743.
Plasmodium of malaria, 828.
Plate-cultures, 182, 184.
Plates, medullary, 948.
Pleuræ, 25.
Pleurisy, 125.
  in diphtheria, 615.
  in rheumatism, 799.
Plumbing, 365.
Pneumonia, 124.
  fœtal, 230.
  in diphtheria, 615.
  in malaria, 839.
  in rheumatism, 799.
  in whooping-cough, 710.
  of fœtal life, 230.
Poisons in the maternal blood, 219.
  pathogenic, 189, 190, 422, 900.
Polar cells, 941.
Polyuria, 130.
Pomeroy, Jesse, a possible cause of his abnormal moral character, 217.
Porcher on the distinction between dengue and yellow fever, 893.
Position of the viscera, 24, 85.
    in the fœtus, 27.
  (*See* also Topography.)
Potassium chlorate in diphtheria, 661.
Potato-culture (bacterial), 184, 185.
Pott's disease, 31.
Poupart's ligament, 32.
Poverty and wealth, their influence on disease, etc., 135.
Precocity, sexual, 93, 390, 392, 410.
Predigestion. *See* Peptonized Foods.
Pregnancy during lactation, 283.
Premature decay, 134.
  ossification, 2.
  puberty, 93, 390, 392, 410.
Prevention of disease by inoculation, 188–190.
  (*See* also Inoculation.)
Primitive streak and trace, 946.
Primordial ova, 940.
Privy-vaults in enteric fever, 482.
Prodigium Willinghamense, 393.
Prœotia, 93, 390, 410.
Prognosis in typhoid, 481.
  (*See* also under the heads of diseases named in this index.)
Proligerous disk, 940.
Prominent belly, its significance, 86.
Pronucleus, female, 942.
  male, 943.
Properties of bacteria, 188.
Prosopo-thoracopagus, 936.
Prostate gland, 40.
Protovertebræ, 948.
Pseudo-erysipelas, 780.
Pseudo-hypertrophic muscular paralysis, 98, 112, 118.
Pseudo-membranes, diphtheritic, 601, 610.
Ptomaines, 189, 190.
  as a cause of disease, 422.
  inoculation with, 190.
  of cholera, 900.
  of diphtheria, 586, 588.
  of enteric fever, 451.
Puberty, 389.
  age of, 390, 392, 410.
  changes in the mastoid at, 15.
  chloranæmia at, 400.
  diseases of, 393.
    in the female, 394.
  evolution of, 389, 391.
  hygiene of, 389, 407, 412.
  hysterical disorders of, 395.
    epilepsy in, 397.
    insanity in, 397.
    paralysis in, 398.
    trance in, 397.
  in females, 390.
  menstrual disorders of, 399.
  nervous disorders of, 395, 415.
  occupation in its relation to health at, 415.
  of males, 392.
  physical and moral culture at, 407, 412.
  premature, 93, 392, 410.
  regimen at, 345.
  signs of. 392, 394.
  special disorders of, 394.
  strumous disorders of, 402.
  treatment of nervous disorders of, 415.
  tuberculosis of, 402.
Puerile breathing, 125.
Pulmonary œdema in diphtheria, 616, 650.
  system, 122.
Pulse, dicrotic, 105.
  in diagnosis, 104.
  in fever, 104, 424.
  in relapsing fever, 507.
  intermittent, 105.
  normal, 66.
  slowness of, 104.
Pultaceous pharyngitis, 607.
Pupil, contracted, 118.
  enlarged, 118, 119.
  oscillating, 118.

- Purposeless movements, infantile, 57, 58.
- Purpuric erythema in rheumatism, 802.
- Pyæmia, how distinguishable from rheumatism, 810.
- Pyelitis, 130.
- Pygopagus, 931.
- Pyramids of Malpighi, 37.
- Pyrexia, 417, 976.
  - its significance, 425, 426, 427.
  - its stages and types, 427.
  - its treatment, 961, 976.
  - of childhood contrasted with that of adult life, 425.
  - symptoms of, 427.
- Pyrogenesis, 421.

Q.

- Qualifications of a nurse, 347–359.
  - of a wet-nurse, 330.
- Quality of food, 275–329.
- Quarantine in pertussis, 716.
  - in yellow fever, 876.
- Quinine in dengue, 898.
  - in diphtheria, 659.
  - in enteric fever, 486.
  - in malaria, 848.
    - dose of, 849.
  - in scarlatina, 575.
  - its routine administration, 969.
  - its uselessness in cerebrospinal fever, 554.
  - rectal administration of, 850.

R.

- Rabbit's ovum, 945, 949.
- Rabenhorst's classification of the bacteria, 150.
- Race, its influence on disease, 132, 146.
- Rachitic œdema, how distinguishable from rheumatism, 810.
  - pelvis, 38.
  - rosary, 86.
- Rachitis, 5, 24, 64, 112, 123.
  - as a cause of various obscure symptoms, 381.
  - blood-vessels in, 5.
  - dentition in, 382.
  - in the wet-nurse, 332.
  - of the fœtus, 230.
  - signs of, 85.
- Radiation, monstrosities by, 924.
- Radius, its development, 48.
- Ratio of chest to head in size, 55.
  - of heart to aorta, 64.
- Ratios of growth to weight, 53, 54, 88, 89, 90.
- Rectum, 41.
  - thermometry of the, 101.
- Recurrent fever, congenital, 229.
  - typhus. *See* Relapsing Fever.
- Reflex movements, 56, 58.
- Reflexes, cutaneous, in enteric fever, 473.
  - tendon, in enteric fever, 472.
    - in diphtheritic paralysis, 633.
- Regimen of the sick, 345.
- Reiset and Peligot on milk, 289, 290.
- Relapsing fever, 504.
  - appetite in, 507.
  - bacteria of, 505, 509.
  - bilious form of, 509.
  - causes of, 505.
  - complications in, 507, 509, 512.
  - contagion of, 505.
  - convalescence from, 513.
  - definition of, 504.
  - delirium in, 508.
  - diagnosis of, 509, 510.
  - diarrhœa in, 509.
  - disinfection in, 511.
  - epistaxis in, 507.
  - eruptions in, 510.
  - etiology of, 505.
  - headache in, 508, 512.
  - heart in, 508, 512.
  - history of, 504.
  - hygiene in, 511.
  - jaundice in, 507, 513.
  - joints in, 508.
  - liver in, 506, 507, 510.
  - medication in, 512.
  - mortality from, 509, 511.
  - nervous system in, 508.
  - nosebleed in, 507.
  - nourishment in, 511.
  - pain in, 508, 513.
  - pathology of, 505.
  - prognosis in, 511.
  - pulse in, 507.
  - relapses of, 506.
  - sleeplessness in, 512.
  - spleen in, 506, 507, 510.
  - symptoms of, 506.
  - synonymes of, 504.
  - temperature in, 507, 509, 512.
  - tongue in, 507.
  - treatment of, 511.
  - types of, 509.
  - vomiting in, 513.
- Remedies, administration of, 967.
  - classification of, 957.
  - in general, 956.
- Remittent fever, 479, 842.
  - (*See* Malaria.)
  - how distinguished from enteric fever, 479.
- Renal disease, 6, 129.
  - in diphtheria, 603, 618, 626.
  - in scarlet fever, 571, 578.
- Reprise, the, in the cry and breathing, 107.
- Respiration, 25, 26, 56, 66, 67.
  - (*See* also Breathing and Cheyne-Stokes.)
  - artificial, 249.
  - establishment of, 236, 248.
  - in fever, 431.
- Respiratory organs, medication of the, 977.
- Respiratory system, 122.
  - in typhoid, 465.
- Retarded dentition, 92, 376.
  - menstruation, 390.
  - walking, 92.
- Revaccination, 753.
- Rex, Dr. O. P., on parotitis, 767.
- Rheumatism, 110, 785.
  - acute, 785.
  - age in, 791.
  - alkalies in, 817, 818, 819.
  - anæmia of, 806, 820.
  - anatomical characters of, 792, 793.
  - ankylosis following, 814.
  - arthritis in, 788, 795.
    - (*See* also Rheumatoid Arthritis.)
  - articular, of the fœtus, 229.
  - bacilli of, 793.
  - causes of, 787.
  - childhood, its peculiarities in, 785.
  - chill as a cause of, 787, 792.
  - chorea in, 803, 814.
  - chronic, 821.
  - definition of, 785, 787.
  - diagnosis of, 809.
  - diet in, 820.
  - digitalis in, 819.
  - duration of, 818.
  - effects of, 786.
  - embolism and thrombosis in, 805.
  - endocarditis in, 786, 797, 811.
  - erythema in, 802.
  - etiology of, 787.
  - examples illustrative of, 806–809.
  - family history in, 113, 789–791.
  - fibrous nodules in, 785, 801, 813.
  - fœtal, 229.
  - head-troubles in, 805.
  - heart-disease in, 786, 794, 797, 806, 812, 817, 819.
  - heredity of, 113, 789–791.
  - hot springs, their waters in chronic cases, 821.
  - iron in, 820.
  - joints in, 796.
  - manifestations of, 786, 806.
  - meningitis in, 805.
  - microbes of, 793.
  - morbid anatomy of, 793.
  - muscular exercise as a predisposing cause of, 787.
  - name of, 785.
  - nervous symptoms of, 805.
  - nodules in, 785, 801, 813.
  - of the fœtus, 229.
  - opium in, 819.
  - pain in, 815.
  - pathology of, 792.
  - peculiarities of, in early life, 785.
  - pericarditis in, 788, 794, 795, 798, 811.
  - perspiration in, 797.
  - pleurisy in, 799.
  - pneumonia in, 799.
  - poison of, 793.
  - predisposing causes of, 787.
  - prevention of, 821.
  - prognosis of, 812.

- Rheumatism, purpuric erythema in, 803.
  - pyæmia mistaken for, 810.
  - rachitis mistaken for, 810.
  - salicin and the salicylates in, 815, 816.
  - scarlatinal, 570, 788.
  - scrofulous swellings mistaken for, 810.
  - sex in, 791.
  - stiffness of tendons from, 795.
  - stimulants in, 819.
  - symptoms of, 786, 794, 806.
  - syphilis of the bones mistaken for, 811.
  - temperament in, 791.
  - temperature in, 796, 820.
  - tendinous nodules in, 785, 801, 813.
  - thrombosis in, 793, 805.
  - tongue in, 797, 815.
  - tonsillitis in, 796, 800, 811.
  - transmissibility of, to offspring. 113, 789–791.
  - treatment of, 814, 821.
  - Whipham's table illustrative of treatment, 818.
- Rheumatoid arthritis, 822.
  - anatomical characters of, 822.
  - causes of, 822.
  - definition of, 822.
  - diagnosis of, 823.
  - etiology of, 822.
  - morbid anatomy of, 822.
  - pathology of, 822.
  - prognosis of, 823.
  - symptoms of, 822.
  - treatment of, 823.
- Rhythm of breathing, 25, 26, 56, 66, 67, 123.
  (*See* also Cheyne-Stokes.)
- Ribs, beading of the, 86.
- Rickets. *See* Rachitis.
- Ridges, epiblastic, 948.
- Rita-Christina, 930.
- Rokitansky's opinion as to the effects of maternal impressions, 193.
- Rosary, rachitic, its significance, 86.
- Roseola epidemica. *See* Rubella.
- Rotch, Dr. T. M., on infant-feeding and weaning, 270.
- Rötheln, 684.
- Round worms, 129, 972.
- Rubefaction, 981.
- Rubella, 684.
  - age in, 688.
  - albuminuria in, 696.
  - bacteria of, 688.
  - causes of, 687.
  - complications in, 697, 702.
  - contagion of, 687.
  - definition of, 684.
  - desquamation in, 693.
  - diagnosis of, 698.
  - diet in, 701.
  - epidemics of, 686, 687.
  - eruption of, 690.
  - etiology of, 687.
  - glands in, 695, 697.
  - history of, 684.
  - incubative stage, 689, 699.
  - infectiousness of, 687.
- Rubella, influence of sex in, 688.
  - invasion of, 689.
  - measles, its differences from, 699.
  - mortality from, 698.
  - names of, 684.
  - prodromes of, 689, 699.
  - prognosis in, 698.
  - pulse in, 696.
  - rash of, 691, 693.
  - relapse in, 698.
  - scarlet fever, its differences from, 699.
  - sex in, 688.
  - sore throat in, 695.
  - stage of eruption, 690, 694.
    - of incubation, 689.
    - of invasion, 689.
  - symptoms of eruptive stage, 694.
  - synonymes of, 684.
  - throat in, 695.
  - tongue in, 696.
  - treatment of, 700.
  - urine in, 696.
  - vomiting in, 695.
- Rubeola. *See* Measles.
- Rules for feeding, 273.
- Rupture of the colon, intra-uterine, 232.

S.

- Saddle-back curvature of the spinal column, 98.
- Saint Bénoît twins, 930.
- Salaam convulsions, 121.
- Salicin and the salicylates, 816.
- Salicylic acid in scarlatina, 574.
- Salivary secretion, 67.
- Sanitaria, cautions with regard to the choice of, 407.
- Sarcina (the genus), 152.
- Sarcolactic acid a possible cause of the sense of fatigue in growing children, 55.
- Sarcoma of the kidney, 130.
- Scalp, 11.
  - blood-tumor of, 97.
  - wounds of, 262.
- Scapula in infancy, 46.
- Scarlatina. *See* Scarlet Fever.
- Scarlatinal diphtheria, 564, 568, 569, 574, 607.
  - rheumatism, 788.
- Scarlatinous nephritis, 571, 578.
    - pleural effusions in, 122.
  - pharyngitis, 607.
- Scarlet fever, 555.
  - age in, 559.
  - albuminuria in, 564, 570, 578.
  - alimentary tract in, 570.
  - anatomical characters of, 563.
  - angina of, 568, 569.
  - antipyrin in, 576.
  - belladonna in, 561.
  - brain in, 564.
  - causes of, 556.
  - chlorate of potash in, 577.
  - cold-water treatment of, 576.
  - complications of, 569, 577.
- Scarlet fever, conditions favoring, 557–560.
  - contagium of, 556.
  - convulsions in, 570.
  - coryza of, 569.
  - definition of, 555.
  - diagnosis of, 571.
  - diet in, 579.
  - diphtheritic symptoms in, 564, 568, 569, 574, 607.
  - disinfection in, 561, 575.
  - diuretics in, 579.
  - dropsy of, 573.
  - ear-troubles after, 570.
  - epidemics of, 559, 560.
  - eruption of, 563, 565, 567, 578.
  - etiology of, 556.
  - eye-troubles in and after, 570.
  - febrile symptoms of, 575.
  - fœtal, 228.
  - forms of, 565.
  - geography, etc., 139.
  - heart in, 569.
  - history, 555.
  - immunity from, 562.
  - incubation of, 561.
  - inoculation with, 557.
  - inunctions in, 561, 578.
  - in utero, 228.
  - irregular types of, 568.
  - isolation of, 561, 575.
  - kidneys in, 122, 564, 570, 571, 578.
  - malignant form of, 569.
  - measles, its differences from, 699.
  - mercuric iodide in, 574.
  - milk as a vehicle for, 557.
  - mortality from, 574.
    - as influenced by poverty, etc., 559.
  - nephritis of, 122, 564, 570, 571, 578.
  - nervous system in, 570.
  - of the fœtus, 228.
  - onset of, 565.
  - pathological anatomy of, 563.
  - pathology of, 562.
  - pilocarpine in nephritic complications of, 579.
  - portability of, 556.
  - potassium chlorate in, 577.
  - predisposition to, 558, 559.
  - prevention of, 560.
  - prognosis of, 573.
  - prophylaxis of, 560.
  - pulse in, 567.
  - quinine in, 575.
  - regular type of, 565.
  - relations to the Hendon disease, 557.
  - rheumatism after, 570, 788.
  - rubella, its difference from, 699.
  - salicylic acid in, 574.
  - season of the year for, 559.
  - second attacks of, 562.
  - sequels of, 569.
  - sex in, 559.
  - small-pox, its differences from, 738.
  - sore throat in, 556, 564, 568, 569, 577.
  - stimulants in, 577.

- Scarlet fever, susceptibility to, 559, 562.
  - symptoms of, 565.
  - synonymes of, 555.
  - temperature of, 566.
  - throat-symptoms in, 556, 564, 566, 568, 569, 577.
  - treatment of, 574.
  - types of, 565.
  - uræmic symptoms in, 579.
  - urine in, 567.
  - varieties of, 565, 568, 569.
  - virus of, 556, 557.
- Schizomycetes, 150.
- Schools, overpressure in, 410.
- Sclerema, 109.
  - how distinguishable from erysipelas, 780.
- Scoliosis, 3.
- Scrofula as related to tuberculosis, 403.
  - in the wet-nurse, 331.
  - its geographical range, etc., 146.
- Scrotum, 41.
- Sea-bathing, 367.
- Seat-worms, 129, 972.
- Second summer, 341.
- Section of the body, 20.
  - of the head, 14, 16.
  - of the neck, 21.
- Séguin, Dr. E., his observations on the effects of maternal impressions, 192.
- Seminal granule, 943.
- Sense-perceptions in early infancy, 62.
- Septic vibrion, 154.
- Septicæmia of the fœtus, 229.
- Sequels of enteric fever, 476.
- Sero-sanguineous infiltration, 258.
- Serous envelope, 952.
- Sewer-gas as a vehicle of diphtheria, 591.
- Sexual excitement as caused by improper food, 345.
  - precocity, 93, 390, 392, 410.
- Shakespeare, Dr. E. O., on cholera infectiosa, 900.
  - on practical bacteriology, 147.
- Shedding of the temporary teeth, 375.
- Shoes of children, 370.
- Siamese twins, 936.
- Sick child, the, 349.
- Sickness, management in, 354.
  - signs of, 350.
- Sick-room, 349. 351, 361.
- Sight in early infancy, 62.
- Sigmoid flexure, 35.
- Significance of heightened temperature, 425.
- Signs of disease in a wet-nurse, 333.
  - of sickness in children, 350.
- Sinus terminalis, 953.
- Sinuses at birth, 12.
- Skeleton, development of, 38, 45.
- Skin, 71.
  - as influenced by maternal impressions, 210.
  - in fever, 419, 429.
  - in typhoid, 473.
- Skin-diseases, as related to teething, 385.
  - how discriminated from small-pox, 738.
- Skoda's tympanitic percussion-sound, 123.
- Skull, hydrocephalic, 12, 95.
  - infantile, 2, 3, 12.
  - rachitic, 95.
- Slavering, 380.
- Sleep, talking in, 121.
  - terrors in, 121.
  - walking in, 121.
- Sleeplessness in relapsing fever, 512.
  - in typhoid, 487.
- Sleep-walking, 121.
- Slow pulse, the significance of, 104.
- Small intestine, 35.
- Small-pox. *See* Variola.
- Smell, the sense of, in early infancy, 63.
- Smith, Dr. J. L., on cerebro-spinal fever, 514.
  - on diphtheria, 580.
- Social condition as influencing disease, 135, 408, 413.
- Sodium benzoate in diphtheria, 669.
- Solid culture-media, 178.
- Solutions used in the staining of bacteria, 161.
- Solvents in diphtheria, 670.
- Somatopleure, 948.
- Somnambulism, 121.
- Sordes in enteric fever, 488.
- Sore throat, 128.
  - (*See* also Diphtheria, Rubella, Scarlet Fever, and Tonsillitis.)
- South Carolina twins, the, 932.
- Soxhlet's sterilizing apparatus, 298, 371.
- Spasms, carpo-pedal, 121.
- Specific treatment of typhoid, 495.
- Spermatozoa, 942.
- Spina bifida, 98.
- Spinal column, 43.
  - cord in diphtheria, 613.
  - curvatures, abnormal, 97, 98.
    - normal, 43, 44.
- Spirilli, 156.
- Spirillum, 155.
- Splanchnic layer, 948.
- Splanchnopleure, 948.
- Spleen, 36.
  - enlarged, 85.
  - in diphtheria, 617.
  - in enteric fever, 470.
  - in malaria, 840.
  - in yellow fever, 865.
- Splenic fever of sheep, bacillus of the, 153.
- Spoiled children, 348.
- Sponging in general, 959.
  - in typhoid, 436, 492.
- Spontaneous dislocations, 475.
- Spores, the staining of, for microscopic study, 168.
- Spotted fever. *See* Cerebro-spinal Fever.
- Sputum, absence of, in diseases of respiration, 122.
- Staining of microbes, 160, 162, 168.
- Standing, 61.
- Star-fish, fertilization of ovum in, 944.
  - polar cells of, 941.
- Steam sterilizer, 174.
- Stenosis of the larynx after enteric fever, 466.
- Sterilization of food, 294, 298, 301–305.
- Sterilized milk, 317, 371.
- Sterilizing apparatus, bacteriological studies, 173, 177.
  - for infants' food, 299.
- Sterno-cleido-mastoid muscle, injuries of the, 265.
- Sternopagus, 937.
- Stiffness of tendons after rheumatism, 795.
- Still-birth, apparent, 247.
  - from syphilis, 221, 225.
- Stimulants in children's diseases, 819, 971.
  - in diphtheria, 658.
  - in fever, 437.
  - in scarlatina, 577.
- Stomach, 34, 68.
  - capacity of the, 295.
  - development of, 34.
  - Frolowski's investigations on, 273, 295.
  - in diphtheria, 611, 616, 623.
  - its physiology, 68.
- Stomatitis, 127.
  - in teething, 383.
- Stone in the bladder, 130.
- Stoves, 363, 364.
- Strabismus as a sign of cerebro-spinal fever, 530.
- Streak, the primitive, 946.
- Streptococcus (the genus), 151.
- Stricture of the colon, its seat, 35.
- Strippings, their richness as compared with that of other milk, 289, 290.
- Strumous disorders of puberty, 402.
- Strychnine in diphtheritic paralysis, 673.
- Stupor in cerebro-spinal fever, 531.
  - in enteric fever, 470.
  - in typhus fever, 499.
- Subzonal membrane, 954.
- Sucking-cushions, 17.
- Sugar in infants' food, 324.
  - of milk, 325.
  - teats condemned, 383, 387.
- Sulphur bath, 358.
  - for rheumatoid arthritis, 824.
- Sulphurous mineral waters
  - their use in chronic disorders of puberty, 406.
  - their use in rheumatoid arthritis, 824.
- Sunlight, 361.
- Sunstroke, 439.
- Superior vesical artery, its development, 256, 257.
- Supernumerary teeth, 377.
- Superstitions about teething, 386.
- Sweating, its significance, 106.

Symptoms. *See* the names of the various diseases in this index.
  of fever, 427.
Syncephalus, 935.
Syncope of the newly-born, 250.
Synophthalmia, 935.
Synote, 935.
Synotia, 935.
Syphilis as a cause of still-births, 221, 225.
  as an inherited disease, 980.
  bacillus of, 167.
  fœtal, 221.
    diagnosis of, 222.
    osteochondritis in, 223, 224.
    prognosis of, 225.
    treatment of, 225.
  from vaccination, 747.
  geographical range of, 144.
  how distinguished from rheumatism, 811.
  in the wet-nurse, 332, 334.

T.

Table for the differential diagnosis of relapsing fever, 510.
    of rubella, measles, and scarlet fever, 699.
  of defects of the upper extremities due to maternal impressions, 205.
  of first menstruations, 391.
  of infantile weights, 52.
  of rules for feeding, 273.
  showing relative size of the head and chest, 94.
  showing significance of temperature, 103.
Tables of defects supposed to be due to maternal impressions, 196–202, 205, 207, 208, 209, 210.
  of temperatures in typhus fever, 498.
  showing averages of height and weight, 53, 54, 89, 90, 91.
  showing results from various methods of treating rheumatism, 818.
Taches bleuâtres, 474.
Tact essential in the diagnosis of children's diseases, 81, 82.
Tape-worm, 973.
Tarsal bones, development of, 50.
Taylor, Dr. W. M., his views regarding diphtheria, 590.
Tears, absence of, 108.
Teeth, absence of, 377.
  anomalies of, 376, 377.
  care of the, 367.
  congenital, 376.
  deciduous, 373, 375.
  development of, 69, 373, 375.
  eruption of, 375, 377.
  grinding of the, 381.
  Hutchinson, 379.
  in childhood, 17.
  malformation of, 378.
Teeth, malposition of, 378.
  permanent, 375.
  shedding of first, 375.
  supernumerary, 377.
  (*See* also Dentition.)
Teething as the cause of disease, 75.
  delayed, 92, 378.
  diarrhœa in, 383.
  disorders of, 350, 379.
  its effect on the fœtus, 219.
  management of, 386.
  multiple, 377.
  precocious, 376.
  retarded, 92, 376.
  superstitions about, 386.
  symptomatology of, 379.
  treatment of, 386.
Temperature, charts of, in enteric fever, 458, 460, 462, 476.
    in relapsing fever, 508.
    in typhus, 498.
    in yellow fever, 864.
  daily curve of, 102.
  in disease, 98, 425–427.
  in typhoid, 462.
  (*See* also under the names of the various diseases.)
  instability of, a feature in the diseases of young children, 425.
  normal, 71.
  of the nursery, 364.
Temporal bone, 15.
Tenderness of the abdomen, 86.
Tendon reflexes in diphtheritic paralysis, 633, 672.
  in enteric fever, 472.
Tendon-stiffness in rheumatism, 795.
Terata anadidyma, 925, 926.
  anakatadidyma, 926.
  katadidyma, 925, 926.
  (*See* also Joined Twins.)
Teratology, 922.
  literature of, 938.
Terminal sinus, 953.
Terrors, nocturnal, 121.
Testicle, descent of the, 31, 32.
Testicles, 42.
Tetanus of the newly-born, 138.
Tetany, 121.
Tetrapous monsters, 932.
Theca folliculi, 940.
Therapeutics of infancy, 8, 956.
Thermic fever, 439.
  how discriminated from yellow fever, 871.
Thermogenesis, 418.
Thermolysis, 419.
Thermometry, 98.
Thoma, R., his observations on the growth of the blood-vessels, 6, 255.
Thoracic complications in typhoid, 491.
Thoracopagus, 937.
Thorax, the, 24.
  size of, at birth, 55.
    in relation to head-diameter, 55, 94.
Throat, examination of, 128.
Thrombosis due to injections for nævus, 17.
  in rheumatism, 793, 805.
Thrombus of the ductus arteriosus, 254.
  neonatorum, 261.
    blood in, 4.
    causes of, 262.
    treatment of, 263.
Thrush, 128.
Thymus gland, 25.
Tibia, development of, 49.
Tied tongue, 19, 121.
Tobacco, its use in early life, 411.
Toilet, 366.
Toilet-powders, 366.
Tongue, defects of the, 19.
  in fever, 429.
Tongue-tie, 19, 121.
Tonic remedies, 969.
Tonsillitis, 128.
  in rheumatism, 800.
Tonsils, anatomy of, 20.
Tooth-rash, 77.
Topography of the body, 23, 24, 26, 28, 30, 32, 36.
  of the neck, 23.
Torticollis, 22, 97.
  traumatic, 265.
Touch, the sense of, in early infancy, 63.
Toxæmia. *See* Blood-poison.
Trace, the primitive, 946.
Tracheo-bronchial glands in whooping-cough, 710.
Tracheotomy, 22.
Training, mental, moral, and physical, 409, 413.
  of girls of the upper classes, 413.
Trance, hysterical, 397.
Transfusion of blood, 966.
Transmission of disease by heredity, 110.
    by milk as a vehicle, 557, 594, 901.
Traumatism of the fœtus, 232.
Treatment of disease by the skin, 956.
  of nervous disorders of female puberty, 415.
  specific, of typhoid, 495.
  (*See* also under the names of diseases in this index.)
Tremor in enteric fever, 488.
Triangle, Hesselbach's, 33.
Trichinosis, how distinguished from typhoid, 481.
Trismus neonatorum, its geographical range, 138.
Trunk, etc., as affected by maternal impressions, 209.
Trypsin in diphtheria, 672.
Tube-cultures, 183, 184.
Tubercle, miliary, 404.
Tubercular meningitis, convulsions in, 428.
    its diagnosis from enteric fever, 119.
Tuberculosis, acute, simulating typhoid, 480.
  as related to scrofula, 403.
  bacillus of, 152, 165.
  causes of, 403.
  contagious quality of, 403, 404.
  curability of, 403.
  fœtus usually exempt from, 229.
  inoculations with, 403.

Tuberculosis of puberty, 402.
  of the breast in the wet-nurse, 331, 335.
  scrofula as a cause of, 403.
  tendencies to, 111.
  treatment of, 404.
Tubes, Esmarch's, 183.
  feeding-, 298.
Tumors, internal, diagnosis of, 85.
Turkeys, diphtheria in, 593.
Turpentine in diphtheria, 617.
Twins, joined, 922.
  (*See* Joined Twins.)
  Siamese, 936.
Two-headed Nightingale, 930.
Tympany in enteric fever, 489.
Typhoid fever. *See* Enteric Fever.
Typho-malaria, 842.
Typhus fever, 497.
  anatomical characters of, 498.
  causes of, 497.
  contagion of, 498.
  definition of, 497.
  diagnosis of, 499.
  etiology of, 497.
  history of, 497.
  hospital treatment of, 498.
  how distinguished from hemorrhagic small-pox, 500.
  maculæ of, 498, 499.
  pathology of, 498.
  prognosis of, 501.
  recurrent. *See* Relapsing Fever.
  sanitation of, 497.
  symptoms of, 498.
  synonymes of, 497.
  temperature in, 498.
  treatment of, 502.
    in hospitals, 498.
Tyrotoxicon, 371, 970.

U.

Ulceration of the bowels in enteric fever, 454.
Ulcero-membranous pharyngitis, 608.
Ulna, its development, 48.
Umbilical artery, closure of, 252.
  cord, 237, 955.
    in monstrosities, 937.
  vein, 255, 256.
  vessels, 6, 255.
Umbilicus, 30.
Understanding, the development of the, 64.
Unilateral deformity of the head, 97.
Unsanitary surroundings as a cause of tuberculosis, 403.
Upper extremity, 46.
  as affected by maternal impressions, 205.
Urethra, 40.
Urinary calculus, 130.
  fistulæ, 30.
  sediments, 130.
Urine, 70.
  examination of, for signs of disease, 75.
Urine in fever, 432.
  in sickness, 353.
  in typhoid, 473.
Urticaria, how distinguishable from erysipelas, 780.
Uterine disease, its effect on the fœtus, 219.
Uterus, 42.
  and bladder, their relative position in the infant, 18, 42.
Uvula in diphtheria, 613, 614.

V.

Vaccination, 743, 749.
  age best suited for, 752.
  best season for, 752.
  conveyance of other diseases in, 747.
  dangers of, 747, 751.
  definition of, 743.
  degeneration of the lymph of, 748.
  erysipelas from, 751, 774.
  etymology of, 747.
  forms of the lymph of, 749.
  history of, 743.
  in early infancy, 752.
  irregular effects of, 751.
  lymph used in, 747, 749.
  methods of, 749.
  nature of its effects, 746.
  operation of, 749.
  phenomena of, 750.
  preservation of the lymph of, 749.
  protection afforded by, 751.
  repetition of, 753.
  season of the year for, 752.
  selection of lymph for, 749.
  susceptibility to its effects, 752.
  syphilis from, 747.
  virus of, 747, 748.
Vaccinia. *See* Vaccination.
Vaccine lymph, 747, 748.
  degeneration of, 747.
  forms of, 749.
  preservation of, 749.
  selection of, 749.
Vapors, medication by, 978.
Varicella, 754.
  age of patients, 756.
  anæmia following, 766.
  causes of, 756.
  complications of, 763.
  constitutional effects of, 761, 765.
  contagion of, 757.
  cutaneous lesion of, 764.
  defined, 754.
  diagnosis in, 763.
  distinctness from variola, 755, 758, 763.
  eruption of, 760.
  etiology of, 756.
  gangrenous, 762.
  history of, 754.
  incubation of, 754.
  inoculability of, 757.
  invasion of, 764.
  literature of, 756.
  macules of, 760.
  mucous membranes in, 761.
  names of, 754.
Varicella, nephritis following, 763.
  pathological anatomy of, 759.
  prognosis in, 766.
  propagation of, 757.
  rash of, 760, 761.
  scars from, 761.
  sequels of, 763.
  symptoms of, 759.
  syphilis simulating, 765.
  treatment of, 766.
Variola, 722.
  age of patients, 723.
  anatomical characters of, 724.
  bacteria of, 724.
  causes of, 723.
  complications of, 725, 733, 736.
  confluent, 729, 731.
  contagion of, 723.
  corymbose, 729.
  death from, 736.
  decline, stage of, 731.
  definition of, 722.
  desiccation of, 731.
  diagnosis of, 737.
  digestive tract in, 734.
  discrete, 729.
  ear-troubles in, 735.
  eruptive stage of, 728.
  etiology of, 723.
  eye-troubles in, 726, 734.
  fœtal, 228, 723.
  geographic range of, 144.
  heart in, 734.
  hemorrhagic, 729, 732.
  history of, 722.
  how distinguished from enteric fever, 479.
  incubation of, 726.
  initial rash of, 727, 728.
  inoculation of, 743, 744.
  in utero, 228.
  invasive stage of, 727.
  iron in, 741.
  keratitis in, 734.
  lesions in, 725.
  maturation, stage of, 730.
  modified or varioloid, 727, 729, 732.
  names of, 722.
  pathology of, 724.
  pitting in, 740.
  prognosis in, 736.
  prophylaxis against, 742.
  (*See* Vaccination.)
  range, geographical and racial, 144, 723.
  rash, preliminary, 727, 728.
  second attacks of, 724.
  secondary fever of, 730.
  semi-confluent, 729.
  sequelæ of, 733.
  sex of patients, 723.
  stages of, 726–731.
  symptoms of, 726.
  synonymes of, 722.
  throat-troubles in, 735.
  treatment of, 739.
  types of, 729.
  varieties of, 729.
Varioloid, 727, 729, 732.
Vasculous area, 953.
Vaughan and Novy's discoveries, 450, 451, 970.

Vaughan and Novy, their proposed definition for infectious disease, 45.
Veins, omphalo-mesenteric, 953.
Ventilation, 354, 362, 363.
Vermiform appendix, 970.
  its position, 35.
Vernix caseosa, 71, 239.
Vertebral column in infancy, 3, 43.
Vesical artery, its development, 256.
Vesication in therapeutics, 981.
Vesicle, germinal, 940.
Vesicular emphysema in diphtheria, 615.
Vessels, lymphatic, 22, 40.
Vibrio (the genus), 154.
Vibrion septique, 154.
Viscera, position of, 24.
  (*See* also Topography.)
Vitelline membrane, 952.
Vitello-intestinal duct, 952.
Vitellus, 940.
Voice in hysteria, 396.
Volitional movements, 56.
Vomiting from overfeeding, or from improper food, 341.
  in enteric fever, 466.
  in meningitis, 428.
  its significance, 126.
  of yellow fever, 866, 867.
Vulvitis, 130.
  diphtheritic, 625.

W.

Walking, 61.
  delay in, 92.
  loss of the power of, as a sign of rachitis, 92.
Warming the nursery, 362.
Warren, Dr. J. C., on the closure of the ductus arteriosus and of the umbilical and hypogastric arteries, 252.
Washing the newly-born child, 240.
Wasting, 91.
  as a diagnostic sign, 88.
Water-closet, 365.
Water for drinking, 372.
Watery solutions of dyeing-material in bacteriological studies, 161.
Waxham, Dr. F. E., on measles, 675.
Wealth and poverty as factors in disease, 135.
Weaning, 270, 287, 387.
  diet after, 337.
Weekly gain in weight, 271–273.
Weight and height as influenced by food, 90, 92.
  as a diagnostic index, 87.
  as a guide in prognosis, 91.
  its relations to height, 90, 92.
  normal gain or growth in, 5, 52, 273.
  of the blood as compared with that of the body, 65.
Weight of the heart as compared with that of the lungs, 5.
  ratios of height to, 53, 88, 89, 90.
  weekly gain in, 271–273.
Westmanna Islands, trismus neonatorum in, 139.
Wet-nurse, 286, 330.
  diet of, 336.
  examination of, 333–336.
  moral qualities of, 330.
  physical condition of, 331.
  signs of disease in, 333.
  syphilis in, 332, 333, 334.
Wetting the bed, 130.
Wharton, the jelly of, 955.
Whey, wine, 342.
Whipham's table showing the results of various plans of treatment for rheumatism, 818.
Whooping-cough. *See* Pertussis.
Will, development of the, 56.
Willingham prodigy, the, 393.
Wilson, Dr. J. C., on enteric fever, 441.
Windows, 363, 365.
Wine whey, 342.
Winter residence for tuberculous children, 407.
Woman's milk, 275, 279, 280, 282, 283, 284, 289, 290, 303, 306, 308, 310.
Wood, Miss Catherine, on the nursing of sick children, 347.
Wormian bones, 12.
Worms, 129, 343, 972.
Wounds of the fœtus, 232.
  of the scalp and face, 262.
Wry-neck, 22, 97.
  traumatic, 265.

X.

Xiphopagus, 936.

Y.

Yale, Dr. L. M., on nursery hygiene, 360.
Yellow fever, 853.
  accidental epidemics of, 858.
  anatomical characters of, 864.
  as a children's disease, 853.
  black vomit in, 867.
  blood in, 865.
  cadaver after, 864.
  causation of, 857.
  charts illustrating, 855, 865.
  compared with dengue, 893.
  complications of, 870.
  contagiousness of, 856, 860, 863.
  deaths from, tabulated, 856.
  definition of, 853.
  diagnosis of, 870.
Yellow fever, differential comparison of, with dengue, 893–895.
  effect on the fœtus, 230.
  epidemic quality of, 858, 861.
  etiology of, 857.
  facies in, 866.
  fever of, 865.
  focal zone of, 858.
  fœtal, 230.
  fomites in, 860.
  food in, 875.
  geography of, 858.
  hemorrhages in, 869.
  history of, 853.
  in children, 853.
  incubation of, 863, 865.
  jaundice of, 866.
  kidneys in, 864.
  liver in, 864.
  morbid anatomy of, 864.
  mortality from, 856, 859.
  muscles in, 864.
  names of, 853.
  nervous symptoms in, 869.
  odor in, 867.
  pathology of, 864.
  perifocal zone of, 858.
  perspiration in, 867.
  prognosis in, 872.
  prophylaxis of, 875.
  pulse in, 865.
  quarantine in, 875.
  race in, 875.
  second attacks rare, 870.
  sex in, 863.
  skin in, 866, 875.
  spleen in, 865.
  stages of, 863.
  stimulants in, 875.
  stomach in, 864.
  symptoms of, 865.
  synonymes of, 853.
  tables of death-rate, 851.
  temperature in, 865, 866.
  thermic fever distinguished from, 871.
  tongue in, 867.
  transmission of, 856, 860, 863.
  treatment of, 872.
  typhoid condition in, 869, 874.
  urine in, 867, 874.
  vomiting of, 866, 867.
  zones of, 858.
Yellow milk, bacterium of, 152.
Yolk-sac, 952.
Youth, dangers which beset, 408, 409.

Z.

Zeiss's oil-immersion apochromatic lens, 158.
Zenker's degeneration, 613.
Zona pellucida, 940.
  radiata, 941.
Zones of yellow fever, 858.
Zoogloea, 156.
Zopf's classification of the bacteria, 150.

END OF VOLUME I.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

AUG 7 1967

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
300 PASTEUR DRIVE
PALO ALTO, CALIF.

P23 Keating, J.M. 14109
K25 Cyclopaedia of the di-
1889 seases of children.
v.1

NAME	DATE DUE
Santa Clara	AUG 7 1967